Diabetes Mellitus Management, Needs Reconsideration

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Abstract

Diabetes mellitus is progressively increasing worldwide and India is considered as 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 hard workers and 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 showing increased tolerability to high blood sugar level and growing resistance to continuing drug create suspicion regarding etio pathogenesis of hyperglycaemia.

Objective of the study: To ascertain the incidence of diabetes mellitus among hard workers and pathophysiology of increased tolerance to high blood sugar level.

Material & method: For the purpose 20,000 population of 20 Dalit hamlets and 10 villages of Nawada district aged > 35 years were interrogated thoroughly, examined clinically and screened for blood sugar urine sugar and other bio parameters. In addition patients of Diabetes mellitus attending Institute Of Applied Endocrinology and Aarogyam Punarjeevan Patna 14 were also evaluated accordingly.

Result: Study reveals adjuvant hepatogogue with antidiabetic drug and dietary restriction check circadian variation of blood sugar and ensures 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 β cells in the pancreas and decline in insulin release manifesting as hyperglycemia while altered hepatic profile and better glycemic control on adjunction of hepatogogue with antidiabetic drug and restricted first diet to 100 calories suggest hyperglycaemia as a combined effect of hepatic and pancreatic dysfunction.

In addition no patients were given any anti diabetics without repeated screening for fasting, post prandial, before and after lunch and dinner to adjudge any circadian variation.

Majority patients of study group had complete bioregulation of blood sugar without any adjuvant or adversity.

Conclusion: Thus in present scenario prior to advocation of anti-diabetic drugs in newly detected cases and alteration in dose of antidiabetic drugs in old diabetics Patients must be reassessed after due change in –

- Diet, life style, stress redressal
- Timely advocation of drugs and diet to ensure blood sugar bio regulation.

Introduction

Globally prevalence of diabetes mellitus increasing progressively and quadrupled in past three decades affecting presently 1 in 11 adult world wide establishing diabetes as 7th major cause of death. Asia is a major area of the rapidly emerging type 2 diabetes mellitus global epidemic, with India at the top [1-4]. The latest estimates show a global prevalence of 382 million people with diabetes in 2013, expected to rise to 592 million by 2035. Diabetes can lead to multisystem complications of microvascular endpoints, including retinopathy, nephropathy and neuropathy, and macrovascular endpoints including ischaemic heart disease, stroke and peripheral vascular disease. Diabetes currently affects more than 62 million Indians, which is more than 7.1% of the adult population. Nearly 1 million Indians die due to diabetes every year [5-8].

Diabetes mellitus a diseases of luxury 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 [9-11]. 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 but patients becoming non-responsive or resistant even to Insulin supplementation [12-14].

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 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 complications 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 [15-18].

Objective of The Study

To assess the role of dietary control, first oral diet and hepatogogue co administration with Oral hypoglycaemic and Insulin supplementation in blood sugar bioregulation.

Materials and Method

Design of study

Controlled comparative evaluation of hepatic function improvement on therapeutic outcome of Diabetes mellitus management. In Diabetes mellitus focus must be given 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

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 Institute of Applied Endocrinology And Aarogyam Punarjeevan, Patna 14, Bihar were considered and 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, non-healing 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 prandial blood sugar, before and after lunch and dinner, urine sugar haematological 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 intake restricted to 100 calories. Continuing oral hypoglycaemic or Insulin 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 anti diabetics (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 year and monthly for next 1 year to adjudge the therapeutic outcome and disease sequel.

Observations

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).

Age group (in years)	Number of Patients		Total
	Male	**Female**	
30 35	109	87	196
35-40	117	71	188
40-45	97	62	139
45-50	104	69	173
50-55	115	74	189
55-60	103	68	171
60-65	84	46	130
Total	729	477	1206

Table 1: Distribution of patients as per age & sex.

