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Research Article

Effect of Initial Dietary Dose on Therapeutic Response and Quality of Life in Diabetes Mellitus

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Abstract

Introduction: Progressively increasing prevalence of Diabetes mellitus in India for which India is known as World Diabetic capital, is solely due to increasing non-nutrients in dietary constituents which alters body metabolism by affecting hepatic, pancreatic and incretin biokinetics adjunct with stress due to changed life style.

In spite of modalities for early detection, diagnosis and advancement in therapeutics, quality of life remain at stake and most encumbrance.

Objective of Study: To ascertain the effect of low caloric diet as initial diet on therapeutic outcome of diabetes mellitus.

Material and Method: 1000 patients of diabetes mellitus attending Centre for Endocrinology and Metabolism, Aarogyam punarjeevan, Ara Garden Road, Jagdeopath, Patna 14 and Institute of applied endocrinology, National Institute of Health and Research, Warisaliganj (Nawada) Bihar been considered for the proposed study.

After due confirmation of their diabetic status and basic haemato-hepato-renal parameters patients were divided in to two equal group and one group were advocated < 100 calories diet while other more than 100 calories, with Oral hypoglycaemics to control blood sugar < 200 mg.

Result: The present study affirm that initial diet of < 100 calories prompt optimal incretin function and ensure blood sugar bioregulation, as this ensure optimal insulin secretion and insulin receptor sensitivity, thus attain sustained and progressive glycaemic control without any drug adversity, as no patients of this group shows post prandial blood sugar rise > 60 mg% and 165 (33%) patient show post prandial blood sugar even less than fasting blood sugar. While other group majority patients show post prandial blood sugar surge > 60 mg.

Conclusion: Hence optimal initial diet (< 100 calories) ensure better diabetes mellitus control and checks diabetic sequel.

Keywords: Diabetes Mellitus; Non-Nutrients; Hepatic; Pancreatic; Incretin; Insulin Receptor; Post Prandial Blood Sugar; Glycaemic Control

Introduction

Diabetes mellitus is progressively increasing worldwide and India is considered as diabetes capital of world with projected incidence of 109 million by 2035.

Even IDF doubts 347 million cases of Diabetes mellitus and WHO too recommendation to reassess diet and recommendation of physical activity to curb Diabetes mellitus.

Diabetes mellitus is reaching potentially endemic progression in India. The level of morbidity and mortality due to diabetes mellitus and its potential sequel is also grave.

Diabetes mellitus is the commonest metabolic disorder due to emergence on dietary non-nutrients which competes with various enzymes in the body and alters metabolic process compromising both hepatic and pancreatic function and presents with hyperglycaemia and hyper lipidaemia [1-10].

In addition increased tolerability of patients to even very high blood glucose level reflects altered metabolic process than mere disturbed endocrinal function [11,12].

Restriction in Carbohydrate up to 45% results in marked decline in HbA1C in 6 months i.e. from 12.6 to 5.6.

Though to evaluate diabetes mellitus HBA1C is the gold standard but still prevails the apprehension of insulin prick, concern over body weight gain and hypoglycaemic presentation.

As food intake induces secretion of gastric inhibitory peptides which increases glucose dependant insulin secretion from the pancreatic β cells, proliferation, inhibit apoptosis and expands pancreatic β cell mass.

GIP enhances post prandial glucagon receptor while GLP1 suppress prandial glucose rise. In addition declined glucose in the hepatic parenchyma facilitate glycogenesis and check gluconeogenesis. Varied post prandial glucose surge due to incretin insufficiency and initial diet more than optimal glucose load (< 1 mM) to induce adequate incretin-insulin response, as supplement of GLP1 analogue and Dipeptidyl peptidase 4 (DPP-4) shows promising result.

Post prandial variation of blood sugar in diabetics even with Oral hypoglycaemic agent Or Insulin supplement suggest variation in status of Incretin function in blood sugar bio regulation [13,14].

Thus to reduce disease burden early detection, awareness, self-management counselling, dietary restriction and suggestions to optimise diabetic care to ensure normoglycemic state and restrict diabetic sequel.

Hence considering the optimum calorific intake required to induce incretin function, a clinical study was carried on to adjudge the efficacy of initial dietary dose on glycaemic control and quality of life in diabetics.

