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Initial Dietary Dose in Diabetes Mellitus

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Abstract

Progressively rising prevalence of Diabetes mellitus in India established India 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 remains at stake and most encumbrances.

Objective of study: To ascertain the effect of low caloric diet as initial diet on therapeutic out come and quality of life in patients of diabetes mellitus.

Material & method: 1000 patients of diabetes mellitus attending Centre For Endocrinology &Metabolism, Aarogyam Punarjeevan, Ara Garden Road, Jagdeopath, Patna 14 and Institute of applied endocrinology, National Institute of Health & Research, Warisaliganj (Nawada) Bihar been considered for the proposed study. After due confirmation of their diabetic status and basic haemato-hepatorenal parameters patients were divided in to two equal group and one group were advocated <100calories diet while other more than 100 calories, with Oral hypoglycaemic to control blood sugar <200mg.

Result: The present study affirm that initial diet of <100 calories prompt optimal incretin function and ensure blood sugar bio-regulation, 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 >60mg % 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 >60mg, all had grade I clinical response, 99% show FBS <100mg, 93% PPBS- <150mg and HbA1C <6 without any sequel with declined serum cholesterol improved quality of life.

Conclusion: Optimal initial diet (< 100 calories) ensures better control of diabetes mellitus and checks diabetic sequel.

Keywords: Diabetes mellitus; Non nutrients; Hepatic; Pancreatic; Incretin; Insulin receptor; Post prandial blood sugar; Glycaemic control

Abbreviations: WHO: World Health Organization; IDF: International Diabetes Federation; GLP-1: Glucagon Like Peptide-1; GIP: Gluten Immunogenic Peptides; DPP: Dipeptidyl Peptidase; IBW: Ideal Body Weight

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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 of dietary non nutrients which competes with various enzymes in the body and alters metabolic process compromising both hepatic and pancreatic function resulting in 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. Carbohydrate restriction up to 45% results in marked decline in HbA1C in 6 months i.e.- from 12.6 to 5.6 and HBA_1C is considered the gold standard for diabetic evaluation [11,12].

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 facilitates glycogenesis and check gluconeogenesis. Varied post prandial glucose surge due to incretin insufficiency and initial diet more than optimal glucose load (<1mM)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]. To reduce disease burden early detection, awareness, self assessment counselling, dietary restriction and suggestions to optimise diabetic care to ensure normoglycemic state and restrict diabetic sequel. Hence considering the optimum calorific intake required inducing 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 initial diet intake on sugar bio regulation in patients of Diabetes mellitus.

Duration of Study

January 2013 to September 2017, this includes follow up period also.

Material

Patients of diabetes mellitus attending at Centre for Endocrinology & Metabolism of Aarogyam Punarjeevan, Ram Bhawan Ara Garden Road Patna 14, Institute of Applied endocrinology, National Institute of Health & 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 (Table 1).

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

Table 1: Clinical severity of the patients was indexed as per their fasting and posts prandial blood sugar.

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

i. Group A: 100 calories (25 gram cereal) as initial diet
ii. Group B: Initial diet > 100 Calories (Cereal >25 gm)

Both groups were advocated – Calorific requirement as per blood sugar status to keep the body weight as IBW (Ideal body weight).

Anti-diabetic 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 < 200mg without any variation (Table 2).

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 <50mg which ascertain bio regulation.
III	Decline in blood sugar but not under normal level or Normal post prandial rise without any adversity.

Table 2: Clinical response was grading.

Observations

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 3).

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 3: Age and sex wise distribution of patients.

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

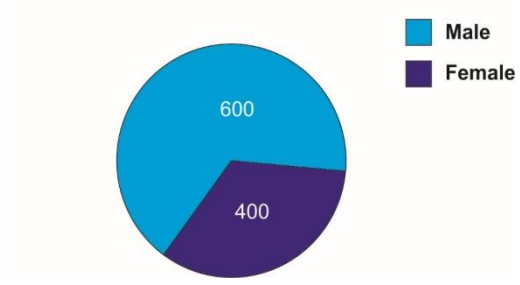

Figure 1: Pie diagram showing Male-Female Composition.

Out of all majority 24.5% were having fasting blood sugar 140-150mg % while 32.4% were with post prandial blood sugar 300-350mg%,15% were with fasting blood sugar >200mg and 10.6% with post prandial blood sugar >500mg% (Table 4).

Diabetic parameters	Number of patients
HbA_1C:	
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 4: 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).

