Unusual cause of Coma

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Introduction

Confusion and reduced levels of consciousness are a common cause of admission to medical wards. The differential diagnosis is long and varied, but it is always satisfying (both for the clinician and the patient) to identify a potentially treatable cause of the illness. We present an unusual cause of coma, which after diagnosis and treatment resulted in complete recovery of the patient.

Case report

A 40 year old Malawian woman was admitted to hospital in coma. A few days before admission she had felt tired, and had been given iron tablets and anti-helminthics. The day before admission she had been febrile and had taken chloroquine 600 mg base. On the day of admission she had been found unconscious by her relatives.

Examination revealed a well nourished woman with normal hydration status who was in deep coma. The general examination was normal, and there were no neurological lateralising signs.

Initial investigations showed: thick film for malaria parasites negative; blood glucose 48 mg/dl; serum sodium 129 mEq/l; serum potassium 4.0 mEq/l; CSF was clear fluid with no white cells, protein 25 mg/dl, glucose 25 mg/dl.

Initial management consisted of an intravenous bolus of 50% glucose. She regained consciousness, but remained confused and uncommunicative. Over the following 5 days, despite a continuous dextrose infusion, she had recurrent symptomatic hypoglycaemia which responded only temporarily to bolus doses of 50% dextrose.

On the 6th day of admission she was sufficiently awake and co-operative to give a history. One year previously she had delivered her 5th child by Caesarean section. She had breast fed the other four children, but produced no milk for her 5th child. She failed to menstruate following delivery, and noticed loss of axillary hair.

The main features on examination were: puffy face; slow and slurred speech; no axillary hair; supine pulse rate 52 beats/minute, erect pulse rate 68 beats/minute; supine blood pressure 115/80 mm Hg, erect systolic blood pressure 75 mm Hg.

A clinical diagnosis of hypopituitarism secondary to postpartum infarction (Sheehan's Syndrome) was made. After appropriate blood tests were taken she was given intravenous hydrocortisone 100 mg 6 hourly for 2 days, followed by prednisolone 5 mg mane and 2.5 mg nocte. Blood glucose levels normalised within 24 hours, and the patient's clinical condition improved dramatically. She was started on L-thyroxine 100 µg daily.

She was discharged from hospital on the 10th day on the above doses of prednisolone and thyroxine. She was also given pyrimethamine 25 mg daily. She was able to return to work a month after discharge and follow-up after 4 months showed her to be very well.

Results of investigations prior to hydrocortisone were:

serum cortisol	36 nmol/l	(normal 150 - 650 nmol/l)
serum thyroxine	0.1 pmol/l	(normal 8.0 - 12.0 pmol/l)
serum TSH	0.32 mIU/l	(normal 0.4 - 4.0 mIU/l)
serum FSH	4 IU/l	(normal 1 - 8 IU/l)
serum LH	1.0 IU/l	(normal 1 - 11 IU/l)
serum prolactin	2.0 mg/ml	(normal 2 - 15 mg/ml)

These results confirmed a diagnosis of hypopituitarism. A skull xray was normal with no enlargement of the sella turcica; an abdominal ultrasound showed a normal pancreas and no evidence of calcified adrenal glands.

Discussion

This patient's presenting illness and subsequent course in hospital illustrate a number of important points. First, hypoglycaemia must be considered in any case of coma, and if blood glucose measurements are not available it is always worthwhile administering a bolus of 50% glucose. Second, once hypoglycaemia has been proven the differential diagnosis must be explored (TABLE 1).

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TABLE 1 CAUSES OF HYPOGLYCAEMIA

Factitious - Insulin or sulphonylureas
Ethanol induced
Drugs - e.g. salicylates
Acute infections - malaria, septicaemia
Severe liver disease
Sarcoma, hepatoma, other malignant tumours
Addison's disease, hypopituitarism
Insulinoma

In the patient there was no reason to suspect factitious, ethanol induced or drug induced causes of hypoglycaemia. There was no clinical evidence of septicaemia, severe liver disease or malignancy. This left the possibility of Addison's disease, hypopituitarism or an insulinoma. Hypoadrenalism was suspected by the initial low serum sodium on admission, and the marked postural hypotension despite normal hydration status. Hypoadrenalism is due either to disease of the adrenal glands or secondary to pituitary dysfunction. In this case, hypopituitarism was considered by the clinical features of gonadotrophin deficiency (failure of menstruation and loss of secondary sexual characteristics), prolactin deficiency (failure to breast feed following delivery) and hypothyroidism (puffy face, slow mentation and speech and bradycardia with an abnormal response to drop in blood pressure).

Third, in considering the causes of hypopituitarism (TABLE 2), the advent of clinical features of gonadotrophin and prolactin deficiency following pregnancy made post-partum infarction of the pituitary gland the most likely possibility.

TABLE 2 CAUSES OF HYPOPITUITARISM

Congenital	
Pituitary tumours	
Malignant disease	
Infections	- tuberculosis; syphilis; encephalitis
Granulomatous disease	- sarcoidosis
Vascular disease	- "Sheehan's syndrome"
Iatrogenic	

During pregnancy the pituitary gland becomes hyperplastic, and is therefore susceptible to ischaemia and infarction in the event of hypotension from postpartum haemorrhage, anaesthetic procedures etc. Sheehan's syndrome (as it is called) is thought to be the commonest cause of hypopituitarism in Africa. Initially, there is failure of secretion of LH, FSH and prolactin, and this is followed by various degrees of hypothyroidism and/or adrenocortical insufficiency. These latter deficiencies may only be unmasked by some stressful situation such as infection and trauma, and this was presumably the case in our patient. Although the confirmed diagnosis of hypopituitarism requires refined tests, it can be suspected by attention to the history and clinical examination. We were quite happy to make the diagnosis on the history and examination, and to institute treatment accordingly.

As health workers in Africa, we are continually confronted with scarce resources, and decisions have often to be prioritised. It is therefore of vital importance that treatable causes of illness are identified. Hypoglycaemia is a treatable cause of coma, and hypoadrenalism/hypopituitarism are treatable causes of recurrent and protracted hypoglycaemia.