Diabetic Ketoacidosis Presenting as Neurosurgical Emergencies

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Summary

Three cases of ketoacidosis in previously unsuspected diabetics are described. Each was admitted to hospital as a neurosurgical emergency. The recognition of this rare presentation is stressed since prompt treatment of the metabolic disturbance rapidly resolves the neurological abnormality.

Introduction

Diabetes mellitus is a ubiquitous disease. Its manifestations or complications may affect any bodily system. In most cases diagnosis presents no difficulty; glycosuria is noted on routine examination, there is a history of polydipsia and polyuria, perhaps with polyphagia. There may be a family history, or a common complication may be recognized. In a minority onset is acute with acidic coma. When this conforms to the description in standard medical texts the correct diagnosis is seldom mistaken—deterioration over days or hours to coma with acidoic or Kussmaul respiration, ketone bodies in the breath, dehydration, and glycosuria, often precipitated by infection or surgery. Less well recognized is the case where the patient develops focal neurological signs which may mimic neurosurgical conditions. Personal experience of three cases admitted to a specialist unit as neurosurgical emergencies who were found to be suffering from diabetic ketoacidosis is described to emphasize this uncommon presentation that it may be more easily recognized.

Case 1

A 63-year-old woman was involved in a road traffic accident before admission to a neurosurgical unit. She suffered superficial lacerations of head, bruising of chest and both legs, and she was concussed for a few seconds. No bony injury was noted on examination at another hospital, but she was detained for observation for 24 hours and then discharged. Next morning though able to do housework she was unsteady. This increased over two days when she became drowsy and her husband had difficulty rousing her from sleep. The next morning her general practitioner was unable to wake her, found her breathing irregular, suspected subdural haemorrhage, and arranged emergency admission to the Midland Centre for Neurosurgery and Neurology (M.C.N.N.).

For several years she had taken chlorpropamide and benzhexol under psychiatric outpatient supervision for a schizophrenic condition, which remained well controlled. There was no relevant past history.

On examination she was semi-comatose, responding only to most painful stimuli with appropriate withdrawal of each limb. Optic fundi and pupillary reactions were normal. There was no paresis or sensory deficit and tendon reflexes were present and equal and plantars flexor. Healing superficial lacerations were noted in left supraorbital region and bruising was evident over the left anerior chest wall and both shins. Pulse was 120 beats/min, blood pressure 100/70 mm Hg, and respirations, 24/min, were regular and of deep sighing character.

As her breathing appeared acidotic the level of sugar in the blood was measured together with urea, electrolytes, and acid base status. Results were: sugar 706 mg/100 ml, urea 135 mg/100 ml, sodium 160 mmol/l, potassium 3.5 mmol/l, chloride 123 mmol/l, pH 7.24, Pco2 26.5 mm Hg, base deficit 15.3 mEq/l, and standard bicarbonate 14.2 mEq/l. Urine obtained by catheterization contained sugar and ketones, and Klebsiella and Psuedomonas were subsequently cultured. Results of further investigations were: haemoglobin 16.5 g/100 ml, W.B.C. 11,000/ mm3, and E.S.R. 40 mm in one hour. A small pleural effusion was seen at the left lung base on chest x-ray examination, but there was no bony injury. X-ray examination of the skull showed nothing abnormal.

Diabetic ketoacidosis was diagnosed and appropriate treatment given with insulin, fluid and electrolyte replacement, and ampicillin for the urinary infection. Within 24 hours her conscious level improved and she was alert, lucid, and rational in 48. Deep vein thrombosis of the left leg developed and needed anticoagulation.

Four days after admission she was transferred to another hospital for stabilization of her diabetes, neurologically intact.

Case 2

Three weeks before admission to a psychiatric hospital a woman of 59 became confused and disoriented. Her relatives found her wandering aimlessly in the street, and her bizarre behaviour increased leading to her admission to hospital. No abnormal neurological signs were initially evident, but the day after admission she had a focal epileptic seizure of the left side of her body with postictal paresis. Despite anti convulsant medication she had several more left-sided fits in the subsequent two days, resulting in dense left hemiparesis. Cerebral tumour was suspected and emergency admission to M.C.N.N. requested.

