

## CONTINUOUS GLUCOSE MONITORING IN DIABETIC KETOACIDOSIS USING GLUCOSE MONITOR GM-1320

Wangnoo SK\*, Zargar AH, Mattoo V, Reshi AR, F. Ahmed Dar.

Diabetic Ketoacidosis (DKA) is not an uncommon complication of Diabetes Mellitus. An occurrence rate of 30 to 46 per 10,000 diabetic population has been reported<sup>1,2</sup>. Though mortality rate is still high (upto 20%), a downtrend has been recently reported (1-9%)<sup>1,3,4</sup> largely because of more rational use of insulin, fluids and alkali therapy and frequent blood sugar monitoring.

Continuous blood glucose monitoring became available in the sixties with the availability of modified technicon auto-analysers incorporating double lumen catheter permitting extracorporeal heparinization. This technique was extensively used to demonstrate the true quality of blood glucose control, attained in patients on conventional insulin therapy.

We report our observations on eight patients of DKA admitted at SKIMS in last four years.  
*Key Words* : Diabetic Ketoacidosis, Glucose monitoring

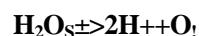
### Material and Methods

Eight hundred patients of diabetes mellitus were seen in last four years. Out of these 8 patients developed DKA or presented for the first time as DKA. The criteria for diagnosing DKA were : (1) Hyperglycaemia (blood glucose levels greater than 17 mmol/l), (2) Ketonuria and (3) Metabolic acidosis (PH below 7.2 and/or plasma bicarbonate levels less than 9m Eq/L). Following parameters were recorded :

(1) Duration of diabetes prior to hospitalization (2) Precipitating factors (3) Clinical findings at admission (4) Bio-chemical parameters at admission and during therapy (5) Type of glucose monitoring employed and (6) Hospital course of the patients.

Continuous glucose monitoring was possible in three patients using Glucose Monitor GM-1320. Glucose Monitor GM-1320 (Kyoto—Daiichi, Japan), performs continuous measurements on blood glucose level by sampling blood continuously from patients with a double lumen catheter. The sampling volume was adjusted to 3ml/hour. The results are displayed with concentration value and concentration curve at the same time.

The blood specimen collected while being diluted with heparinized saline by means of the double lumen catheter connected to the patient is mixed with isotonic buffer solution to deliver to the glucose electrode. The glucose constituent in specimen together with water and oxygen is reacted by glucose oxidase (GOD) immobilized enzyme membrane attached to the glucose electrode. The generated H<sub>2</sub>O<sub>2</sub> is oxidised on the platinum surface of electrode and an electric current proportional to the concentration of glucose is then sought by measuring the electric current :



---

Department of Endocrinology, SKIMS, Soura, Srinagar J&K

## Results

The clinical and biochemical data of these patients on admission are given in Table 1.

## Management

The treatment was directed towards correction of fluids and electrolyte imbalance, hyperglycaemia, acidosis and precipitating factors :

a) *Fluids and Electrolyte treatment replacement :*

Our patients generally received 1 litre of 0.15mol/L NaCl during the initial hour followed by 0.5 litre hourly during the following 8 hours depending upon the clinical state of the patient. When blood glucose concentration decreased to below 14 mmol/L the normal saline was changed to a 5% glucose solution in order

**Table 1**  
**Clinical and Biochemical Details of Light Patients of DKA**

Name	Age/Sex	Duration of Diabetes	PPT Factors	Blood Sugar on Admission (mg/dl)	Coma	Ketone in Urine	HC03/Ph	Total Insulin 1st 24 HR
SN	17/F	5 Years	Missing insulin dose	580	GR I	++	—	90 Units
GJ	26/M	10 Years	post operative (pylo lithotomy)	221	GR I	++	2.0/6.9	200,,
SJ	35/F	8 Years	U.T.I.	359	GR I	++	—	180,,
KM	2i/F	First time detected	U.R.T.I.	310	GR II	++	2.7/7.04	40,,
DR	55/M	10 Years	Acute diarrhoeal illness & missing insulin dose	282	GR I	++	2.5/7.2	18,,
WM	14/M	6 Years	U.T.I.	470	GR II	++	7.0/7.2	100,,
DL	50/M	5 Years	Acute diarrhoeal illness	520	GR III	++	7.0/7.2	80,,
SM	40/M	First time detected	Acute pancreatitis	740	GR II	++	8.0/7.1	80,,

to prevent the development of hypoglycaemia. Potassium was replaced at rates of 10-30 mmol/h according to frequently measured serum potassium concentration.

b) *Insulin treatment :*

Low doses of insulin were used at rates between 5&10 u/hour IV, preceded by a single loading dose of 0.2U/kg body weight for the correction of hyperglycaemia and acidosis.

c) *Bicarbonate replacement:*

Alkali treatment was instituted only in the presence of severe acidosis when PH fell below

