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Management of Post Prandial Hyperglycemia: An Indian Consensus statement on AGIs

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ABSTRACT

The global burden of diabetes as per the International Diabetes Federation 2013 amounts to 382 million and is fast gaining the status of a potential epidemic in India with more than 65 million diabetic individuals currently diagnosed with the disease[1, 2]Diabetes Mellitus is a chronic metabolic disorder which causes high mortality and morbidity due to its micro vascular and macro vascular complications.[3]Although there is an increase in the prevalence of type 1 diabetes, the major driver of the epidemic is the more common form of diabetes, i.e. type 2 diabetes mellitus (T2DM) which accounts for more than 90% of all diabetes cases.[4]Even though diabetes poses high economic burden in India, the real burden of the disease is mainly due to its associated complications.

Keywords: International Diabetes Federation, Type 2 diabetes mellitus

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INTRODUCTION

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Large controlled clinical trials have demonstrated that intensive treatment of diabetes can significantly decrease the development and/or progression of microvascular and macrovascular complications of diabetes.[6-9]

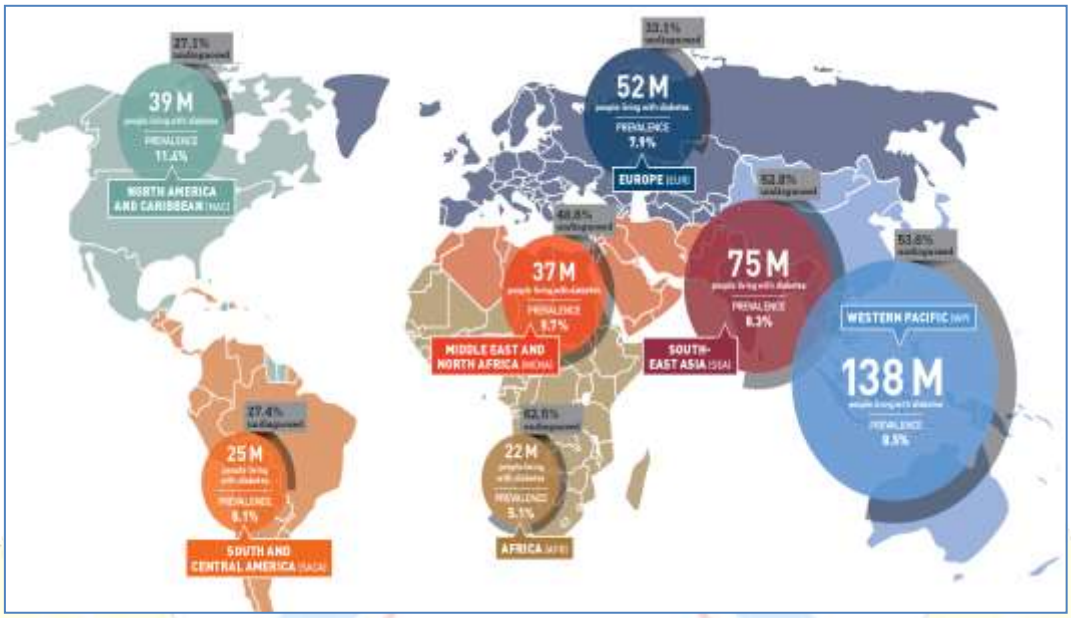

Figure 1. IDF regions and global projection of number of people with diabetes, 2013[1]

Number of people with diabetes by IDF Region, 2013. IDF Diabetes Atlas; Sixth Edition, Pg. no. 11

Postprandial hyperglycemia (PPHG) is a very frequent phenomenon in people with type 1 and type 2 diabetes[10-14]and can occur even when overall metabolic control appears to be adequate as assessed by glycated Hemoglobin (HbA1c) levels.[11,13] This is supported by findings from a study conducted on 3,284 people with non-insulin treated T2DM demonstrated that a post prandial blood glucose value of more than 8.9 mmol/l (160 mg / dL) was recorded at least once among 84% of the patients, even among those with good glycemic control.[13] Until recently, the predominant focus of diabetes therapy has been on lowering HbA1c levels, with a strong emphasis on fasting plasma glucose. Although control of fasting hyperglycemia is necessary, it is usually insufficient to obtain optimal glycemic control.[15]

