

Original Article

Postprandial Glucose as Marker of Glycemic Control in Type 11 Sudanese Diabetics

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Abstract

Objective: To compare the accuracy of fasting blood glucose, two hour post breakfast glucose and three hour post breakfast glucose (FBG, 2hPBG, 3hPBG) in inferring glycemic control as judged by glycated hemoglobin (HbA1c); and to evaluate their association with diabetic complications and medications used.

Method: A comparative cross sectional study was carried at Gaber Abuelez diabetic center, Khartoum. 99 Patients aged 30-70 years, (55% females), with type II diabetes on oral therapy were enrolled at this study. FBG, 2hPBG, 3hPBG were measured three times one-week apart; by the end of the month HbA1c was measured. Patients were evaluated for clinical evidence of complications; drugs used, haemoglobin, serum creatinine and ECG were done.

Main Outcome Measures: Sensitivity, specificity, positive predictive values.

Results: The patients control profile was poor in the majority. Only drugs targeting basal glycemia were used. Correlations among different parameters showed FBG to be strongly correlated with HbA1c ($r=0.601$; $P=0.000$). 2hPBG correlated weakly if at all with HbA1c ($r=0.202$; $p=0.102$) but has good prediction of poor control ($p = 0.000$). 3hPBG correlation with HbA1c ($r=0.547$; $p=0.000$) was less than FBG. 2hPBG, 3hPBG correlated together very well both on single determination and means of the three values ($r=0.912$, 0.900 , $P=0.000$). Correlations with FBG had been less $r=0.830$, 0.841 respectively. Poor correlation was shown between levels of glycemia and long term diabetic complications except for erectile dysfunction ($P=0.035$). When correlated with current oral therapy only measures of basal glycemia correlated significantly, on other hand postprandial glucose and glucose excursions correlated poorly.

Conclusion: Sudanese diabetics should have their postprandial glucose measured, in addition to fasting glucose and/or HbA1c. 2 and 3-hour post breakfast blood glucose can be used alternatively. Medications specifically designed at the management of postprandial hyperglycemia (i.e. repaglinide, acarbose) should be included in the routine treatment of Sudanese diabetics.

Key words: blood glucose, diabetes mellitus, glycemia, Sudan

Introduction

Diabetes is a global epidemic that affects more than 150 millions worldwide, projected to be 300 million⁽¹⁻¹⁰⁾. Diabetes represents the ninth leading cause of hospital admission in Sudan (1.9 %) ⁽¹¹⁾. The prevalence in Sudan was 3.4% (men 3.5%, women 3.4%). Impaired glucose tolerance estimated at 2.9%. Highest in northern Sudan (5.5 %) and low in the western desert -like parts (1.9%) ⁽¹²⁾. The

highest prevalence was seen in Danagla tribe (8.3 %) ⁽¹³⁾. Type II diabetes (non insulin dependant) is increasingly recognized in the young and children ^(14,15). United Kingdom Prospective Diabetes Study (UKPDS) had shown that 50% of patients with type II diabetes had already one or more complications by the time it was diagnosed ⁽¹⁶⁾. Diabetes is progressive. Its diagnosis immediately increases the risk of developing complications ^(8,16-20). Diabetes is

the fourth leading cause of death in US. It doubles the annual mortality compared to non-diabetic (5.4%)^(17,21,22). Life expectancy of a diabetic is decreased by 5-10 years⁽²²⁾.

The management largely depends on diet control and medications classified as: drugs targeting basal glycemia as sulfonylurea, biguanides, glitazones, insulin and drugs that target postprandial glycemia and post-meal glucose excursion as rapid acting insulin (i.e. lispro), insulin secretagogues (i.e. nateglinide, repaglinide), acarbose^(7,8,16,18,20,21,23-25).

This study was carried out to explore the relations among blood glucose levels at different times related to breakfast; fasting (FBG), 2-hour (2HPBG), 3-hour (3HPBG) post breakfast correlated to HbA1c and occurrence of long-term complications of diabetes. In addition to determining the effect of oral antidiabetic on different blood glucose parameters.

Materials and Methods

This study is an outpatient based cross sectional; comparative study. It was carried in the period September 2002 to May 2003, at Gaber Abuelez diabetic center, Khartoum. A total of Ninety-nine Sudanese patients with type II diabetes were enrolled during the study period.