Male: female composition was 729:477 (Fig-1)

Figure 1: Pie diagram showing male: female composition.
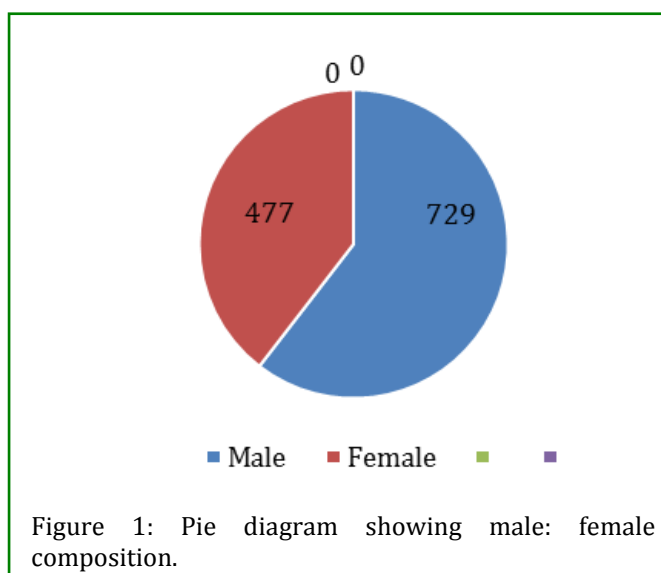
675 and 531 cases were of new and old cases respectively (Figure 2).

Figure 2: Bar diagram showing distribution of patients as per disease status.
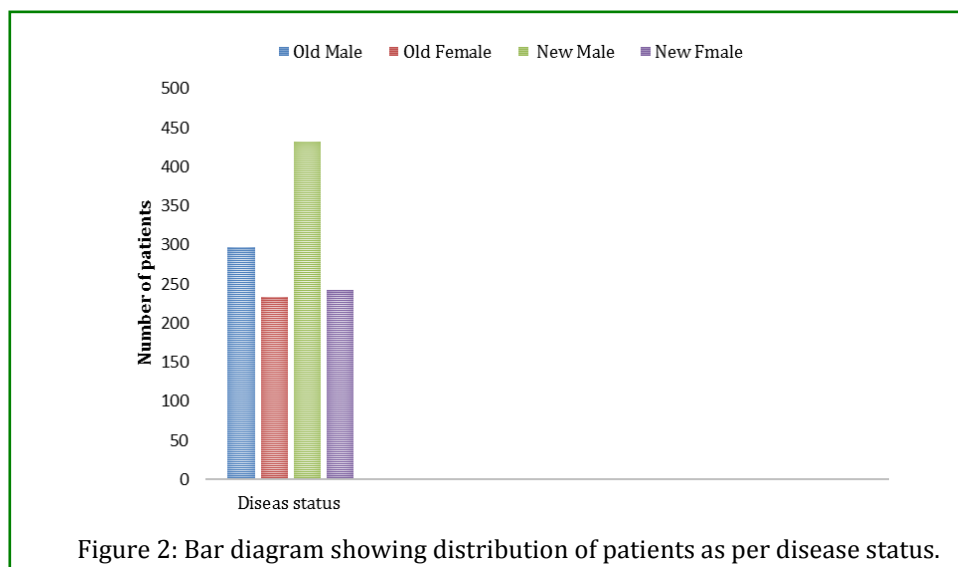
Out of all 27.9%were of middle class income group and 42.6%were daily wage earner (Table-2).

Income group	Number of patients
Below poverty line (BPL)	212
Daily earner	514
Low income	144
Middle class	336

Table 2: Distribution of patients as per economic status.

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

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

Table 3: Distribution of patients as per their nature of work.

68.32% were vegetarian and rest 31.68% were non vegetarian,75%weretaking two times meal while 25% were consuming divided four meals (Table 4).

Particulars	Number of patients
Vegetarian	824
Non vegetarian	382
Two heavy meals	904
Four divided meals	302

Table 4: Distribution of patients as per dietary habit and schedule

33.33% were stressed and 7.89% were none stressed and non-addict (Table 5).

| Personal habits	Number of patients
Alcoholic	112
Toddy	396
Multi narcotics	201
Stressed	402
No habit non stressed	95

Table 5: distribution of patients as per personal status.

40.8% Were with Normal ideal body weight while18.2%with <IBW and 41% with >IBW (obesity) (Table 6)

Age group (in yrs)	Number of patients		
	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 of patients as per body weight.

Out of all 412 were mahadalit and daily wage earner while 794were of other categories Figure 3.