Material and Methods

Design of study: Comparative evaluation.

Objective of Study: To assess the clinical effect of optimal calorific requirement on induction of incretin response in blood sugar bio regulation.

Duration of Study: January 2013 to September 2017

Material

Patients of diabetes mellitus attending at Centre For Endocrinology and Metabolism of Aarogyam Punarjeevan, Ram Bhawan Ara Garden Road Patna [14], Institute of Applied endocrinology, National Institute Of Health and Research were selected for the comparative study.

Methods

Selected patients of diabetes mellitus were thoroughly interrogated for their dietary intake, personal habit and consumed therapeutics. Patients were clinically assessed for diabetes mellitus related sequel and investigated for fasting and post prandial blood sugar, lipid profile, renal and hepatic profile.

Patients were evaluated for post therapy blood sugar level (both fasting, PP and HbA1C), urine albumin, ketone every 15th day, while lipid profile was repeated every 3 month. To ascertain safety profile patients were kept under strict vigil watch for any alteration in haemato- hepatic and renal profile.

Clinical severity of the patients was indexed as per their fasting and post prandial blood sugar.

Grades	Fasting blood sugar	Post prandial blood sugar
Mild	120 - 150	200 - 250
Moderate	150 - 200	250 - 300
Severe	> 200	> 400

Patients were classified in to two groups comprising equal number of patients and were advocated as per following:

- Group A: 100 calories (25 gram cereals) as initial diet
- Group B: Initial diet > 100 Calories (Cereals > 25 gm)

Both groups been advocated.

Calorific requirement as per blood sugar status to keep the body weight as IBW (Ideal body weight).

Antidiabetic drug: Based on their fasting and post prandial blood sugar level Glimepiride and Metformin combination.

Blood sugar modulator in both cases ayurvedic combo (Meta Reg 1 cap 30 minutes before breakfast, lunch and dinner).

Anti-diabetic therapy been modulated to bio regulate blood sugar level < 200 mg without any variation.

Clinical response was graded as:

Grade	Characteristics
I	Patients achieving fasting, post prandial blood sugar within normal range without any adversity.
II	Patients having marked decline in blood sugar with post prandial rise of < 50 mg which ascertain bioregulation.
III	Decline in blood sugar but not under normal level Or normal Post prandial rise without any adversity.

Results

Selected patients were of age > 28 years and out of all 36.6% patients were of age group 30 - 40 years while 6.9% were of age group 25 - 30 years and 5.9% of > 60 years age (Table 1).

Out of all 60% and 40% were male and female respectively (Figure 1).

Figure 1: Pie diagram showing male-female composition.

Age group (in years)	Number of patients		
	Male	Female	Total
25 - 30	41	28	69
30 - 35	88	62	150
35 - 40	106	78	184
40 - 45	108	74	182
45 - 50	102	76	178
50 - 55	96	41	137
55 - 60	21	20	41
> 60	38	21	59

Table 1: Age and sex wise distribution of patients.

Out of all majority 24.5% were having fasting blood sugar 140 - 150 mg% while 32.4% were with post prandial blood sugar 300 - 350 mg%, 15% were with fasting blood sugar > 200 mg and 10.6% with post prandial blood sugar > 500 mg% (Table 2).

Diabetic parameters	Number of patients
HbA1C	
6 - 8	112
8 - 10	228
10 - 12	519
> 12	146
Blood Sugar (mg%)	
Fasting	
120 - 130	76
130 - 140	124
140 - 150	243
150 - 160	168
160 - 170	115
170 - 180	56
180 - 190	38
190 - 200	28
> 200	152
Post prandial	
200 - 250	58
250 - 300	124
300 - 350	324
350 - 400	136
400 - 450	156
450 - 500	96
> 500	106

Table 2: Distribution of patients as per their diabetic parameters.

Based on fasting and post prandial blood sugar and HbA1C level patients were classified as mild, moderate and severe grade of severity, out of all 66%patients were of severe grade (Figure 2).

Figure 2: Bar diagram showing distribution as per degree of severity.

Out of all 24.9% patients had haemoglobin < 10 gm% while 1.5% patients had serum bilirubin > 1 mg% and 1% SGOT and SGPT > 30 IU, 23.8% were with serum cholesterol > 200 mg and 0.5% with blood urea > 26 mg% and serum creatinine > 1.5 mg with urine positive for albumin and RBCs (Table 3).