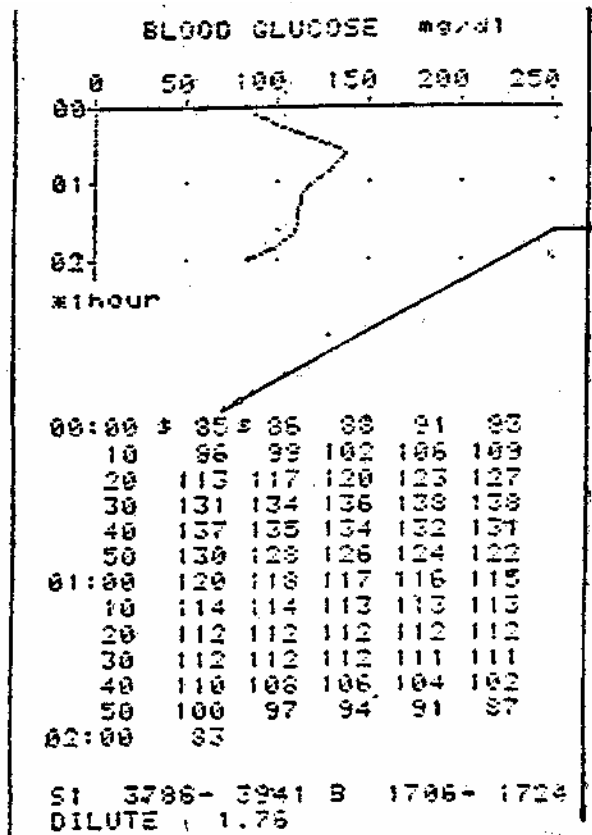
7.1. None of our patients received phosphate replacement.

d) *Antibiotics were used empirically in all the patients.*

e) *Glucose monitoring :*

Three patients were put on continuous glucose monitoring while in rest the monitoring was done by reflectance photometer employing glucose oxidation technique (Reflocheck, Boehringer-Knoll Ltd). A representative tracing of continuous glucose monitoring is shown in Fig. 1.

Fig. 1. [Example of graphic printout in ordinary measurement (in case the chart speed is 1 cm/h)]



The data marked with symbol \$ at its left side shows it was during calibrating

## Treatment Outcome

Of the 8 patients, two died. Both developed altered sensorium and neuro-deficits prior to demise and cause of death was attributed to cerebro-vascular accident.

## Discussion

Data on 8 consecutive patients of DKA were analysed. Three out of these were NIDDM. IDDM is seen infrequently in this part of the world as reported previously by other authorities<sup>5</sup>.

Infection was documented in three out of these patients. Infection is an important precipitating factor in DKA and has been reported in upto 50% of patients of DKA<sup>4,6</sup>.

Omission of insulin is a rare cause of DKA in Western population. But due to peculiar socio-economic conditions prevailing in this part of the world (poverty social milieu and poor education) this is an important precipitating factor and was seen in 25% of our patients.

Initial clinical and laboratory parameters that have been related to subsequent mortality in DKA include hypotension, higher initial blood sugars, hyperkalemia and severity of acidosis<sup>6</sup>.

In our two patients who died, there were no differences in these parameters from the survivor group. This is most likely due to small patient sample in our study.

Three of our patients were put on continuous glucose monitoring using GM-1320. This obviated the necessity for frequent blood sugar monitoring. Furthermore, the inbuilt alarm system in the instrument alerts the physician to modify insulin or dextrose therapy instantaneously and has the potential to prevent morbidity and mortality from hypoglycemia.

Though our study population has been small, it is pertinent to note that none of the patients who died were on continuous glucose monitor. Although in these patients mortality could not be related to excessively high or low blood sugars it is obvious that continuous glucose monitoring offers a giant leap forward in the management of patients of DKA particularly in centres where facility of artificial pancreas is not available.

## References

1. Schade DS, Eaton RP, Alberti KGMM, Johnston DG Diabetic Coma, ketoacidotic and hyperosmolar. University of New Mexico Press. Albuquerque, NM, USA 1981.
2. Faich GA, Fishbein HA, Ellis SE The epidemiology of diabetic acidosis : A population-based study. *Am J Epidemiol* 1983; 117: 551-558.
3. Berger W Die Letalitat des Coma diabeticum am Kantonsspital Basal in den zwei aufeinander-folgenden Beobachtungs-perioden 1968-1973 and 1973-1978 mit konventioneller Insulintherapie und Behandlung mit niederen Insulindosen. *Schweiz Med Wochenschr* 1979; 109 : 1820-1824.
4. Hockaday TDK, Alberti KGMM Diabetic coma, *din Endocrinol Metabol* 1979; 751-788.
5. Podolsky S., Viswanathan M. (Eds) : Secondary Diabetes : The spectrum of the Diabetic syndrome. Raven Press, New York 1980.
6. Keller U, Berger W, Ritz R, Truog P Course and prognosis of 86 episodes of diabetic coma. A five year experience with a uniform schedule of treatment, *Diabetologia* 11 : 1975; 93-100.
7. Teutsch SM, Herman WH, Dwyer DM Mortality among diabetic patients using continuous subcutaneous insulin pumps. *N Engl Med* 1984 310 : 361-368.