Research Article

Changing Trend in Diabetes Mellitus

Avinash Shankar[1]*, Abhishek Shankar[2], Shubham[3], Amresh Shankar[4] and Anuradha Shankar[5]

[1]*Post Graduate in Endocrinology and Metabolism (AIIMS), RA Hospital and Research Centre, Warisaliganj (Nawada) Bihar, India*
[2]*All India Institute of Medical Sciences, New Delhi, India*
[3]*Max Hospital, Delhi, India*
[4]*Medical Officer, State Medical Services, Government of Bihar, Patna, India*
[5]*Medical Officer, RBSK, Government of Jharkhand, Ranchi, India*

*Corresponding Author: Avinash Shankar, Post Graduate in Endocrinology and Metabolism (AIIMS), RA Hospital and Research Centre, Warisaliganj (Nawada) Bihar, India.

Abstract

Diabetes mellitus, progressively increasing worldwide but India is considered Diabetes capital of the world with a projected incidence of 109 million by 2035, as this disease of luxury is affecting even down trodden daily wage earner and hard workers both sexes equally due to emergence of toxic non-nutrients in the diet, drinks and oil solely caused by rampant use of fertilizer, chemicals, pesticides, hormones, preservatives and processing.

In addition, patients show increased tolerability to high blood sugar level and create suspicion regarding etiopathogenetic of hyperglycaemia while altered hepatic profile and better glycemic control on adjunction of hepatologic with antidiabetic drug with restricted first diet to 100 calories suggest hyperglycaemia as a combined effect of hepatic and pancreatic dysfunction. In present study 20,000 population of 20 Dalit hamlets and 10 villages of Nawada district aged > 35 yrs screened for blood sugar, clinical examination and patients of Diabetes mellitus attending DRC of RA Hospital and research Centre, Warisaliganj (Nawada) evaluation reveals-Adjuvant hepatologic with antidiabetic drug and dietary restriction check circadian variation of blood sugar and ensure blood sugar bioregulation with continued tapering of anti-diabetic dose without any consequent sequel or adversity. Toxic non-nutrient dietary constituents suppress secretion and production of GLP 1 in the L cells of mucosal lining of the small intestine and stimulate production of Dipeptidyl peptidase 4 which further increases GPL 1 degradation resulting in decrease in volume of insulin secreting B cells in the pancreas and decline in insulin release manifesting as hyperglycaemia.

Keywords: Glycemic Control; Bioregulation; Circadian Variation; Dysfunction

Introduction

Diabetes mellitus incidence is progressively increasing and a diseases of luxury [1,2,3,4] is also affecting daily wage earner poor community without any discrimination which may be attributed to changed life style, dietary habits, declined nutritional value, increased non-nutritive constituent and emergence of some toxic enzymes or molecule in the commonly consumed food, vegetables, fruits, oil, condiment and drinks due to heavy miss use of fertilizers, chemicals, pesticides and hormones to grow and yield more.

Earlier patients with post prandial blood sugar > 400 mg and fasting > 300 mg [5,6] were very less and was considered a dreaded state of diabetes mellitus and usually present the clinician with various complication or in unconscious or semiconscious statue, but these days patients even with fasting blood sugar > 400 mg came walking and narrate their complaints of their own with comfort and ease.

In addition, Insulin supplementation was used to be some most proper therapeutics but these days the trend of multiple dose of Insulin or insulin regulated dose by insulin pump is quite in vogue [7,8,9] but patients becoming non-responsive or resistant even to Insulin supplementation.

In Diabetes mellitus focus must be on dietary restriction rather than poly molecule anti diabetic therapy as circadian variation of blood sugar level will pose threat to human vitality. Thus, creates a suspicion of existence of any supplementary cause in addition to pancreatic and usually present the clinician with various complication or in unconscious or semiconscious state, but these days patients even with fasting blood sugar > 400mg came walking and narrate their complaints of their own with comfort and ease [10,11].

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In Diabetes mellitus focus must be on dietary restriction rather than poly molecule anti diabetic therapy as circadian variation of blood sugar level will pose threat to human vitality. Thus creates a suspicion of existence of any supplementary cause in addition to pancreatic β cell dysfunction as a cause of the present hyperglycaemia. Hence to ascertain the variation in etio pathogenesis of hyperglycaemia, a study was planned to ascertain whether hepatic mechanism is also responsible for the present hyperglycaemic syndrome.