The selected patients had satisfied the selection criteria of being on no-insulin treatment; no change in treatment or life style within 3 months before the study; no concomitant chronic disease or recent acute illness; and willingness to complete the study. Those diagnosed within the previous 6 months and pregnant women were excluded. All had given their informed consent.

Methods of Data Collection

Patients taken randomly were interviewed using a pre-tested and pre-coded questionnaire and check-list for physical examination. Each patient came to the diabetic clinic in the morning (8-8:30) after 10 to 14 hours overnight fast. At the time venous blood

was withdrawn to measure blood glucose. The patient then returned to the clinic to measure blood glucose 2 hours after breakfast (2h) (10-10:30), then 1 hour later (3h) (11:30-12:30). The process was carried out three times (7-14 days apart) over one month period at the end of which venous sample withdrawn and HbA1c was measured according to specified protocol⁽²⁶⁾.

Statistics

Standard procedures were used to calculate the means, SD, SEM and simple correlation coefficients. Plus analysis of variance, χ^2 tests, paired t-test, ANOVA, multivariate logistic, multiple linear regression. The cut off point for good glycemic control was 7% for HbA1c, 6.6 mmol/l (120 mg/dl) for fasting glucose, 160 mg/dl (8.9 mmol/l) for postprandial glucose^(26,27). Accordingly an increase of >2.2 mmol/l (40 mg) in glucose levels 2h/3h after meal was regarded as exaggerated⁽²⁶⁻²⁸⁾.

Results

Of the total 99 enrolled (45% men and 55% women) 68 completed the study. The mean age was 55.66 ± 9.86 , duration of diabetes averaged 8.91 ± 7.31 years. Treatment consisted of diet only in 18.2% of patients, sulfonylurea in 58.6%, metformin in 4.1%, combination of both metformin and sulfonylurea in 19.1%.

Glucose profiles:

Blood glucose averaged 174.12 ± 59.79 mg/dl (9.67 mmol/l) in the fasting state, 2h after breakfast it was 247.1 ± 72.072 mg/dl (13.72 mmol/l) and at 3h was 224.49 ± 76.84 mg/dl. Blood glucose was significantly lower in the fasting than the fed state.

Most patients 79.4% had glucose level of >120 mg/dl before breakfast (FBG), only 20.9% (n=14) showed good control <120 mg/dl. Most patients had blood glucose level >160 mg/dl post meal: 91.2% (62) at 2 h after breakfast, only 8.8% (6) showed satisfactory postprandial control. 87.3% showed

value >160 mg/dl at 3h after breakfast in single determination, whereas mean levels (three readings over one month time) the percentage was 70.6% (48), 29.4% showed value <160 mg/dl (20). When the cut off point for three hours post breakfast was reduced to 140 mg/dl 83.8% (57) of patients recorded blood glucose value more than 140 mg/dl, 16.2% (11) <140 mg/dl. Among patients with FBG less than 120 mg/dl (20.6%), 82% had levels more than 160 mg/dl after 2h, 29.4% had levels more than 160 mg/dl after 3h from breakfast, and percentage went 70% 3h levels more than 140mg/dl.

The average absolute increase in blood glucose 2h, 3h after breakfast (over the fasting blood glucose levels) was 73.0098 ± 40.1855 mg/dl and 50.370 ± 41.846 mg/dl respectively. The frequency of distribution showed that blood glucose excursion 2h after breakfast was >40 mg/dl in 79.4% of patients, where as that of 3h was in 55.4%. The difference in glucose values at 3h and 2h following breakfast averaged 22.6397 ± 33.5477 mg/dl ; favoring the 2h which was higher than 3h glucose in 80% of samples.

HbA1c:

HbA1c averaged $8.474\% \pm 1.97$. We found that 25% of patients had values less than 7%. Among them 47.05% had fasting glucose more than 120 mg/dl, 82% had 2h excursion more than 40 mg/dl. 29.4% had excursion more than 40 mg/dl at 3h post breakfast.

Correlations:

Table (1) and table (2) reports simple correlations among blood glucose levels at different times related to breakfast. That between FBG and 2h was 0.830 for the average means, when compared at single determination it was 0.82 ($p<0.0000$), between FBG and 3h was 0.841, between 2h and 3h approached one in single determination 0.912 ($p<0.0000$) and 0.900 for the

average. All of the correlations were highly significant but that between 2h and 3h and fasting was not particularly strong on average.

