limbs showed an upper motor neurone type of lesion with complete loss of power, brisk knee- and ankle-jerks, absent abdominal reflexes, and bilateral extensor plantar response. Superficial as well as deep sensation was lost up to about 5 cm. above the umbilicus, thus suggesting localization of the lesion at about the eighth dorsal segment. The spine was normal.

Investigations.—Routine blood examination showed haemoglobin 11.5 g./100 ml., total leucocyte count 6,750/c.mm. (normal differential), erythrocyte sedimentation rate (Westergren) 32 mm. in first hour. Urine and stool examination did not reveal any abnormality. The cerebrospinal fluid was clear and under normal tension; it contained no leucocytes or red blood cells. On biochemical analysis the protein content was 80 mg./100 ml. with globulins ++, chlorides 680 mg./100 ml., and sugar 35 mg./100 ml. Serological examination of blood and C.S.F. was negative.

On the third day of admission he had an attack of generalized convulsions, followed by transitory unconsciousness. An x-ray film of the skull did not reveal any abnormal calcification. X-ray examination of the dorso-lumbar spine as well as pectoral regions showed multiple areas of calcification. On myelography by the lumbar route the myodil column was found displaced to the left side as a whole, opposite the lower border of the eighth dorsal vertebra. The diagnosis of spinal compression by a cysticercus cyst, in view of the evident calcification in the striated muscles, was made, and the case was prepared for laminectomy.

At operation the cord was swollen opposite the sixth and seventh dorsal vertebral spines. A midline incision was made in the cord: an intramedullary cyst bulged out and was delivered by gentle pressure. Naked-eye examination revealed it to be a glistening white cyst with a knobby area of calcification measuring 2.9 by 1 cm. Histological examination of the cyst wall showed it to be a cysticercus cyst.

During the post-operative period there was marked deterioration in the general health of the patient due to progressive bedsores and urinary infection, and he ultimately succumbed to his ailment.

COMMENT

Spinal cysticercosis is a rare cause of paraplegia and is likely to be missed unless calcification of cysticercus cysts is found on routine radiological examination. In the present case an attack of epileptiform seizures during the patient's stay in hospital offered a further clue towards the diagnosis, though there was no such history prior to this episode.

It is suggested that spinal cysticercosis should be borne in mind while investigating a case of obscure paraplegia.

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Hyperosmolar Non-ketoacidotic Diabetic Coma in a Patient with Necrotizing Pancreatitis

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Sament and Schwartz (1957) and De Graeff and Lips (1957) gave the first clinical description of hyperglycaemic non-ketotic coma. Since then over two dozen cases have been reported and the condition is being recognized more often. Davidson (1964) published the only report that drew attention to its association with severe acute pancreatitis.

In the case described below unsuspected necrotizing pancreatitis was responsible for the development of diabetic coma without ketoacidosis.

CASE REPORT

A 56-year-old man was admitted to a surgical ward with complaints of low backache and abdominal pain of four days' duration. There was nothing relevant in his past history. He was mildly obese. Blood-pressure was 160/100, pulse 80, normal sinus rhythm. Temperature was 101.4° F (38.6° C). There was slight tenderness and fullness in the left iliac fossa. His urine contained sugar + +. No other abnormalities were present.

A diagnosis of diverticulitis was made and he showed a good initial response to broad-spectrum antibiotic therapy and low-residue diet. A week later he suddenly became confused. In view of the rapid deterioration in his condition medical opinion was sought.

On examination he was semicomatose. He did not answer questions, but would occasionally respond to commands. Reaction to painful stimuli was diminished. Frequent shaking and twitching

of extremities was seen. There were no other neurological abnormalities. The tongue was very dry. Skin texture was normal. Blood-pressure was 80 mm. Hg systolic; the diastolic pressure could not be recorded. Pulse was 120, regular, and of weak volume. There was slight tenderness in the left iliac fossa.

Urine: sugar + + + +, no acetone. Blood sugar 800 mg./100 ml. Serum sodium 170 mEq/l., potassium 2.8 mEq/l., chloride 135 mEq/l., bicarbonate 20 mEq/l. Blood urea 60 mg./100 ml. Plasma specific gravity 1032+.¹ Serum osmolarity 388 mOsm/kg. H₂O.² White-cell count 27,000/c.mm. E.C.G. within normal limits. Serum amylase 25 Street-Close units.

Treatment was begun with intravenous fluids and intravenous and intramuscular insulin. In the first 12 hours the patient received 7 litres of intravenous fluids comprising 1.5 litres of normal saline and 5.5 litres of 5% dextrose, and 450 units of insulin. The blood sugar fell to 360 mg./100 ml., the serum sodium to 156 mEq/l., and the plasma specific gravity to 1027. These changes were accompanied by marked improvement in the patient's general condition and mental state. The pulse rate settled at 90 per minute and the blood-pressure at 140/85.