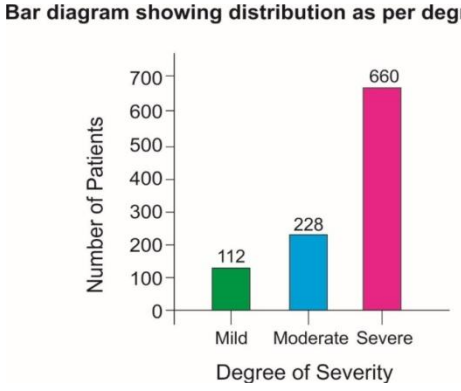

Figure 2: Bar Diagram showing distribution as per degree of severity.

Out of all 24.9% patients had haemoglobin <10gm% while 1.5% patients had serum bilirubin >1mg% and 1% SGOT and SGPT > 30 IU, 23.8% were with serum cholesterol >200mg and 0.5% with blood urea >26mg% and serum creatinine >1.5mg with urine positive for albumin and RBCs (Table 5).

Particulars	Number of Patients	Percentage %
Haematological: Haemoglobin:		
< 10 gm %	249	24.9
>10 gm %	751	75.1
Hepatic profile: Serum bilirubin:		
<1mg%	985	98.5
>1mg%	15	1.5
SGOT:		
<30 IU	990	99.0
>30 IU	10	01
SGPT:		
<30 IU	990	99.0
>30 IU	10	01
Renal profile: Serum creatinine		
<1.5 mg	995	99.5
>1.5 mg	05	0.5
Blood Urea:		
<26mg	995	99.5
>26mg	05	0.5
Urine: Albumin		
Present	05	0.5
Absent	995	99.5
RBC		
Absent	995	99.5
Present	05	0.5
Lipid profile Total Serum Cholesterol		
<200mg	762	76.2
>200mg	238	23.8

Table 5: 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, and corresponding HbA_1C without any adversity or post prandial blood sugar surge. While patients of group B shows mild decline in blood sugar and corresponding HbA_1C but post prandial blood sugar surge was very common (Figure 3).

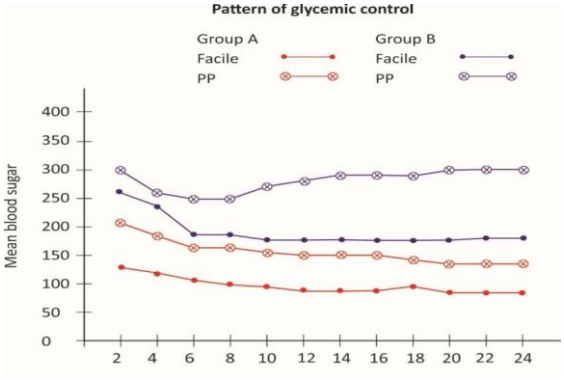

Figure 3: Graph showing Glycaemic control.

Hyper cholesterolemia 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 <60mg while 17.6% of group B and 33 % of group A shows post prandial blood sugar less than fasting blood sugar and 84.2% of group B show post prandial blood sugar surge >60mg (Table 6).

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
>60mg	-	-	-	248	164	412
< Fasting	99	66	165	-	-	-

Table 6: Distribution of patients as per post prandial blood sugar surge.

Among group A 99% patients achieved fasting blood sugar <100mg,93% post prandial blood sugar <150mg and HbA_1C <6 with better quality of life and excellent grade of clinical response in all the cases while of group B none had blood sugar <100mg (Fasting) and <150 (post prandial), HbA_1C <6, increased serum cholesterol >200mg in 62.6%, increased blood urea >26mg and presence of albumin in 0.8%, crippled and agonising life in 55.8% and non had excellent grade of clinical response rather 63.8% shows very poor clinical response (Table 7).

Parameters	Number of Patients			
	Group A	Percentage %	Group B	Percentage %
Blood Sugar: Fasting:				
< 100mg	495	99	-	00
100 - 120mg	05	01	98	19.6
>120mg	-	00	402	80.4
Post prandial:				
<150mg	465	93	-	00
150-200mg	35	07	182	36.4
>200mg	-	00	318	63.6
HbA_1C:				
<6	465	93	-	00
6-7	35	07	182	36.4
>7	-	00	318	63.6
Lipid profile: Serum cholesterol				
<200mg	500	100	187	37.4
>200mg	-	00	313	62.6
Renal profile Blood Urea				
<26mg	500	100	494	99.2
>26mg	-	00	04	0.8
Serum Creatine				
<1.5mg	500	100	500	100
>1.5mg	-	00	-	00
Urine Albumin				
Present	None	00	04	0.8
Absent	500	100	496	99.2
Quality of life				
Crippled and agonising	None	00	279	55.8
Normal & joyous	500	100	221	44.2
Clinical outcome				
Excellent	All	100	-	00
Good	-	00	181	36.2
Poor	-	00	319	63.8

Table 7: Showing outcome of the study.