Definition of PPHG

American Diabetes Association (ADA) 2013 defines post-meal hyperglycemia as a 2-h plasma glucose level of more than 200 mg/dL(11.1 mmol/l) during an oral glucose tolerance test (OGTT). It recommends the use of a glucose load equivalent of 75 g anhydrous glucose dissolved in water as prescribed by WHO.41

A plasma glucose level of 7.8 mmol/l (140 mg/dL) or more, after 1-2 hours of food ingestion is defined as post prandial hyperglycemia by International Diabetes Federation (IDF) 2011.42Furthermore, various epidemiological studies have shown a strong association between PPHG and cardiovascular risk and outcomes suggesting that PPHG may be a better

predictor of cardiovascular risk than is FPG or HbA1c alone(Table 1).[16-20]PPHG is also linked to retinopathy,[21] cognitive dysfunction in elderly people,[22]and certain cancers.[23-27]

It has been reported that PPHG is a widespread condition in Asia which is often under diagnosed. Dickinson et al found that Asian Indians display a marked rise in prandial glucose excursion after consumption of 75-gm of bread meal.[28]PPHG is strongly associated with the carbohydrate content of the diet which is one of the major sources of food in Indian diet.[29]Most studies confirm that the amount and type of carbohydrate consumed in a meal is a major determinant of the PPHG excursion.[30]Consumption of carbohydrate-rich foods lead to alteration in blood glucose levels or glycemic index (GI).[31]Consumption of rice is very high in South India which is associated with 4-5 fold increase in risk of diabetes.[32]Furthermore, a recent dietary survey from India estimated that dietary carbohydrates account up to 64% of daily calories in patients with T2DM.[33]Also, PPHG leads to higher lipemic peaks and links to CVD in patients with T2DM.[34]Thus it may be anticipated that Asians tend to show higher rates of PPHG, increased activity of glucosidase enzyme and incretin hormones in the gut.[35,36]Therefore, there is a growing need to employ intense measures for reducing PPHG excursions and excessive glycemic readings.

Table 1:Morbidity and mortality related to post-prandial hyperglycemia

Study	Patients	Key Findings
DECODE study[19]	10 prospective studies among 15,388 men and 7,126 women not previously diagnosed with diabetes	2-hour blood glucose levels following 75-g OGTT better predictor of all cause and cardiovascular deaths from FPG levels
Chicago Heart Association[20]	12,220 men with diabetes or asymptomatic hyperglycemia	Increased risk of CVD mortality with higher PPG (after 50-g-OGTT)
Temelkova Kurktschiev et al[16]	582 men and women at risk for type 2 diabetes	2 hour blood glucose levels and spikes more strongly associated with CIMT than FPG or HbA1c
Diabetes Intervention Study[17]	1139 men women with newly diagnosed type 2 diabetes	PPHG but not FPG, significant risk factor for MI and mortality
Companion Postprandial Hyperglycemia Study[18]	93 men and 82 women with type 2 diabetes, not previously drug treated	Reduction of PPHG, but not FPG, associated with reductions in CIMT

Managing Post Prandial Hyperglycemia with Alpha-Glucose Inhibitors

Various international bodies have proposed different optimal goals for post meal glucose(Table 2).[37-40] While International Diabetes Federation (IDF) 2011 advocates a level of less than 9.0 mmol/l (160 mg/dL) (as long as hypoglycemia is avoided), American College of Endocrinology Conference recommends 2-hour postprandial plasma glucose to be at a level lower than 140 mg/dL whereas American Diabetes Association (ADA) suggests more

relaxed targets i.e. lesser than 180 mg/dL.[41,42]However, targets for glycemic control must be individualized (between 140 to 180mg/dL) based on each patient's clinical status, which includes socioeconomic circumstances, lifestyle behaviors, medications used, level of motivation, and compliance to therapy.

