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Diabetic ketoacidosis in a district general hospital, 1981 – 1986

D J Eedy, A J McNeill, W J Andrews

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SUMMARY

The outcome of therapy of poorly controlled insulin-requiring cases of diabetes mellitus needing admission to a district general hospital from 1981 to 1986 was examined. There were 156 admissions to the hospital, 17 of these classified as severe diabetic ketoacidosis (serum standard bicarbonate less than 14 mmol/l). A 'low dose' insulin regimen was used in each case of severe ketoacidosis. No patient who was admitted died within a six-month period. These figures emphasise the value of a policy of direct hospital admission for poorly controlled diabetics and suggest that early diagnosis in general practice is vital to allow the application of relatively simple and standard hospital treatment.

INTRODUCTION

The outcome of treatment of diabetic ketoacidosis has improved dramatically during this century. In the pre-insulin era, severe ketoacidosis was uniformly fatal. Despite current therapy, recent studies still indicate a mortality of 5 – 10% in good centres, but suggest mortality rates of 20 – 25% in average district hospitals.[1, 2, 3, 4] Diabetic ketoacidosis is the fourth major cause of death in diabetics and the most common in patients under the age of 20 years.[5] Lower mortality figures do not necessarily appear to be related to the centres with the largest number of hospital admissions, suggesting that clinical experience in hospital may not improve diagnosis in the community.[3]

The aim of this study was to ascertain the mortality from diabetic ketoacidosis over a 66-month period from January 1981 to June 1986 at Whiteabbey Hospital, and to compare this with published figures. Whiteabbey Hospital is a district general hospital with 67 general medical beds staffed by three general physicians, one of whom has an interest in diabetes mellitus. There is no intensive care unit. About 200 patients per month are admitted, the vast majority by direct admission from general practitioners who traditionally have a close relationship with the hospital. There is an accident and emergency unit which is closed from 11.00 pm to 9.00 am weekly and from 5.00 pm to 9.00 am at weekends. A diabetic clinic is held on 1½ sessions per week and approximately 1,500 diabetics attend, 300 of whom are insulin-requiring.

Whiteabbey Hospital, Doagh Road, Newtownabbey BT37 9RH.
D J Eedy, MB, MRCP, Medical Registrar.
A J McNeill, MB, MRCP, Senior House Officer.
W J Andrews, MD, MRCP, Consultant Physician.
Correspondence to Dr Andrews.

PATIENTS AND METHODS

The hospital records and death certificates of all insulin-requiring and non-insulin-requiring diabetics admitted to Whiteabbey Hospital from January 1981 to June 1986 were examined. There were 156 admissions with poorly controlled diabetes. Patients were considered to be severely ketoacidotic only if ketonuria was noted on ward testing of urine and metabolic acidosis was found on blood gas analysis (standard bicarbonate less than 14 mmol/l). Clinical data on admission, venous plasma glucose, serum electrolyte status, insulin dosage and fluid and electrolyte therapy were gleaned from the medical records. Five patients admitted with hyperosmolar non-ketotic diabetic coma were not included in the study.

RESULTS

Fifteen patients (11 female, four male) were admitted in hyperglycaemic ketoacidosis, two female patients being admitted twice. Only three were previously undiagnosed. Mental state on admission ranged from fully conscious to deeply unconscious. The clinical data (Table) are broadly comparable in age, blood glucose and electrolyte parameters with other studies.[6, 7, 8] None of these patients died within six months of admission. Capillary blood glucose was measured hourly using a reflectance meter (Ames Ltd UK) and venous blood was taken for serum glucose, urea, sodium amd potassium measurement on admission, two, four and eight hours after admission, and then daily. Blood gas analysis for pH, the standard bicarbonate concentration and the calculated and base excess values were obtained on admission and at two, four and eight hours. Three patients on admission had a blood pH of less than 7.0, and in two of these it was below 6.8. Insulin was given by the 'low dose' regimen[9] via the intramuscular route in 14 of the cases. Three patients were given only subcutaneous insulin. Mean insulin dosage over 24 hours was 63 ± 8 units.

