CONTINUOUS GLUCOSE MONITORING IN DIABETIC COMA USING GLUCOSE MONITOR-GM 1320

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In the sixties, continuous blood glucose monitoring was made available thanks to the pioneering work of Ferrari, Bonnafe, Weller and others, who combined the double lumen catheter, permitting extracorporeal heparinization, with the modified technicon Autoanalyser. This new technique was used extensively by physicians like Molnar and service in the United States, Mirouze in France and Kruse-Jarres in Germany, to demonstrate the true quality of blood glucose control, attained in the individual patient by conventional insulin therapy. We have also used Glucose monitor-GM 1320 in patients of diabetic ketoacidosis.

The terms DKA, precoma and coma are used somewhat interchangeably to describe differing degrees of acute decompensation of diabetes of which clinically the cardinal features are dehydration and alterations in the sensorium. The biochemical hallmarks of the disorder are hyperglycaemia (Blood glucose levels usually greater than 17 mmol/L); Ketonaemia (Plasma ketone levels usually greater than 5 mmol/L) and acidosis (plasma bicarbonate levels usually less than 9 mEg/L or pH below 7.2).

The incidence of DKA has been relatively constant in Western countries during the past decades despite an improvement in general medical care (Schade DS, et al, 1981). A recent epidemiological survey indicated an annual incidence of 46 cases per 10,000 diabetic subjects (Faich GA, et al, 1983). Our hospital data has shown an overall incidence of 8 cases per 800 diabetic subjects admitted during last 3 years.

Mortality rates ranged from 1 to 19% in different hospitals and countries in recent years (Berger. W, 1979). Although the mortality of diabetic coma has been reported to have declined in the past years, its average rate of 14% has remained high.

Diabetic ketoacidosis develops in a milieu of severe insulin deficiency and excess levels of counter regulatory hormones. While the respective contribution of these factors is the subject of controversy, insulin deficiency perse is an essential factor for the development of ketoacidosis, although insulin antagonism may accelerate its progress or compound its severity.

CLINICAL PRESENTATION;

The clinical and biochemical data on 8 patients of DKA admitted at SKIMS it summarised in the following table:

Name	Age/Sex	Duration Of Diabetes	Ppt. factor	Blood sugar on admissio (mg/dl)	Coma \	Keton in urine	HC03/pH	Total Insulin 1st 24 hrs.
SN	17/F	5 years	Missing Insulin dose	580	GrI	+ +		90 units
GJ	26/M	10 years	Post operative (Pyelolithot my)	221	GrI	+ +	2.0/6.9	200 units
SJ	35/F	8 years	U.T.I.	359	GrI	+ +	—	180 units
KM	2/F	Recent onset	URTI	310	GrII	+ +	2.7/7.04	40 units
DR	55/M	10 years	Acute diarrhoeal & Missing Insulin dose	282	GrI	+ +	2.5/7.2	180 units
WM	14/M	6 years	un	470	GrII	+ +	7.0/7.2	100 units
DL	50/M	5 years	Acute diarrhoeal illness		Grill	+ +	7.0/7.2	80 units
SM	40/M	Recent onset	Acute pancreatitis	520	GrII	+ +	8.0/7.1	80 units

Treatment Goals (Details shall be discussed)

The treatment was directed towards correction of fluids and electrolyte imbalance, Hyperglycaemia, acidosis and precipitating factors. Some patients were put on glucose monitor GM 1320 as soon as the biochemical diagnosis was achieved. Glucose monitor GM 1320 (Kyoto-Daiichi; Japan) performs continuous measurements on blood glucose level by sampling blood continuously from patient with a double lumen catheter. The sampling volume can be adjusted (1—5ml/h) according to the patients condition or the sampling purpose. The automatic calibration with internal standard solution in every 2 minutes provides highly reliable results. The results are displayed with concentration value and concentration curve at the same time, and the pattern for the blood glucose variation can be observed directly. The omission of data in case of power suspension is prevented by employing the memory back up circuit. An alarm sounds when the blood glucose value exceeds the pre set upper or lower limit 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 con-

stituent in specimen together with water and oxygen is reacted by glucose oxidase (GOD) immobilized enzyme membrane attached to the glucose electrode. The generated H 202 is oxidised on the platinum surface of electrode and an electric current proportional to the concentration of H2 02 flows in the electrode. The concentration of glucose is then sought by measuring the electric current (H2 02->2H+ +2).

The correction of hyperglycaemia was achieved by intermittent low dose. Insulin therapy according to the concentration of glucose as observed continuously on the glucose monitor. The use of glucose monitor in such circumstances avoids the trouble of frequent sampling for glucose analysis and development of hypoglycaemia. The adequacy of the treatment can be judged according to the rate of fall of glucose per hour. With most centres in our country, lacking facility of artificial endocrine pancreas for the management of DKA, glucose monitor to some extent minimises the problem of frequent blood glucose estimation in conditions like DKA, diabetic patients during and after operation, for evaluation of insulin doses for S/C therapy and during delivery of diabetic mothers. Stable and reliable results are obtained even in long time monitoring, as the variation of dilution ratio in the catheter with lapse of time can be compensated, with the catheter connected to patient, by employing the calibration method for dilution ratio based on double speed system.