The urinary output was only 200 ml. in the next 24 hours in spite of clinical and biochemical evidence of rehydration. The blood urea rose to 184 mg./100 ml. The diagnosis of tubular necrosis secondary to dehydration and hypotension was made and peritoneal dialysis instituted. Renal function did not recover, and dialysis had to be maintained continuously until the patient's death two and a half weeks later.

The course of his illness was further complicated by the development of gross ischaemia of the left lower leg. All pulses below the femoral artery became absent. Anticoagulation did not prevent rapid progression to gangrene. A mid-thigh amputation was performed. Atheroma with overlying organizing thrombus was seen in both posterior tibial and popliteal arteries.

The patient needed 80-100 units of insulin daily for the first four days after his collapse; then the insulin requirements rapidly

¹ Readings become inaccurate over 1032.

² Calculated from serum sodium and glucose concentrations.

diminished. By the seventh day 10 units of soluble insulin was adequate in maintaining the blood-sugar level between 80 and 160 mg./100 ml. He developed progressive anaemia, dizziness, hypotension, a rising blood urea, and eventually died four weeks after admission.

Post-mortem Examination.—The significant abnormal findings were limited to the pancreas, kidney, and brain. The architecture of the pancreas was completely destroyed throughout its length by massive necrosis. Haemorrhage was not a prominent feature. Widespread fat necrosis was present in the peritoneal cavity, with cloudy ascitic fluid confined mainly to the lesser sac. Both kidneys were greatly enlarged, weighing 260 and 270 g. The surface was smooth. On section they appeared oedematous, with swelling of both cortex and medulla. Microscopical examination showed some groups of tubules lined by necrotic epithelium, others by regenerating epithelium. Many tubules contained small round calcified cysts. The glomeruli were normal. An area of recent infarction was seen in the right parietal region of the brain. Terminal septicaemia was suggested by the finding of clumps of bacteria in the capillaries of almost every organ.

COMMENT

Prompt recognition and treatment of this potentially dangerous condition is essential. Di Benedetto *et al.* (1965) quote a 44% mortality from review of the literature.

The patient is usually elderly. Previous history of diabetes is lacking in most cases. Clouding of consciousness or coma is invariably present. Focal convulsions are common. Maccario *et al.* (1965) described seven cases in which seizures were a prominent feature. They suggest that hyperosmolality or the possibly direct toxic effect of hyperglycaemia is responsible for the neurological dysfunction. It is possible, however, that the action of these lies in unmasking an epileptic tendency in an already diseased brain.

Severe dehydration is a main feature of non-ketotic coma. Often, as in this case, the presence of hypernatraemia indicates relatively greater loss of water than of sodium. Very large amounts of intravenous fluids may have to be given before rehydration is complete. Lucas *et al.* (1963) administered 17 litres in 36 hours to one of their patients. In view of the hypernatraemia and intracellular dehydration the use of isotonic saline is best avoided. Diuresis is often delayed because of the severe dehydration. The possibility that oliguria is secondary to tubular necrosis has to be considered early. Tubular necrosis was a complicating factor in this case, and has been reported by Mach and de Sousa (1963) and by Bergoz and Hausser (1964).

Severe pancreatitis was noted in two cases, including the present one (Davidson, 1964). Patchy involvement of the pancreas was described by Ward (1963) and by Bergoz and Hausser (1964). They represent about 20% of cases coming to necropsy. Acute pancreatitis could be a major aetiological factor in the production of non-ketoacidotic diabetic coma. Its association with diabetic acidosis and coma is already well documented (Hughes, 1961; Tully and Lowenthal, 1958).

Serum amylase estimations should be performed routinely. This may help in detecting pancreatitis in patients who recover from their coma. The normal serum amylase in the present case can be accounted for by complete necrosis of all pancreatic tissue.

Ketosis increases the insulin requirements of diabetic coma. Bergoz and Hausser (1964) comment that the need for insulin in coma without ketoacidosis is small. However, even their cases required 360 units of insulin in the first 12 hours to reduce the blood sugar to 440 mg./100 ml. Administration of 450–560 units of insulin in the first few hours is not unusual. If the patient survives the acute illness good diabetic control can be achieved by diet or oral hypoglycaemic agents.

Danowski and Nabarro (1965) divide non-ketotic coma on the basis of the plasma bicarbonate being above or below 18 mEq/l. Elevated serum lactic acid could be responsible for the acidosis (Daughaday *et al.*, 1962). Di Benedetto *et al.* (1965) also draw this division according to the presence or absence of lactic acidosis and emphasize the different treatment in the two groups.

Polymorphonuclear leucocytosis and elevated temperature are common. These features sometimes initiate an unrewarding search for an underlying infection. Dehydration can be responsible for these signs, which disappear once rehydration has been achieved. It is likely that the involvement of several organs by bacteria in this case was a terminal event.

ADDENDUM.—Since sending this report for publication further cases of hyperosmolar coma have come to light and a more detailed account of this variant of diabetic coma has been published (Halmos, P. B., Nelson, J. K., and Lowry, R. C., 1966). The above case is included as Case 6.

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