Table (3) and table (4) presents simple correlations between HbA1c and blood glucose at different times related to breakfast. The correlation coefficients were significant between fasting and 3 hours post breakfast and HbA1c at $r=0.601$ and 0.547 ($p<0.000$) respectively. The relation between the fasting glucose levels and HbA1c was particularly strong. The correlation between HbA1c and changes with 2 hours after meal was weak but positive correlation was shown at single determination.

Table (5) presents correlations among glucose excursions; it shows the significant increase of glucose from the fasting value at 2h and 3h, and the changes at 3h from the glucose levels at 2h. The correlation between glucose excursion at 2h and 3h was strong $r=0.666$ ($P=0.000$). The excursion between 2h and 3h has half the r value of 2h glucose excursion $r=0.367$ ($P=0.002$).

Negative correlation was found between glucose excursion at 3h post breakfast and the excursion between 2h and 3h $r=0.449$ ($p=0.000$). Paired T test of different blood glucose levels the difference between FBG and 2h post breakfast glucose when grouped and when paired is significant ($t=14.982$, $p=0.000$) CI:95%.

Also the differences between 3h post breakfast glucose and FBG (9.926 ; $p=0.000$); the difference in values of 2h and 3h was significant but t value was remarkably low ($t=5.565$, $p=0.000$). Although differences were significant, 2h increased by 22.64 mg/dl more than 3h glucose, the maximum difference never exceeded 30 mg/dl the lower was 14.59 mg/dl; confidence interval was 95%. In less than 10% 3h glucose was more than 2h glucose. Correlation between the two values was 0.900.

Table (6) shows the relationship among blood glucoses at different times related to breakfast. It presents their value in predicting glycemic control when glycated haemoglobin was taken as standard; control was classified as good when value of HbA1c is less than 7% and poor when it exceeds 8.5%.

Table (7) summaries the main clinical features of diabetes including different glucose parameters correlated to different types of treatment, duration in males averaged 11.4 years, that in females was shorter with mean of 6.91 years.

Table 1: Simple correlation of blood glucose levels at different times related to breakfast in non-insulin treated type II diabetes – (single determination)-Khartoum 2003

Blood glucose (N=97)	Correlation	P value
FBG vs. 2hPBG	0.820♣♣	0.000
3hPBG vs. FBG	0.727♣♣	0.000
3hPBG vs. 2hPBG	0.912♣♣	0.000

Table 2: Simple correlation of blood glucose levels at different times related to breakfast in non-insulin treated type II diabetes – (mean values)Khartoum -2003

Blood glucose (N=68)	Correlation	P value
FBG vs. 2hPBG	0.830♣♣	0.000
3hPBG vs. FBG	0.841♣♣	0.000
3hPBG vs. 2hPBG	0.900♣♣	0.000

Table 3: Simple correlation between HbA1c and blood glucose levels at different times related to breakfast in non-insulin treated type II diabetes (Single determination)–Khartoum -2003

Blood glucose (N=97)	Correlation	P value
HbA1c vs. FBG	0.584♣♣	0.000
HbA1c vs. 3hPBG	0.649♣♣	0.000
HbA1c vs. 2hPBG	0.669♣♣	0.000

Table 4: Simple correlation between HbA1c and blood glucose levels at different times related to breakfast in non-insulin treated type II diabetes (mean values) –Khartoum -2003

Blood glucose (N=68)	correlation	P value
HbA1c vs. FBG	0.601♣♣	0.000
HbA1c vs. 2hPBG	0.202	0.102
HbA1c vs. 3hPBG	0.547♣♣	0.000

♣♣ Correlation is significant at 0.01 level (2-tailed) .Means were used

To determine the values. Correlation is significant at the 0.01 level (2-tailed): FBG: Fasting blood glucose, 2hPBG: Two hour post breakfast blood glucose. 3hPBG: Three-hour post breakfast blood glucose.

