Spontaneous Hypoglycaemia

REPORT ON TWO POSSIBLE CASES IN AFRICANS

BY

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For some while I have been aware of an interesting disorder in the adult African admitted to the ward in a comatose, stuporose or mentally confused state in which a very low serum glucose occurs and which appears to respond dramatically to glucose. At first I was inclined to disregard the levels of the serum glucose as being too low and did not take the findings too seriously. However, as several such cases have been noted it is worth while recording the findings of two cases which were better documented than the others.

In a very fine study Neame and Joubert (1961) in Durban recorded a large series of African and a few Indian patients who suffered from post-alcoholic hypoglycaemia and toxic hepatitis. Alcohol in their experience is the most common cause of hypoglycaemia.

The subjects usually admit to having been in good health or to have experienced vague symptoms such as headache for a week or more before the onset of the disorder. As far as I can recall the subjects have all been males and otherwise in good health. There may be a history of a similar attack on previous occasions.

The importance of recognising the nature of the disorder lies in the rapid recovery which can usually be effected when glucose is administered. Where facilities exist for a blood glucose estimation the diagnosis can be made fairly quickly, but often this is not possible in many hospitals. Without this the diagnosis cannot be made, but if other common reasons for coma or stupor have been eliminated as far as possible, glucose will effect a quick return of consciousness. In this instance the diagnosis would only be presumptive. However, it would be a wise procedure to take a sample of blood in all such cases when the cause is not immediately clear.

My impressions are that the condition is benign and that after the restoration of consciousness the person resumes his usual state of good health until after a very variable interval of time, which may be months, the next attack sets in.

ILLUSTRATIVE CASES

The patient was an African male, aged about 24 years, and was employed by a European as a cook. He claimed that he was in good health until the 4th March, 1961, when he noticed that he had become constipated. After a few days he developed a headache, for which his employer prescribed an "Aspirin." He was no better, however, and by the next day he became so ill that he was unable to recall anything until after his recovery in hospital. He was taken to the out-patient department of Harare hospital, where he was found to be mentally confused. He seemed to have a severe headache and was restless. At no time did he vomit and there was no fever.

He was admitted to the ward at 2 a.m., when he was seen by Dr. O. Meyer, my registrar, who found him to be in a state of stupor, restless, but not violent. His speech was slurred. A lumbar puncture revealed a clear fluid under a normal pressure. Being aware of the possibility of a severe hypoglycaemia in African practice, and not finding any other obvious cause for the stupor, he administered 50 ml. of 50 per cent glucose by the intravenous route. By 8 o'clock the next morning the patient was very much improved, and by the third day after his admission we had the greatest difficulty in keeping him in the ward in order to carry out some relevant tests.

On admission to hospital his temperature was 99.6° F., but by the next day it had returned to normal. His pulse rate was 72 per
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SPONTANEOUS HYPOGLYCAEMIA

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minute and the rhythm regular. His respiration was 16. The heart and lungs were normal and blood pressure 140/100. No abnormality was detected on abdominal examination. His liver and spleen were not palpable. The deep reflexes were normal and equal and plantar responses doubtful.

Liver function tests: Van den Bergh, negative; serum bilirubin, less than 0.2 mg. per cent.; alkaline phosphatase, 4 King Armstrong units; thymol turbidity, 2; thymol flocculation, negative; serum albumin, 4.6 per cent.; serum globulin, 2.9 g. per cent.; A/G ratio, 1.6:1.

The cerebrospinal fluid was clear, with less than one leucocyte per c.m.; sugar, 77 mg. per cent.; chlorides, 760 mg. per cent.; protein, 40 mg. per cent.; Nonne-Apelt, positive; Wassermann reaction, negative.

A fasting blood glucose taken on three days later was 56 mg. per cent.

The same morning (15th March) the patient could not be persuaded to remain in hospital any longer and left, fully restored in health.