Examination showed an uncooperative semicomatose woman with left hemiparesis involving face, arm, and leg and left homonymous hemianopia. Further left-sided focal fits, each of several minutes duration were observed. There was sinus tachycardia of 120/min, blood pressure was 160/90 mm Hg, and respirations of 18/min were regular.

Electroencephalography showed paroxysms of bilateral spike and wave activity with intervening alpha activity, which was only intermittently evident on the right. Technetium isotope brain scan showed nothing abnormal, and emergency right carotid angiogram showed an atheromatous plaque at the origin of the internal carotid artery, normal intracranial vessels, and no evidence of infarction or space occupation. Haemoglobin was 16.1 g/100 ml, W.B.C. 8,000/mm³, and E.S.R. 27 mm in one hour. Blood urea was 66 mg/100 ml, sugar 456 mg/100 ml, sodium 143 mmol/l, potassium 4.4 mmol/l, chloride 112 mmol/l, pH 7.31, Pco2 29.5 mm Hg, and standard bicarbonate 16.4 mEq/l, and base deficit 10 mEq/l. Urine obtained by catheterization contained sugar and ketones.

Diabetic ketoacidosis was diagnosed and treatment given with insulin and replacement of fluid and electrolytes. This abolished the fits in 36 hours, and within three days she was fully conscious and co-operative and the left hemiparesis and hemianopia had largely resolved. She said that she had been excessively thirsty for several months and her grandmother had been diabetic. She was transferred to another hospital for stabilization of the diabetes and made an uneventful recovery.

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Case 3
A mentally subnormal woman of 37 was known to have suffered bilateral otitis media for several years. Four days before admission she complained of giddiness and her mother noted her left leg was abnormal as “it would throw sideways when she walked.” Three days later when stooping she felt dizzy and developed paraesthesiae in her left arm and leg, and these rapidly became parietic. That evening she was drowsy and incontinent of urine and by 4 a.m. had lapsed into coma. She was admitted to a local hospital, and immediate transfer to M.C.N.N. was arranged for exclusion of cerebral abscess.

Examination showed an unconscious, dehydrated, underdeveloped woman. Pulse was 144 beats/min, temperature 38°C, and respiration 30/min. N. P. T. were normal. There was a dense left hemiparesis affecting face, arm, and leg, with hyperreflexia and extensor plantar responses. The right limbs would withdraw purposively after painful stimulation. Mild neck stiffness was present and the right external auditory meatus was moist and the tympanic membrane perforated.

Right temporal and bilateral posterior parietal burr holes were made. No abscess was found on needleling the right temporal lobe, and C.S.F. in the lateral ventricles was under normal pressure and contained 6 lymphocytes/mm³. Protein was 7 mg/100 ml and sugar 396 mg/100 ml, and no organisms were evident on Gram stain. Air ventriculography showed ventricles of normal size, moderate cortical atrophy, and no evidence of space occupation. Haemoglobin was 15-2 g/100 ml, W.B.C. 24,000/mm³, and E.S.R. was 80 mm in one hour. Blood urea was 64 mg/100 ml, sugar 861 mg/100 ml, sodium 135 mmol/l., potassium 5-6 mmol/l, chloride 102 mmol/l, pH 7-2, Pco₂ 18-7 mm Hg, standard bicarbonate 9-8 mmol/l, and base deficit 19-7 mmol/l. Urine obtained by catheterization contained sugar and ketones.

Diabetic ketoacidosis and otitis media were diagnosed and treatment given with insulin, fluid and electrolyte replacement, and ampicillin. This produced some improvement in conscious level but she had several left-sided epileptic seizures. Culture of ventricular C.S.F. now yielded haemolytic streptococci and penicillin was added to the drug regimen. She returned to the referring hospital next day for stabilization of her diabetes and died suddenly the day after. Permission for necropsy was refused.