Table 5: Glucose excursions in non-insulin treated type II diabetics Khartoum 2003

Glucose excursions relations	Correlations:	P value
At 2 hours (FBG–2hPBG) vs. 3 hours (FBG-3hPBG)	0.666	0.000
2 hours vs. (3hPBG-2hPBG)	0.367	0.002
3 hours (3 hPBG-FBG) vs. (3h BG-2hPBG)	-0.449	0.000

Correlation is significant at 0.01 level (2-tailed). FBG: Fasting blood glucose, 2hPBG/3hPBG: Two/Three hour post breakfast blood glucose

Table 6: The relationships between blood glucoses at different times related to breakfast in non-insulin treated type II diabetics – Khartoum 2003

HbA1c (n=67)	Less than 7% (n=17)	7–8.5% (n=20)	More than 8.5% (n=30)	Tests	P
FBG <120mg/dl	9 (52.9%)	3 (15%)	2 (6.66%)	Pearson χ^2	0.000
FBG >120mg/dl	8(47.88)	17 (85%)	28 (93.3%)	Likelihood ratio	0.000
				Linear/linear association	
2 h excursion				Pearson χ^2	0.251
<40 mg/dl	3(17.64%)	2 (10%)	1 (3.33%)	Likelihood ratio	0.248
>40 mg/dl	14(82.35%)	18(90%)	25(96.66%)	Linear/linear association	0.599
3h Excursion				Pearson χ^2	0.000
<40	12(70.58%)	6 (30%)	2 (6.66%)	Likelihood ratio	0.000
>40	5 (29.4%)	14 (70%)	28 93.33%	Linear/linear association	0.000

Correlation is significant at 0.01 level (2-tailed). FBG: Fasting blood glucose, 2hPBG: Two hour post breakfast blood glucose.3hPBG: Three hour post breakfast blood glucose.HbA1c: glycated hemoglobin

Table 7: Main features and glucose parameters correlated to treatment in non-insulin treated type II diabetic patients -Khartoum 2003(N=68):

Glucose Parameter	Treatment								
	Diet		SU		MET		MET +SU		P
AGE (Years)	55.86	510	56.84	55.85	60	51	59	53.56	0.630
Duration of diabetes (Years)	8.14	5.6	11.92	6.84	1.5	4	14.4	10.67	0.021
BMI	25.8441	29.6836	24.9482	28.014	25.6029	29.5129	27.92	30.3581	0.352
FBG mg/dl	107.300	144.556	173.850	173.850	158.25	295.33	197.417	198.2	0.021
2HBG mg/dl	175.967	202.83	246.158	252.758	262	390.000	260.28	261.983	0.035
3HBG mg/dl	138.987	183.722	223.67	23.4.652	241.083	293.333	234	250.685	0.049
Hb A1c %	5.760	8.233%	8.62	8.436	8.9	9.6	9.35	9.225	0.024

BMI: body mass index. FBG: Fasting blood glucose. 2hPBG/3hPBG: 2/3hour post breakfast blood glucose. HbA1c: glycated hemoglobin. SU: sulfonylurea. MET: Metformin. Correlation is significant at 0.01 level (2-tailed).

Discussion

Most diabetic patients with apparently good glycemic control as inferred from HbA1c <7% or by fasting <6.6% (120 mg/dl) had indeed high glucose levels after meals and/or exaggerated glucose excursions following meals reaching unexpectedly high levels. In these patients, one should consider using medications that are particularly effective in blunting postprandial glucose bursts. The study indicated that monitoring glycemic control and efficacy of treatment could not be restricted to fasting glucose and HbA1c. Both of which are poor indicator of blood glucose levels at different times of the day especially postprandial (29-31).

Three hours post breakfast glucose, which was not assessed on its own in similar studies, has proved a

significant association with fasting, 2h post breakfast and HbA1c. Its relation to 2h was particularly strong at single determination, however less strong when the means of the two tests were compared (0.912, 0.900 respectively). When means of values were compared, the association was mildly stronger with the fasting values. Glucose excursion at 3 hours was significant and high with similar values and strength as that shown by Bonora et al (28) at 3h post lunch and 3h post dinner. Its significance is comparable to that obtained at similar timing shown by Lerman et al (prelunch) (31). 3h post breakfast glucose could be an additive armamentarium in the array used in following glycemic control and could be used as an alternative to 2h and fasting, further studies are needed however.

Avington⁽³⁰⁾ and A French study had proved the strength of extended postprandial glucose (5-hour post lunch) in predicting glycemic control and had shown it to be similar in power to 2h post meal. Our study signaled out another option that is near in timing to the gold standard test preprandial (prelunch)^(1,27,31). Controlling fasting glucose alone ameliorates HbA1c only partially as in UKPDS⁽³²⁾, whereas correcting glucose all throughout the day resulted in greater reduction of HbA1c as in DCCT⁽³³⁾ and the Kumamoto study⁽³⁴⁾. The differences between the two studied group was 1% in UKPDS and 2% in DCCT and Kumamoto.