Table 2: Summary of postprandial hyperglycemia guidelines

Organization, Year	Type of Diabetes	FPG (mg/dL)	PPHG (mg/dL)	PPD Timing
ADA , 2015[37]	Type 2 diabetes	<126	<200	2 hours post prandially
IDF , 2015[38]	Type 2 diabetes	-	<160	1-2 hours post prandially
AACE, 2015[39]	Type 2 diabetes	<100	<140	2 hours post prandially
ADA/EASD , 2012[40]	Type 2 diabetes	<130	<180	-

IDF-International Diabetes Federation; EASD- Eurapoean Association for the Study of Diabetes; ADA- American Diabetes Association; AACE- American Association of Clinical Endocrinologists

Findings from large, randomized, clinical trials demonstrate that intensive management of glycemia, as assessed by HbA1c, can significantly decrease the development and/or progression of chronic complications of diabetes.[6-8,43] Diet and exercise is the first step in the treatment of T2DM. But if these measures alone fail to sufficiently control blood glucose levels, starting oral drug therapy is recommended. Metformin, Sulfonylurea and Thiazolidinediones are observed to reduce HbA1c by 1%-2%, whereas AGIs, GLP-1, DPP-4 and SGLT-2 reduces from 0.4%- 1.45% (Table 3).[44-70]Although biguanides, sulfonylureas and thiazolidinedionesare effective in controlling fasting hyperglycemia, a high percentage of patients have sustained elevated HbA1c because of persistent elevation of PPHG[71]Similarly, despite clinical benefits of insulin therapy for patients with T2DM, many potential barriers are observed with insulin therapy making it reluctant for patients and physicians as an efficacious approach for the management of T2DM.[72]Barriers for initiating insulin treatment includes inconvenience due to self administration of multiple insulin injections every day, injections being painful (for needle phobic patients), slow absorption of insulin (slow onset of action, delayed peak insulin level, prolonged duration of action), perception of hypoglycemia incurred by observing other diabetic patients on insulin therapy and weight gain. In addition to this, lifestyle challenges such as travelling and busy schedules, inability to control food cravings and eating habits, lack of self-management education and cost are other factors that impede the initiation of insulin therapy. Thus physiologic insulin therapy limits mimicking normal insulin secretion and hence measuring PPHG appears to be prominent characteristic of glycemic dysregulation in patients with T2DM.[73,74]

Table 3: Role of drugs and its mechanism in lowering post prandial hyperglycemia

Name of drugs	**MOA in lowering PPHG**	**Reduction in HbA1c levels**
Metformin[44,45]	Metformin reduces postprandial levels of glycemia, triglycerides and free fatty acids. Metformin also reduces high levels of fasting and postprandial cholesterol and insulin	HbA1c reduction by 1%- 2%
Sulfonylurea[46,47]	Sulfonylurea triggers insulin release from the pancreatic beta cell after meals	HbA1c reduction by 1% - 2%
AGIs like Acarbose, Miglitol, Voglibose[48-51]	AGIs delay the absorption of carbohydrates from the gastrointestinal tract, thereby limiting post-meal plasma glucose excursion. They inhibit α-glucosidase located in the brush border of the proximal small intestine that breaks down disaccharides and more complex carbohydrates	HbA1c reduction by 0.5 - 1.5%
Glucagon like peptide (GLP-1) derivatives like Liraglutide, Exenatide[52-54]	GLP-1 derivatives are effective in reducing postprandial plasma glucose by lowering the HbA1c levels, delaying gastric emptying and increasing satiety. GLP-1 derivatives are usually associated with significant weight loss	HbA1c reduction by 0.7% - 0.9%
Dipeptidyl peptidase-4 (DPP-4) inhibitors like Linagliptin, Saxagliptin, Sitagliptin and Vildagliptin[55-59]	Inhibition of DPP-4 by DDP-4 inhibitors increases prandial insulin secretion and suppresses glucagon secretion, thereby decreasing hepatic glucose production and improving peripheral glucose uptake, which decreases PPHG in patients with type 2 diabetes	HbA1c reduction by 0.4% - 1.1%
Rapid acting insulin analogs like Insulin Lispro, Aspart,Glulisine[60-62]	Insulin analogs are rapid acting and produce a rapid and short acting insulin spike to improve PPHG.[9] They provides more rapid absorption compared to regular human insulin	HbA1c reduction by 2.2%
Sodium glucose co-transporter-2 (SGLT-2)[63-66]	Renal glucose release increases more than 2-folds during the 4.5-hour postprandial period.SGLT-2 reduces renal glucose reabsorption and induce urinary glucose excretion, thereby lowering plasma glucose	HbA1c reduction by 0.7% - 1.45%
Thiazolidinediones[59,67-70]	Thiazolidinediones decreases insulin resistance, FPG, insulin, and free fatty acids. It lowers HbA1c by 1–1.5%	HbA1c reduction by 1.3% - 1.6%