Particulars	Number of patients	%
Haematological		
Haemoglobin		
< 10 gm %	249	24.9
>10 gm %	751	75.1
Hepatic profile		
Serum bilirubin		
< 1 mg %	985	98.5
> 1 mg %	15	1.5
SGOT		
< 30 IU	990	99
> 30 IU	10	1
SGPT		
< 30 IU	990	99
> 30 IU	10	1
Renal profile		
Serum creatinine		
< 1.5 mg	995	99.5
> 1.5 mg	5	0.5
Blood Urea		
< 26 mg	995	99.5
> 26 mg	5	0.5
Urine		
Albumin		
Present	5	0.5
Absent	995	99.5
RBC		
Absent	995	99.5
Present	5	0.5
Lipid profile		
Total Serum Cholesterol		
< 200 mg	762	76.2
> 200 mg	238	23.8

Table 3: Distribution of patients as per basic bio parameters.

Patients of group A shows sustained and progressive decline in both fasting and post prandial blood sugar without any adversity or post prandial blood sugar surge. While patients of group B shows mild decline in blood sugar but post prandial blood sugar surge was very common (Figure 3).

Figure 3: Graph showing Glycaemic control.

Hyper cholesterol more common in patients of group B than group A patients as no patients of group A shows altered HDL/LDL ratio.

Out of all 67% patients of group A shows post prandial blood sugar rise < 60 mg while 17.6% of group B and 33% of group A shows post prandial blood sugar (Table 4) less than fasting blood sugar and 84.2% of group B show post prandial blood sugar surge > 60 mg (Table 4).

Difference of Post prandial and fasting blood sugar	Number of patients					
	Group A			Group B		
	Male	Female	Total	Male	Female	Total
< 60 mg	201	134	335	52	36	88
> 60 mg	-	-	-	248	164	412
< Fasting	99	66	165	-	-	-

Table 4: Distribution of patients as per post prandial blood sugar surge.

Discussion and Conclusion

Diabetic complication in spite of drug therapy is very rampant either due to non-control of blood glucose level or drug induced circadian variation of blood sugar or drug adversity as persistent blood sugar > 200 mg and variable body blood sugar poses threat of diabetic sequel.

Usually patients are prescribed anti-diabetics i.e. Oral hypoglycaemic agent Or Insulin supplementation without any awareness regarding the disease, dietary intake and life style to bio regulate blood sugar and improve quality of life.

Present study affirming better glycaemic control and check on post prandial blood sugar surge with excellent clinical outcome in patients taking 25 gm of cereal or 100 calories diet as initial intake than patients taking variable diet.

Patients on < 100 calories initial diet show sustained and progressive decline in both fasting and post prandial blood sugar, post prandial blood sugar surge < 60 mg in 67% and < fasting in 33% cases, while other taking > 100 calories initial diet, 82.4% show post prandial surge of > 60 mg.

This clinical effect and superior therapeutic outcome can be explained as- Food intake causes secretion of gastric inhibitory peptides Or commonly known as incretin hormones which enhances glucose dependant insulin secretion from pancreatic β cells and adipose tissue to promote insulin dependant translocation of the Glut-4 glucose transporter to plasma membrane and exclusion of Fox 01 transcription factor from the nucleus in adipocytes. Effect of GIP on adipocytes require action of both cAMP /Protein kinase and phosphoinositol 3 kinase. Gastric inhibitory peptide control adipose insulin sensitivity via activation of cAMP [15-18].

Figure a

GIP and Glucagon like peptide (GLP)are incretin hormones secreted from the intestine on ingestion of glucose or nutrient to stimulate insulin secretion from pancreatic B cells. Both GIP and GLP-1 exert their effect by binding to their specific receptors i.e. GIPR and GLP-1 R which are G protein complex receptors [19].

Receptor binding activate and increases the level of intra cellular cAMP in pancreatic B cells and stimulate insulin secretion.

Optimal Glucose concentration required to trigger GLP1 release is 5 - 100 mM i.e. maximum 25 gm of cereals to provide 18 gm of glucose [20,21].

Figure b

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