Discussions

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 > 200mg 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 affirms better glycaemic control i.e.- decline in both fasting and post prandial blood sugar, corresponding HbA_1C 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 >60mg.Majority (97%) attended HbA1C <6 in 3 months therapy.

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 dependent 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 requires action of both cAMP /Protein kinase and phospho inositol 3 kinase. Gastric inhibitory peptide control adipose insulin sensitivity via activation of cAMP [15-18] (Figure 4).

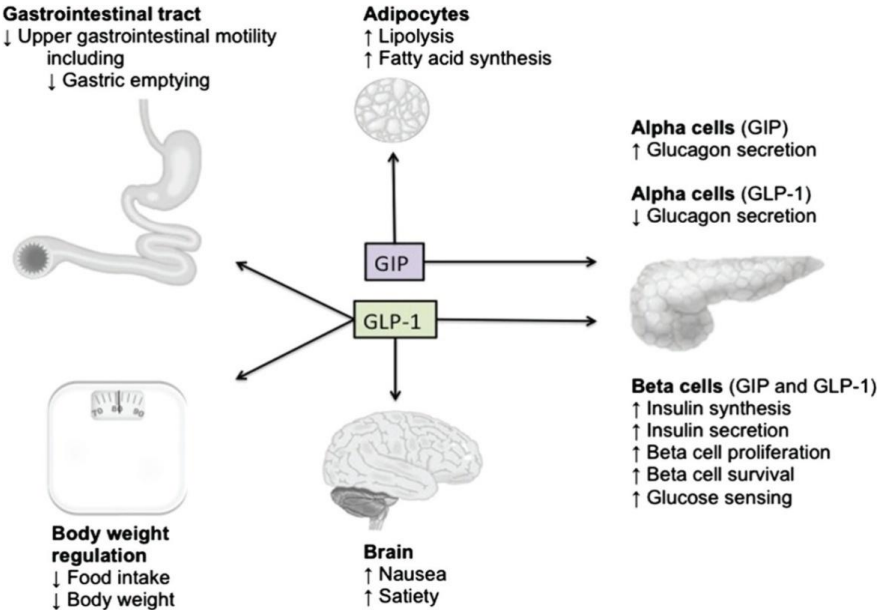

Figure 4: Schematic presentation of incretin effect.

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] (Figure 5).

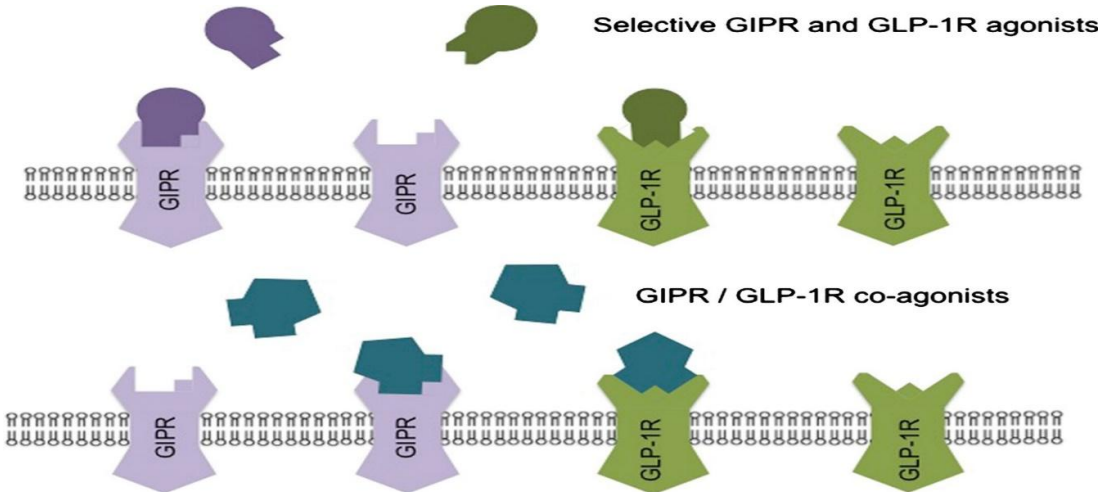

Figure 5: Schematic presentation of effect on GIP & GLP receptors.

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 ie maximum 25 gm of cereals to provide 18gm of glucose [20,21].

Conclusion

Optimal initial dietary intake of food valued <100 calories prompt incretin secretion, facilitate B cell mass of pancreas, initiate insulin secretion and prompt insulin sensitivity resulting in metabolic bioregulation achieving blood sugar within normal range and modulate HbA1C at normal level ensuring better quality of life.

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