Material and Method
Design of Study:

Controlled comparative evaluation of hepatic function improvement on therapeutic outcome of Diabetes mellitus management.

Material:

To asses changing pattern of Diabetes mellitus 10 hamlets and 20 villages of Nawada district were randomly evaluated for urine sugar and blood sugar both fasting and pp in suspected cases of age > 20 yrs of either sex. In addition, patients attending at Diabetic Research Centre of RA Hospital and Research Centre, Warisaliganj (Nawada) were considered but patients with Diabetic sequelae were excluded from the present study.

Method:

Each person showing positive either for urine sugar, blood sugar or both were thoroughly interrogated for history of increased frequency of urine, increased thirst, increased appetite, lethargy, tingling numbness, recurrent boils, nonhealing wound, itching, general debility, exertional dyspnoea, sexual debility, personal habit, dietary habit ,schedule of diet, nature of work, duration of work and any family history of Diabetes mellitus, investigated for fasting and post prandiol blood sugar, urine sugar hematological parameters, hepatic profile and renal profile. The investigation is repeated after 3 and 6 months of therapy to establish the etiopathogenesis and therapeutic outcome.

Among the hospital detected patients either fresh or old cases taking treatment (Oral hypoglycemic or Insulin Supplement Or both) with dietary restriction and presenting with varied glycemic level were considered and divided in to two groups i.e.,

Both Group:

Diet: Carbohydrate restricted diet, with first oral restricted to 100 calories.

Continuing Oral hypoglycemic or Insulin or both in old cases while fresh cases were advised accordingly.

Group A (Study group):

Hepatogogue both oral and parenteral

Group B (Control group):

Placebo

Both group patients were given a follow up card and Glucostix to evaluate their urine for sugar, in case of manifestation like forgetfulness, lethargy, semi consciousness or complete absence of sugar in urine, attend the DRC for estimation of blood sugar, continuing OHA or Insulin supplement were tapered down with maintained normoglycemic level.

Initially patients were followed up weekly for 6 months, every 15th day for 1 yr and monthly for next 1 year to adjudge the therapeutic outcome and disease sequel.

Observation

Selected patients were of age group 30 - 65 yrs and out of all 196(16.2%) cases were of age < 35 yrs while 130(10.7%) were of age > 60 yrs (Table-1).

Items	N	%
Children sociodemographic data		
Sex		
Male	23	45.8
Female	27	54.2
Age group Mean age 9 years (SD = 3.33)		
6-8 years	23	46
9-12 years	27	54
Number of siblings		
4 and less	13	26.0
5-9	21	42.0
above 10	16	32.0
Mothers sociodemographic data		
Family monthly income		
Less than $ 220	28	56
$ 221-521	14	28
$ 521-750	8	16
Mother education		
Elementary	4	8.0
Primary	12	24.0
Secondary	20	40.0
Diploma	11	22.0
University	2	4.0
Post graduate	1	2.0
Father education		
Elementary	2	4.0
Primary	16	32.0
Secondary	21	42.0
Diploma	8	16.0
University	1	2.0
Post graduate	2	4.0
Father work		
Unemployed	9	18.0
Simple worker	16	32.0
Skilled worker	15	30.0
Employee	7	14.0
Merchant	3	6.0
Mother job		
House wife	46	92
Employee	4	8

Table 1: Sociodemographic characteristics of children with cancer (N = 50).

The male female composition was 729:477 (Pie diagram showing male: female composition).

Among the selected cases 675 and 531 were newly detected and old cases respectively (Bar diagram showing distribution of newly detected and old cases of Diabetes mellitus).

Out of all 27.9% were of middle class income group and 42.6% were daily wage earner (Table-2).

Income groups	Number of persons
Below poverty line	212
Daily earner	514
Low income	144
Middle class	336

Table-2: Shows Distribution of Patients as per their Economic Status.

34.8% were leading sedentary life while 25.3% were hard workers (Table-3).

Nature of work	Number of person
Sedentary	384
Exertion:	
Mild	119
Moderate	299
Severe	99
Hard worker	305

Table-3: Distribution as per Nature of Work.

68.32% were vegetarian and 31.6% were non vegeterain,75% were taking two times meal while 25% were consuming divided 4 meals (Table-4).

Particular	Number of persons
Vegetarian	824
Non-Vegetarian	382
Dietary schedule:	
Two heavy meals	904
Four divided meals	302

Table-4: Distribution as per Dietary Habit and Dietary Schedule.