Case 2—Abrosi

Abrosi, an unmarried adult African male, was employed at the time of his present illness as a domestic cook. He was an African, aged about 45 years, of the traditional or rural type, and although he lived in town he could not speak English.

He was admitted in coma to Harare hospital on 2nd September, 1960. Except for a slight cough which he had had for six years and a strange attack of weakness in 1957, he enjoyed good health.

The history obtained after the patient had recovered from the state of coma was that at 6 a.m. on the day he was admitted to hospital he awoke feeling both extremely weak and thirsty and at once drank two pints of water. However, he was so weak at the time that he could not rise from his bed and from now onwards he could not remember what happened. There was no preceding history of polyuria.

He admitted, on closer questioning, to experiencing during 1957 a strange incident. One morning at about 8 o'clock while he was on his way to work, he felt suddenly very cold and a marked weakness overtook him. He was obliged to sit down, and when he did so was unable to lift himself up. He was, however, fully conscious. Passers-by finding him in this state removed him to hospital, where he was treated, and he soon recovered completely.

The patient, when admitted to hospital, was in deep coma. He did not respond to painful stimuli. His pulse was full, regular and rate 54 per minute. The heart was normal, his blood pressure being 190/110. Throughout both lungs scattered sonorous rhonchi and crepitations were audible. There was no oedema in the lower extremities. The abdomen was flat and soft, with no enlargement of the liver or spleen. He was incontinent of urine.

Examination of the head revealed no sign of injury. The pupils were small, regular in outline and reacted normally to light. His neck was not stiff.

The tone of his muscles appeared to be normal. All the reflexes were present and equal, the right plantar being flexor and the left equivocal.

Shortly after his admission to hospital Dr. Matondo decided that as he could not account for the state of coma, he would take blood for an urea and sugar estimation, and he also performed a lumbar puncture. In the meantime he started a slow intravenous drip infusion of 5 per cent. glucose saline. By the next morning the patient was no better and continued to be in a deep coma. About 10 a.m. he learnt that the blood sugar was 26 mg. per cent. and blood urea 38 mg. per cent. The spinal fluid was normal except for its sugar content, which was reported as being less than 10 mg. per cent.
Chlorides, 670 mg. per cent.; protein, 40 mg. per cent.; Nonne, negative; while blood cells were less than 1 per c.mm. Dr. Matondo decided to treat him by setting up a 60 per cent. glucose intravenous drip, and from then onwards the patient made an impressive recovery, taking some eight hours to emerge from the coma. By the next day he was completely better.

The patient admitted to drinking large quantities of African beer, especially at weekends, when he would spend approximately 7s. on it. He smoked 20 cigarettes a day. His diet seems to have been very low in protein. For breakfast he had tea, sugar and bread, and at noon stiff maize porridge twice a week and vegetables five days a week. He did not eat any food after this until the next morning.

Other investigations carried out on this patient during or shortly after his recovery from his stupor were: Hb, 16.2 g. Total leucocyte count, 9,850 per c.mm. Differential count: neutrophils, 69 per cent.; lymphocytes, 31 per cent. The liver function tests (done on 30th September, 1960) were Van den Bergh reaction, positive; serum bilirubin, 1 mg. per cent.; alkaline phosphatase, 5 KA units; total serum protein, 6 g. per cent.; albumin, 4.8 g. per cent.; serum globulin, 1.2 g. per cent.; A/G ratio, 4.0:1; blood Wassermann reaction, negative.

Blood sugar estimations were done on a number of different occasions. All these, except two, were fasting blood sugar determination, the last meal being at 6 p.m. and the blood taken at 8 a.m. On two occasions he was kept off all food except water from 12 p.m. until noon the next day, but no symptoms of hypoglycaemia were induced.