TABLE

Clinical data on admission in 17 cases of severe diabetic ketoacidosis

	Mean ± *SEM*
Age	30.1 ± 4.3 years
Serum glucose	29.1 ± 2.3 mmol/l
Blood pH	7.10 ± 0.04
Serum HCO_3	6.4 ± 1.0 mmol/l
Calculated base excess	−21.9 ± 1.7 mmol/l
Serum sodium	132 ± 1.5 mmol/l
Serum potassium	5.1 ± 0.3 mmol/l
Serum urea	8.9 ± 1.1 mmol/l

Intravenous fluids were administered for at least 24 hours via a peripheral vein. No central venous pressure monitoring was performed. A mean of 3.8 litres ± 0.3 litres 0.9% saline and 2.9 ± 0.3 litres 5% dextrose was administered in the first 24 hours. In addition, six patients received a mean of 176 ± 47 mmol of hypertonic (435 mmol/l) sodium bicarbonate. No patients developed signs or symptoms of fluid overload.

All patients received intravenous potassium chloride supplementation, mean 119 ± 23 mmol, in the first 24 hours.

There was evidence of an underlying infection in six cases: one patient had pneumococcal pneumonia and one patient had two admissions following exacerbation of bronchiectasis. One patient had a myocardial infarction prior to admission. Three patients had omitted their insulin (one on two occasions) and one patient omitted his oral hypoglycaemic agent because of vomiting and inability to retain food: one of these four had supplemented their food intake with a proprietary glucose drink. One patient had omitted both insulin and food in an effort to lose weight. One patient developed ketoacidosis following pancreatitis.

DISCUSSION

Despite the success in our hospital treatment of diabetic ketoacidosis, we do not wish to under-emphasise the real risk of death in this condition,[5, 10] nor do we claim any special ability. Recent literature supports these suppositions,[1, 2, 3, 4] but there are undoubtedly a number of factors which have significantly contributed to our zero mortality rate over a five-year period.

Firstly, the hospital serves a surburban and rural population and has very close contacts with the family doctors of the area. Indeed, a direct admission policy to our medical wards for emergency cases has been operative since before 1981, partly because of this close contact and partly because of the part-time nature of our accident and emergency service. This system is of vital importance in reducing the time lag between diagnosis of diabetic ketoacidosis and initiation of hospital treatment, but effective operation depends on the enthusiasm and ability of the general practitioners who refer the patient.

Secondly, we have applied the basic low dose insulin regimen,[9] given intra muscularly for simplicity (the majority of our junior staff rotate six-monthly). Three patients were given insulin subcutaneously rather than intramuscularly with equally satisfactory clinical and biochemical outcomes. A guide sheet is present in each ward for inspection and reference by junior staff. In addition we have attempted to give intravenous fluids gradually with potassium replacement from the beginning, hoping to achieve a gradual and steady metabolic improvement. Empirically we have given small amounts of hypertonic sodium bicarbonate, (4.2%, 435 mmol/l) intravenously slowly for patients with pH 7.0 or less. Because of the small numbers we would not wish to make any pronouncements on the benefits or otherwise of this policy — one case who received bicarbonate when admitted unconscious with pH 6.78 remained unconscious for a further 48 hours, and at a much later time after recovery was found to have residual granulation at the site of the infusion.

Thirdly, we may have been fortunate in that the more gravely acidotic admissions were young diabetics (mean age 30 ± 4.6 years). The three newly diagnosed diabetics in our group are of interest, and maybe this relatively high proportion of a group of severely ketoacidotic diabetics would suggest that the diagnosis had not been made sufficiently early in the community. We have omitted five cases of hyperosmolar non-ketotic diabetic coma in order not to confuse the paper: one of these died within six months of admission.

The zero mortality for the treatment of diabetic ketoacidotics who reach our wards might suggest that some of our more elderly patients, who are more likely to have a high mortality, may not be reaching hospital at all or may be providing

the general practitioners with greater diagnostic problems and thus going through the 'Belfast Emergency Bed Service'. In practice, the Emergency Bed Service copes with elderly patients with social problems or uncertain diagnoses, allowing admission to one of the main Belfast teaching hospitals through the 'on-call' take-in unit.

In conclusion we have been fortunate in achieving a zero mortality over five years for the hospital treatment of diabetic ketoacidosis: some peculiarities of our situation as outlined above may have contributed to these figures. Our results may indicate that earlier diagnosis of diabetic ketoacidosis, at least in young people, is taking place in the community. A simple regimen can be successfully followed even by relatively inexperienced staff provided that good nursing and laboratory facilities are available.

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