33.33% were stressed and 7.8% were non-addict and non-stressed. (Table-5).

Personal status	Number of persons
Alcoholic	112
Toddy	396
Tabacco and Alc + Toddy	201
Stressed	402
No habit and non-stressed	095

Table-5: Distribution as per Personal Status.

40.8% persons were with normal ideal body weight while 18.2% with < IBW and 41% with > IBW (obese) respectively (Table-6).

Age group (in yrs)	Body weight		
	IBW	<IBW	>IBW
30-35	77	20	99
35-40	90	30	68
40-45	70	20	69
45-50	66	29	78
50-55	79	40	70
55-60	60	39	72
60-65	50	40	40

Table-6: Distribution as per Body Weight.

Out of all 412 were mahadalit and daily earner whereas 794 were of other categories (Table-7).

Particulars	Number of persons
Msahadalits	412
Others	794

Table-7: Distribution as per their Community.

56% were with fasting blood sugar > 200 mg and 50.2% with pp

blood sugar > 300 mg, 85% shows altered hepatic enzymes (SGOT, SGPT and Alkaline phosphatase) (Table-8).

Parameters	Number of persons
Blood Sugar:	
Fasting	
120-140	109
140-160	108
160-180	174
180-200	229
>200	676
Post prandiol:	
200-230	069
230-260	076
260-290	148
290-320	308
>320	605
Hepatic profile:	
SGOT:	
<35	182
>35	1024
SGPT:	
<35	180
>35	1026
Alkaline phosphatase	
<	
>	

Table-8: Distribution of Persons as per their Basic Biostatus.

Majority of study group (Group A) had marked and sustained decline in blood sugar with its bio regulation and progressive decline in continuing anti diabetic drug with complete withdrawal of anti-diabetic drug in 62% newly detected cases with normoglycemic state during 2 yrs of rigorous follow up without any circadian variation while majority in control group (Group B) persisted with fasting blood sugar > 150 mg% and post prandiol blood sugar > 225 mg % even with similar dietary restriction and anti-diabetic therapy. In addition, all cases of study group achieved and retained normal hepato renal status while control group 40% patients presented with altered hepato renal function. and progressive increase in dose of anti-diabetic drugs (Table-9).

Particulars	Number of patients					
	Group A			Group B		
	1st	2nd	3rd	1st	2nd	3rd
Blood Sugar:						
Fasting -						
<100mg	202	392	603	-	-	104
>100mg	401	211		603	603	499
Post prandiol:						
<170mg	202	392	603	-	-	104
>170mg	401	211	-	603	603	499
Hepatic profile:						
SGOT						
<35	124	399	603	91	100	603
>35	479	204	-	512	503	-
SGPT:						
<35	124	399	603	91	100	100
>35	479	204	-	512		
Alkaline phosphatase						
< 100 unit/L	244	512	603	242	298	291
>100unit/L	359	91	-	361	305	312

Table-9: Shows outcome of the Study.

## Discussion

Diabetes mellitus rampantly spreading disease was thought to be purely due to defunct pancreatic Beta cell function [14,15] and these days affecting hard workers and daily wage earner, considerably due to emergence of non-nutrients in routinely consumed diet and toxic substances which is not only affect the hepatic parenchyma and pancreas but also potentiate the Dipeptidyl peptidase 4 secretion and dampen the secretion of Glucagon like peptide I(GLP-I) and Glucose dependent Insulin tropics (GLI) in the small intestine. Altered hepatic parameters in majority detected cases and response of hep [apologue adjuvant with anti-diabetic therapy ensure decline in blood sugar with bioregulation and without circadian variation. Also prompted elimination of toxic non-nutrient of the diet and help suppression of Dipeptidyl peptidome 4 thus delays degradation of GLP and GIP ensuring insulin bio regulation and progressive decline in continuing anti diabetic drugs [10,11,12,13]. High sustainability to higher blood sugar is due to glucose un utilized by liver for Glycogenesis, thus this study affirms the hyperglycaemic manifestation as a combined effect of hepatic, pancreatic and intestine hormone dysfunction, secondly incidence in daily wage earner is due to consumption of similar cereals irrespective of the economic strata whose non-nutrient constituent affect alike.