These results pointed out that glucose levels in the post breakfast states are not merely drift of fasting (prebreakfast) glucose but reflect the ability of the beta cells of the pancreas to mount an early phase (burst) of insulin secretion in response to food intake. Early insulin release is known to be impaired in the majority of diabetics and that bolus-basal (injection-basal) regimen is probably the best method to ensure optimal glycemic control^(35,36).

We found HbA1c to be better correlated with fasting ($r=0.601$; $P=0.000$); similar to the result shown by Attbani ($r=0.634$; P less than 0.001)⁽³⁷⁾.

Three hours post breakfast glucose correlated well with HbA1c but less than that with the fasting glucose ($r=0.547$; $p=0.000$). Despite the strong correlation seen on single determination of blood glucose levels, HbA1c correlation to postprandial glucose mean levels is poor ($r=0.202$; $p=0.102$). This is hard to explain. It varies with results shown by Avington⁽³⁰⁾ and Lerman⁽³¹⁾ and others^(26,27). However it is in agreement with Bonora⁽²⁸⁾. It is also consistent with the conclusion reached by panel of experts designed by ADA to review the available data on postprandial glucose⁽³⁸⁾ and goes with Unger statement⁽²¹⁾.

On multivariate regression analyses, all glucose parameters were independent predictors of glycated

haemoglobin. The most constant was 3 h postbreakfast glucose which showed strong correlation when compared alone ($P=0.00000$), with fasting ($P=0.000$), with 2 hour (0.008) and when the three compared together only the 3h was significant ($P=0.004$). Individually both fasting and 2h postprandial glucose showed significant power in prediction of HbA1c; ($p=0.00$; 0.043 respectively). The correlation with 2h postmeal was marginally significant. Possible explanation is that more hours are spent in the interprandial and nocturnal periods than in the postprandial phases; most Sudanese confine themselves to three meals. Consequently the average daily blood glucose, which is the main determinant of the extent of the glycation process, is a function more of interprandial and nocturnal glucose levels than of glucose spikes after meals⁽²⁸⁾.

Data from the National Health and Nutrition Examination Survey Studies (NHANES) and IOEZ⁽³⁹⁻⁴¹⁾ based on medications targeting postprandial glucose in patients who were not given basal medications yielded a reduction in postprandial glucose but did not substantially change HbA1c. However when the two regimens were given they resulted in lower value of HbA1c⁽³⁹⁻⁴³⁾.

Thus assessment of HbA1c is poorly informative of the degree of postprandial glucose content. However, postprandial glucose demonstrated well the degree of poor control.

Recent studies had shown that postprandial glucose level might exert stronger deleterious effect on the cardiovascular system than does fasting glucose⁽³⁵⁻⁴⁴⁾. This had been further substantiated by the fact that when glucose control aimed at normalizing fasting glucose alone, as in UKPDS, the effect on macroangiopathy was minimal^(32,45).

Our finding that HbA1c is essentially dependant on FBG might explain the minimal effect of controlling HbA1c on CVD as in UKPDS or

Veteran Administration Cooperative Study⁽⁴⁶⁾. Whereas targeting postprandial glucose, as in the Kumamoto or DIGAMI study⁽⁴⁷⁾ had gained better cardiovascular outcome. Furthermore, numerous observational studies had documented the increased risk of CVD associated with postprandial and/or post challenge glucose⁽⁴⁴⁻⁵³⁾. Several experimental trials had supported the injurious effect of postprandial glucose on arterial wall and its proatherogenic properties^(35,50-51). More studies are awaited to clarify the subject; whether HbA1c suffice or postprandial should take the lead as goal of therapy, at least to prevent cardiovascular disease (CVD).

Comparing different glucose parameters and main clinical features with the type of treatment revealed that gender, duration of diabetes were major factors associated with the type of treatment but not the age or BMI, much in agreement with Bonora et al⁽²⁸⁾. Fasting blood glucose, HbA1c, 2h post breakfast glucose, 3h post breakfast glucose had shown significant correlations (P values, in order, were: 0.021, 0.021, 0.024, 0.035, 0.049).

The type of treatment showed marginal significance with 2h and 3h; the relation with fasting, HbA1c and duration of diabetes was strongly significant. The latter pointed to the progressive nature of type II diabetes and stressed the insignificance of anthropometric measures like BMI in the evaluation of glycemic control.