**Fasting Blood Sugar Samples**

<table>
<thead>
<tr>
<th>Date</th>
<th>Blood Sugar</th>
</tr>
</thead>
<tbody>
<tr>
<td>14th September</td>
<td>44 mg. per cent.</td>
</tr>
<tr>
<td>19th September</td>
<td>42 mg. per cent.</td>
</tr>
<tr>
<td>22nd September</td>
<td>87 mg. per cent.</td>
</tr>
<tr>
<td>26th September</td>
<td>60 mg. per cent.</td>
</tr>
<tr>
<td>10th October</td>
<td>55 mg. per cent.</td>
</tr>
<tr>
<td>14th October</td>
<td>43 mg. per cent.</td>
</tr>
<tr>
<td>26th October</td>
<td>38 mg. per cent.</td>
</tr>
<tr>
<td>2nd November</td>
<td>72 mg. per cent.</td>
</tr>
<tr>
<td>7th September</td>
<td>126 mg. (six hours after a meal)</td>
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</tbody>
</table>

A sugar tolerance course was carried out on 6th September, 1960.

The urine was normal clinically and microscopically except for the presence of leucocytes (+ + +). An intravenous pyelogram showed no function on the left side, but the right renal tract was normal in all respects.

**Discussion**

Neame and Joubert (as already mentioned) described a very similar disorder to the one I am now recording, which they refer to as "post alcoholic hypoglycaemia" in Africans and less often Indians of Natal. In all their cases there was a history of alcoholic intoxication on the day of admission or on one or more days before. In one of the two cases I am reporting there was a history of alcoholism, the patient having consumed large amounts of alcohol during the weekends. There was no such history in the other case and it must also be remembered that, in my experience, a high percentage of African males drink alcohol often to excess and therefore it would be risky to attribute this disorder to alcohol alone. There may be other factors, such as an inadequate diet. Further abnormal liver function tests are commonly encountered in the general African population.

Both these subjects were admitted to hospital in a state of coma or stupor, both were found to have abnormally low blood glucose levels and both appeared to respond to intravenous glucose. Further, after they had recovered the serum glucose level remained at a low level.

What comes to mind forcibly is whether we are dealing with pancreatic hyperinsulinism in which the blood sugar is considerably reduced, any figure below 30 mg. per 100 ml. being very suggestive. One of Whipple's clinical triad of organic hyperinsulinism is a persistently low fasting blood sugar, the other two being some degree of cerebral disturbance in the fasting state and relief of the disturbance by glucose. These symptoms may be experienced in the early morning fasting states, but in these two cases, despite a low blood sugar, although not as low as 30 mg. per cent., no symptoms were noticed. In one of the cases I was unable to induce symptoms of any kind after the patient had been without food for nearly 24 hours (ref. Editorial, S. Afr. med. J., 1960).
Hypoglycaemia is one of the features of a fatty liver and occurs typically in kwashiorkor and also in certain forms of cirrhosis of the liver, but other more striking evidence of these disorders will be found in them.

A toxic hypoglycaemia is described recently by Fistein (1960) from Trinidad and Tobago in two young malnourished male East Indians, aged 3½ and 4½ years. One died 5½ hours after breakfast, and at autopsy a fatty liver was shown. The second child fell unconscious and his blood sugar was 20 mg. per cent. He recovered after intravenous glucose and vitamin B therapy were administered. No hepatotoxic agent could be found such as Ackees (Blighia sapida) or bush teas.

Perhaps related to these cases are the interesting states of possession met with in Africans which in the opinion of Field (1960) may be accompanied by a hypoglycaemia. She considers that a hypoglycaemia is probably the greatest aid to dissociation amongst the Ga-people where she carried out her studies. The priest eats nothing on shrine-worshipping days. She noticed that not uncommonly the priest had spent the day hunting or farming and fasted all day and that evening became possessed. I have seen many Africans in this state of possession or what may be more accurately defined as a trance or hysterical dissociation, but the patients I am describing in this paper are in a more advanced state of unconsciousness. The medium in Rhodesia who falls possessed at religious ceremonies does not undergo a preliminary trial of starvation.

REFERENCES