Hence to curb the disease and limit its progressive increase the prime step needed is -

- Restrict first diet to 100 calories or 25 gm of cereals
- Avoid use of rice, potato, sugar and poultry products
- Limit the use of fertilizer, chemical, hormones, pesticides and preservatives
- Prefer fresh food

If the population not consider it urgent India will become Diabetic India.
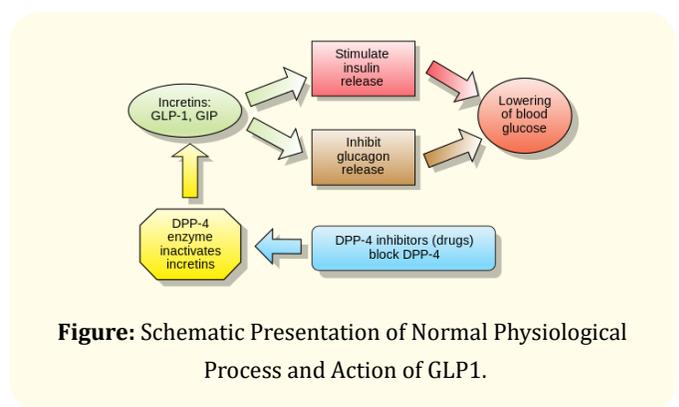
Figure: Schematic Presentation of Normal Physiological Process and Action of GLP1.

Conclusion

The disease known for luxury these days also common among hard worker and daily wage earners due to altered production and secretion of GLP1 and GIP from L cells of mucosal lining of duodenum jejunum and small intestine. Patients of hyperglycaemia either fresh or old taking anti diabetic drugs show altered hepatic function and capacity to sustain its vitality even in a state of highly raised blood sugar. Adjuvant hepatologic in either cases i.e., fresh or old shows marked decline in blood sugar with sustained normoglycemic state without any circadian variation of blood glucose, thus suggest these days hyperglycaemia as a combined effect of glucose conversion and glucose metabolism i.e. alteration in function of both liver and pancreas as a result of increasing non-nutrients in diet and altered life style.

Bibliography

1. Kumar A., *et al.* "India towards diabetes control: Key issues". *Australasian Medical Journal* 6.10 (2013): 524-531.

2. Wild S., *et al.* "Global prevalence of diabetes-estimates for the year 2000 and projections for 2030". *Diabetes Care* 27.5 (2004): 1047-1053.

3. Whiting Dr., *et al.* "IDF Diabetes atlas: Global estimates of the prevalence of diabetes for 2011 and 2030". *Diabetes Research and Clinical Practice* 94.3 (2011): 311-321.

4. Anjana RM., *et al.* "The need for obtaining accurate nationwide estimates of diabetes prevalence in India - rationale for a national study on diabetes". *Indian Journal of Medical Research* 133 (2011): 369-380.

5. Ramachandran A., *et al.* "Diabetes Epidemiology Study Group in India (DESI). High prevalence of diabetes and impaired glucose tolerance in India: National Urban Diabetes Survey". Diabetologia 44.9 (2001): 1094-1101.

6. Arora V., *et al.* "Prevalence of diabetes in urban Haryana". *Australasian Medical Journal* 3.8 (2010): 488-494.

7. Bramley D., *et al.* "Indigenous disparities in disease-specific mortality, a cross-country comparison: New Zealand, Australia, Canada, and the United States". *The New Zealand Medical Journal* 117.1207 (2004): U1215.

8. Sukala WR., *et al.* "Exercise intervention in New Zealand Polynesian peoples with type 2 diabetes: Cultural considerations and clinical trial recommendations". *Australasian Medical Journal* 5.8 (2012): 429-435.

9. Mohan V., *et al.* "Current status of management of diabetes and glycaemic control in India: Preliminary results from the DiabCare India 2011 Study". *Diabetes* 61 (2012): a645-a677.

10. Donnelly D. "Structure and function of GLP 1 and its ligands". *British Journal of Pharmacology* 166.1 (2012): 21-41.

11. Unnikrishnan RI., *et al.* "Importance of Controlling Diabetes Early-The Concept of Metabolic Memory, Legacy Effect and the Case for Early Insulinisation". *Journal of the Association of Physicians of India* 59 (2011): 8-12.

12. Verma R., *et al.* "National programme on prevention and control of diabetes in India: Need to focus". *The Australasian medical journal* 5.6 (2012): 310-315.

13. State-based diabetes prevention and control program. Centers for Disease Control and Prevention. U.S Department of Health and Human Services (2013).

14. *National Diabetes Education Program*. Centers for Disease Control and Prevention (2013).