BMI tends to increase due to gain in weight exerted by most of medications used in diabetes especially sulphonylurea.

The fact that postprandial glucose levels were poorly correlated to the type of treatment given to the patient in contrast to fasting glucose and HbA1c (almost double P value) supports the innovation that the latter two are better correlated together and affect each other. It also elucidates that they were not strongly related to post meal glucose levels

and/or glucose excursion. It further strengthens the accumulating evidence that if post meal glucose is not targeted on its own, its control would be poor and that specified drugs are needed for optimal glycemic control⁽⁵⁴⁻⁵⁶⁾.

The relation between 2h and 3h postprandial glucose is close. Diet like drugs poorly controlled postprandial glucose, which further strengthens the conclusion above.

Gender had a great impact on diabetes, female diabetics have poor prognosis^(7,8,20,21) females in all treatment groups were definitely overweight mean BMI \pm 28 kg/m², had worse glycemic control despite the shorter duration of diabetes mean 6.91 years compared to 11.41 in the male group. The group of metformin and combined treatment had poorer glycemic control, more BMI values clearer in male group; this could be explained by late administration of these drugs in a group with prolonged duration of a progressive disease.

The controversy in correlation between single day reading of 2-hour glucose and means of three readings corresponds with the simple fact that blood glucose levels vary day by day. Good evidence exists to support that several glucose determinations over a period of several weeks are better correlated to HbA1c than a single or few determinations in one day⁽⁵⁷⁾.

The positivity of correlation on single determination with 2-hour postprandial glucose and lack of correlation with the average values may be explained, at least in part by that patients during study period had complied better with treatment and adhered closer to diet.

On average metabolic control was poor in most of the patients, this consisted with data shown by ElMahadi and others^(58,59).

Considerable proportion of patients, many of whom showing satisfactory HbA1c level, indeed had poor glycemic control after meals. Because postprandial

hyperglycemia is an independent risk factor for CVD in type II diabetes specific periodic assessment of postprandial glucose seems to be warranted along with FBG.

The significant position of 3 hour post breakfast glucose signaled out in our study is an interesting finding. It could be extrapolated for 2 hours and/or fasting glucose in monitoring glycemic control. We suggested 140mg/dl as an upper limit. ADA recommended 140 mg/dl level for the 2 hours postprandial⁽¹⁾.

The main results of the present study are that:

The majority of non-insulin treated type II diabetics in Sudan had higher than recommended blood glucose levels and exaggerated glucose excursion after meals.

High postprandial glucose was often found when long-term control and fasting glucose were satisfactory. Three-hour post breakfast glucose is strongly correlated to fasting, two hour and HbA1c. 2 hour post breakfast demonstrated poor correlation with the level of HbA1c. In prediction of glycemic control both fasting and 3h postbreakfast had showed to be sensitive in reflecting both good and poor control. 2hPBG was poor predictor of good control but slightly stronger than both fasting and three hour post breakfast in predicting poor control ; HbA1c>8.5% .

BMI is poor indicator of glycemic control. Multiple regression analysis showed no significant association between diabetic complications and different parameters of glycemic control used in the study.

Glucose monitoring in type II diabetes seems to be more complex than previously thought, because Fasting blood glucose is a rather poor index of glucose levels throughout the day.

HbA1c seems to provide poor information on postprandial glucose levels and it provides no

information on glucose excursion with meals in Sudanese patients.

Remarkable proportion of patients with type 2 diabetes has poor glucose control in the postprandial period even when HbA1c is satisfactory. They might benefit from medications specifically suited for providing more physiological insulin after meal i.e. oral (injection)-basal therapy. Comprehensive management should entail monitoring not only FBG and/or HbA1c level but also glucose at other times especially in the postprandial period. 2 hours has been extensively studied, 3 hours is an attractive option. Patients should not be denied the test for postprandial glucose if they show later than the précised 2 hours. 2-3 hours is a more flexible goal. More studies are awaited. Home glucose monitoring will make adherence easy.

In prevention of diabetic complications, controlling postprandial hyperglycemia is at least as important as controlling fasting glycemia. Stricter control of postprandial glucose using specified drugs is likely to be useful for better control of glycemia and might result in better outcome in type II diabetes.

The management of diabetic patients in Sudan is poor; adherence to guidelines is lacking and intensive health education is needed.

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