MEDICAL MEMORANDA

Successful Treatment of Disseminated Intravascular Coagulopathy Complicating Diabetic Coma

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In view of the abnormalities of vascular endothelium (Goldenberg *et al.*, 1959), platelet adhesiveness (Hassanein *et al.*, 1972), and plasma fibrinogen levels (Mayne *et al.*, 1970) in diabetes mellitus it might be thought that disseminated intravascular coagulopathy (D.I.C.) would occur often in diabetic keto-acidosis. There have been only four cases reported, however, all being fatal (Kwaan *et al.*, 1972; Cooper *et al.*, 1973; Timperley *et al.*, 1974).

We think this is the first description of the successful treatment of a patient with diabetic coma and the D.I.C. syndrome. With increased awareness of this syndrome in diabetic coma earlier recognition and energetic treatment should reduce the mortality of uncontrolled diabetes.

Case Report

A 55-year-old West Indian housewife was admitted semi-comatose. For the previous week she had had increasing thirst with anorexia and abdominal pain but no nausea or vomiting. There was no past history of diabetes mellitus and the family history was negative. On examination she was obese and dehydrated. There were no signs of acidosis. Her pulse was regular at 100/min, blood pressure was 120/70 mm Hg, and temperature was 37°C. Cardiovascular and respiratory systems were normal. She had upper abdominal tenderness with guarding, but normal bowel sounds. Spontaneous movements were decreased in the left arm and leg with hypotonus and hyporeflexia; both plantar responses were flexor. There was no neck stiffness and the fundi were normal.

INVESTIGATIONS

Urine contained 2% glucose with a trace of ketones. Blood sugar was 584 mg/100 ml, sodium 146 mmol/l., potassium 4.5 mmol/l., urea 123 mg/100 ml, calculated osmolality 353 mosmol/l. The arterial pH was 7.3, P_{CO_2} 34 mm Hg, standard bicarbonate 22 mEq/l. The haemoglobin was 18.6 g/100 ml, W.B.C. 12,400/mm³, packed cell volume 57%. Serum amylase was less than 160 IU/l. The sickle cell test result was negative with normal haemoglobin electrophoresis, and chest radiograph and E.C.G. were also normal. Cerebrospinal fluid contained protein 44 mg/100 ml and sugar 500 mg/100 ml. Urine, blood, and cerebrospinal fluid were sterile on culture.

She was given intravenous fluids, soluble insulin, and ampicillin. The dehydration and hyperglycaemia responded satisfactorily, but the patient became hypernatraemic (serum sodium 171 mEq/l.) on the second day after admission. The infusion was continued with 5%

dextrose and the serum sodium fell to normal values over the next 24 hours. Urine output was satisfactory throughout.

The patient remained drowsy and restless and did not improve after treatment with frusemide and mannitol. On the third day she developed haematuria and epistaxis but no petechiae or bruises. Investigations showed the blood sugar to be 265 mg/100 ml, urea 66 mg/100 ml, sodium 145 mmol/l., and potassium 4.0 mmol/l. The haemoglobin was 13.9 g/100 ml, W.B.C. 12,400/mm³, platelets 15,000/mm³, with fragmented erythrocytes on the blood film. The prothrombin time was 15 sec (control 14 sec), partial thromboplastin time 38 sec (normal 20-40 sec), thrombin clotting time after 90-sec incubation 53 sec (control 20 sec). The fibrinogen titre was 1/32 (normal > 1/64) and fibrin degradation products were 200 µg/ml (normal < 10 µg/ml). The cholesterol was 197 mg/100 ml and total lipids 651 mg/100 ml. The sternal marrow was normal.

She was given heparin 10,000 units intravenously every four hours for five days. Within 24 hours of starting heparin she had improved considerably, and bleeding had stopped and consciousness returned without residual neurological signs. The platelets, plasma fibrinogen, and fibrin degradation products returned to normal values within four days. Insulin was discontinued after two weeks and control of the diabetes achieved with diet alone. She was discharged three weeks after admission and remained well when seen at follow up a month later.

Comment

Focal neurological signs are not uncommon in hyperosmolar coma (Jackson and Forman, 1966) and we assumed that this was the cause of the hemiparesis; treatment for cerebral oedema, however, gave no improvement. D.I.C. may also present with focal neurological signs, but it was diagnosed only when bleeding became obvious on the third day. It had been noted that blood taken for investigations 24 hours previously had been slow to coagulate, but the significance of this was not then appreciated.

Investigations gave no evidence that the D.I.C. was secondary to any of the previously recognized causes of the syndrome (Nalbandian *et al.*, 1971). Our patient fulfilled all the criteria for the diagnosis of D.I.C. (Colman *et al.*, 1972), with clinical and haematological improvement after heparin as confirmatory evidence.

It is perhaps surprising that D.I.C. has not been diagnosed more often in diabetes, but in two recent series totalling 740 admissions (Beigelman and Warner, 1971; Soler *et al.*, 1973) no episode was recognized. Possibly the syndrome is not so uncommon since necropsy appearances compatible with D.I.C. have been described in six diabetics, with haematological evidence supporting the diagnosis obtained before death in two (Timperley *et al.*, 1974). We show that treatment can be dramatically effective, and we advise that blood platelet counts and fibrin degradation product estimations should be done in all patients with diabetic coma who fail to improve satisfactorily.

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