Figure 3: Bar diagram showing distribution of patients as per community
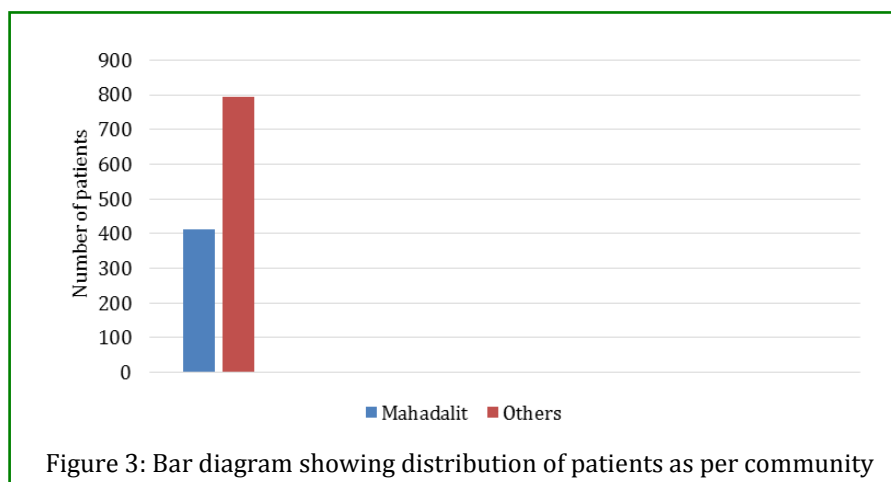
56% were with fasting blood sugar >200mg and 50.2% with post prandial blood sugar >300mg%85% shows altered hepatic enzymes (Table-7)

Parameters	Number of persons
Blood Sugar	
Fasting	
120-140	109
140-160	108
160-180	174
180-200	229
>200	676
Post prandial	
200-230	69
230-260	76
260-290	148
290-320	308
>320	605
Hepatic Profile	
SGOT	
<35 IU	182
>35 IU	1024
SGPT:	
<35 IU	180
>35 IU	1026
Alkaline phosphatase	
<100 IU/L	486
>100IU/L	720

Table 7: Distribution of patients as per their basic bio status.

Majority of study group (Group A) had marked and sustained decline in blood sugar with its bioregulation and progressive decline in dose of continuing antidiabetic drugs (OHA & Insulin) with complete withdrawal of antidiabetic drugs in 62% of newly detected cases with normo glycaemic state during 2 years of rigorous follow up without any circadian variation while majority in control group(Group B) persisted with fasting blood sugar >150mg% and post prandial blood sugar 225mg% even with similar dietary restriction and antidiabetic regime.

In addition all cases of study group achieved and retained normal hepatic and renal profile while control group 40% patients presented with altered hepato renal function in spite of progressive increase in dose of continuing antidiabetics (Table-8).

Particulars	Number of patients					
	Group A			Group B		
	1st	2nd	3rd	1st	2nd	3rd
Blood Sugar Fasting						
<100	202	392	603	-	-	104
>100	401	211	-	603	603	499
Post prandial						
<170	202	392	603	-	-	104
>170	401	211	-	603	603	499
Hepatic Profile						
SGOT						
< 35 IU	124	399	603	91	102	103
>35 IU	479	204	-	512	501	500
SGPT						

| <35IU	124	399	603	91	100	100
>35 IU	479	204	-	512	503	503
Alkaline phosphatase						
<100 IU/L	244	512	603	242	298	291
>100U/L	359	91	-	361	305	312

Table 8: Outcome of the study.

Result

Hepatogogue adjunction with dietary restriction and first oral dose intake bio regulate metabolic process and blood sugar without any circadian variation or untoward effect with continued tapering of continuing antidiabetics.

Discussions

Diabetes mellitus rampantly spreading disease was thought to be purely due to defunct pancreatic β cell function [19,20]. 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.(Figure -4) Altered hepatic parameters in majority detected cases and response of help [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 [21,22]. 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
• Remain stress less for which develops ignorance.
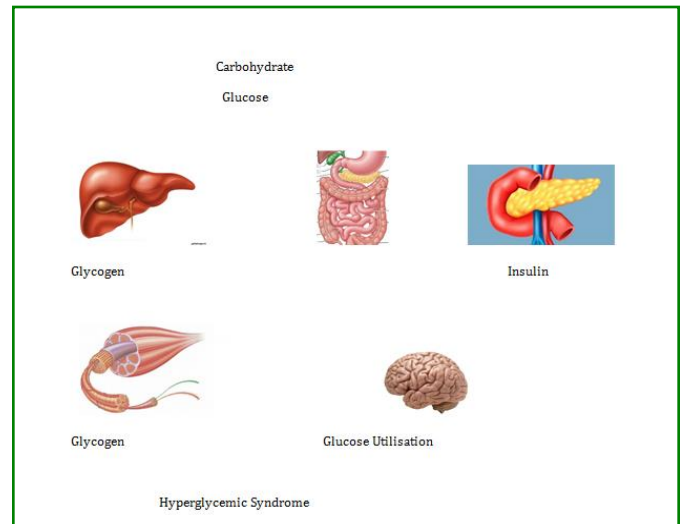
Conclusion

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 hepatogogue in either cases i.e., fresh or old shows marked decline in blood sugar with sustained normo glycaemic 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.