Discussion
That patients in diabetic ketoacidosis may present with overt neurological abnormality is not commonly recognized. For this to simulate acute neurosurgical conditions is rare. None of the three cases described was known to be diabetic before admission to hospital. The history in case 1 of concussion followed by a lucid interval then lapse into coma is characteristic of subdural haematoma. The acidic pattern of respiration was first thought to indicate embarrassment of the respiratory centre by increasing intracranial pressure, and the correct diagnosis was made only when the blood sugar was estimated. Mental changes occasioned admission to a psychiatric hospital in the second case. Focal convulsions, hemiparesis, and decreasing level of consciousness suggested cerebral tumour as the cause, and it was only after angiography when the result of the blood sugar was available that the correct diagnosis was made. In case 3 otitis media followed by ataxia, contralateral focal epilepsy, hemiparesis, and coma led to a diagnosis of cerebral abscess. Diabetes mellitus was diagnosed when the result of the C.S.F. sugar was known.

None of the standard medical texts emphasizes the presenta-
tion of convulsive predominance and abnormal neurological signs other than coma, and few such reports have been published. Pillay (1964) described seven Africans with diabetic coma and abnormal neurological signs and stressed the importance of early diagnosis, as prompt treatment produced rapid improvement of the nervous abnormalities. Maccarrio et al. (1965) noted focal seizures as a manifestation of hyperglycaemic non-ketotic coma, and Maccarrio (1968) expanded these to include hallucinations, focal signs of hemianopia, hemiparesis and hemisensory deficit, myoclonic twitches, ataxia, bulbar muscle spasm, meningeal signs, and respiratory disturbance. He felt the frequency of these complica-
tions in hyperosmolar non-ketotic coma contrasted with their paucity in ketoacidotic coma. McCurdy (1970) reviewing hyperosmolar nonketotic coma noted that neurological signs may be prominent and “because of the neurological findings, the patient’s age and lack of diabetic history, an initial diagnosis of cerebrovascular accident is common.”

The mechanism of production of neurological abnormality is not well understood and several hypotheses are extant. Direct toxic effect of hyperglycaemia has not gained popular acceptance. Danowski and Nabarro (1965) suggested hyperosmolality causes cellular dehydration, so altering consciousness in non-ketotic coma. Maccarrio (1968) thinks that this causes focal neurological dysfunction in these cases. Young and Bradley (1967) described fatal cerebral oedema in two cases of diabetic ketoacidosis which were apparently improving with treatment. They argue that hyperosmolality and dehydration raise blood viscosity, which diminishes cerebral blood flow and oxygenation, so inducing cerebral anaemia with resultant cellular electrolyte imbalance and oedema. Aloia and Nilakantan (1973) postulate that accumulation of the products of the polyol pathway, which is active in uncontrolled diabetes, may interfere with neuronal function by osmotic mechanisms or by derangement of enzyme systems. Posner and Plum (1967) found that acidosis of cerebrospinal fluid rather than systemic acidosis had a deleterious effect on brain function. Ketoacidosis was commonly thought to be the cause of neurological dysfunction but cannot be so in cases of non-ketotic coma.

It seems likely that a combination of these mechanisms operates in most cases causing general disturbance of neural function. Epileptic seizures are often resistant to standard anti-convulsants but respond rapidly to fluid and electrolyte replacement and insulin. Focal signs may be due to structural cerebral lesions such as infarction or haemorrhage but no such damage may be evident, implying the disorder is essentially “metabolic.” In the second case the focal signs were apparently caused by this “metabolic imbalance” acting on a hemisphere already compromised by atherosclerosis since correction of the metabolic abnormality soon restored normality. A similar aetiology may be postulated in the third case though complications of meningitis such as lateral sinus thrombosis were not wholly excluded. That metabolic upset may render latent defect apparent is well illustrated by the case described by Portnoy (1965).

In these three cases, features which indicated ketoacidosis were urinaiy infection and trauma in case 1 and meningitis in case 3. None were found in case 2. Difficulty in obtaining urine specimens from comatose patients doubtless led to delay in diagnosis, samples being obtained only after catheterization on admission to M.C.N.N. Since early recognition and prompt treatment of this condition will result in full recovery a plea is made that a urine sample be obtained, if necessary by catheterization, from all patients admitted to hospital with acute neurological illness and tested for sugar. Perhaps more easily a Dextrostix examination of blood could be made to detect hyperglycaemia.

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