References

1. World Health Organization (2016) Global Report on Diabetes. Geneva, Switzerland.

2. Melmed S, Polonsky K, Larsen PR, Kronenberg H (2012) Williams textbook of endocrinology (12th edn.), Philadelphia: Elsevier/Saunders. 1371–1435.

3. Agardh E, Allebeck P, Hallqvist J, Moradi T, Sidorchuk A (2011) Type 2 diabetes incidence and socio-

economic position: a systematic review and meta-analysis. International Journal of Epidemiology 40(3): 804-818.

4. Jason G (2010) India's Diabetes Epidemic Cuts Down Millions Who Escape Poverty. Bloomberg. Retrieved 8 June 2012.

5. Wild S, Roglic G, Green A, Sicree R, King H (2004) Global prevalence of diabetes-estimates for the year 2000 and projections for 2030. Diabetes Care 27(5) (2004): 1047-1053.

6. Whiting DR, Guariguata L, Weil C, Shaw J (2011) IDF Diabetes atlas: Global estimates of the prevalence of diabetes for 2011 and 2030. Diabetes Res and Clin Pract 94(3): 311-321.

7. Anjana RM, Ali MK, Pradeepa R, Deepa M, Datta M, et al. (2011) The need for obtaining accurate nationwide estimates of diabetes prevalence in India - rationale for a national study on diabetes. Indian J Med Res 133: 369-380.

8. Ramachandran A, Snehalatha C, Kapur A, Vijay V, Mohan V, et al. (2001) High prevalence of diabetes and impaired glucose tolerance in India: National Urban Diabetes Survey. Diabetologia 44(9): 1094-1101.

9. Arora V, Malik JS, Khanna P, Goyal N, Kumar N, et al. (2010) Prevalence of diabetes in urban Haryana. Australasian Medical Journal 3(8): 488-494.

10. Shankar A, Amresh S, Anuradha S (2018) Dietary non nutrients posing Health hazards. Acta Scientific Nutrition Health 2(2): 13-26.

11. Shankar A, Amresh S, Anuradha S (2018) Effect of initial Dietary Dose on Therapeutic response and quality of life in Diabetes Mellitus. Acta Scientific Nutrition Health 2(8): 40-46.

12. Diabetes can be controlled in 80 percent of Cases in India. IANS. News.biharprabha.com. Retrieved 6 February 2014.

13. Kleinfield NR (2006) Modern Ways Open India's Doors to Diabetes. New York Times. Retrieved 8 June 2012.

14. Kumar A, Goel MK, Jain RB, Khanna P, Chaudhary V (2013) India towards diabetes control: Key issues. Australas Med J 6(10): 524-531.

15. Sukala WR, Page RA, Rowlands DS, Lys I, Krebs JD, et al. (2012) Exercise intervention in New Zealand Polynesian peoples with type 2 diabetes: Cultural considerations and clinical trial recommendations. Australas Med J 5(8: 429-435.

16. Mohan V, Shah SN, Joshi SR, Seshiah V, Sahay BK, et al. (2012) Current status of management, control, complications and psychosocial aspects of patients with diabetes in India: Results from the DiabCare India 2011 Study. Indian J Endocrinol Metab 18(3): 370-378.

17. Donnelly D (2012) Structure and function of GLP 1 and its ligands. Br J Pharmacol 166(1): 27-41.

18. Unnikrishnan RI, Anjana RM, Mohan V (2011) 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: 8-12.

19. Verma R, Khanna P, Mehta B (2012) National programme on prevention and control of diabetes in India: Need to focus. The Australa Med J 5(6): 310-315.

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

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

22. Bramley D, Hebert P, Jackson R, Chassin M (2004) Indigenous disparities in disease-specific mortality, a cross-country comparison: New Zealand, Australia, Canada, and the United States. N Z Med J 117(1207): U1215.