Table 4.Comparative parameters of Voglibose, Acarbose, and Miglitol

Parameters		**Voglibose**	**Acarbose**	**Miglitol**
Pharmacokinetics[80,81]	Absorption	Poorly absorbed (<6%); absorbtion is dose dependent; bioavailability is low	Poorly absorbed (<2%), bioavailability is low	25 mg completely absorbed; 100 mg 50-70% absorbed
	Distribution	Lumen of gastrointestinal tract	Extracellular fluid	Extracellular fluid
	Metabolism	Intestinal enzymes and microbial flora	Gastrointestinal tract principally by intestinal bacteria, also by digestive enzymes	None, metabolites not detected in plasma, urine/feces
	Excretion	98% through fecal route, 5% through renal route	51% through fecal route, 34% through renal route	>95% through renal route
Dose[80,81]	Initial and maintenance dose	Start with 0.2 mg with each meal, increase to 0.3 mg/meal	Start with 25 –50 mg before each meal; increase to 50 –100 mg/meal	Start with 25 mg before each meal; increase to 50 –100 mg/meal
Adverse effects[80-82]	Gastrointestinal disturbances	Flatulence,abdominal pain,bloating in 25% of subjects	Flatulence reported in 61.77 % of subjects	Flatulence reported in 56% of subjects
Side effects[83-85]	-	Hypoglycemia, anorexia, (Prescribing info)	Meteorism and elevated liver transaminases (Abott art, 52, 190)	Diarrhea observed in 11.9% of patients (Segal P Elevated liver transaminases(Segal P)
Toxicity[80,87]	-	Elevated levels of liver enzymes	Elevated levels of liver enzymes	Not hepatotoxic
HbA1c[3, 75,86-89,]	-	• Reduction by 1.57%* • Reduction range: 0.48% - 1.6%	• Reduction by 1.14%* • Reduction range: 0.8%-1%	• Reduction by 1.72%* • Reduction range: 0.22%-0.82%
FBG[88,89]	-	• Reduction by 21.43 g/dL^{+} • Reduction by10.8mg/dL	• Reduction by 21.76 mg/dL* • Reduction by 19.62mg/dL	• Reduction by 28.03 mg/d* • Reduction by 9.36 mg/dL
PPHG[87-89]	-	• Reduction by 93.7mg/dL * • Reduction range 43mg/dL - 57mg/dL	• Reduction by 58.46 mg/dL* • Reduced to 41.76 - 64mg/dL	• Reduction by 86.63 mg/dL* • Reduction by 48.6 mg/dL

$^{*}p<0.001$; $^{+}p<0.05$

Alpha-Glucosidase inhibitors (AGIs)control diabetes by inhibiting alpha glucosidase enzymes that limits the breakdown from starch and disaccharides, the major carbohydrate component in food. Through competitive inhibition of this enzyme, AGIs delay intestinal carbohydrate absorption and attenuate PPHG excursions.[75,76]AGIs have been shown in several randomized controlled trials to be effective in controlling PPHG, whether they are used as monotherapy or in combination with other antidiabetic medications.[77-79]Additionally AGIs are safe and nontoxic drugs with minor gastrointestinal side effects of flatulence and diarrhea which can be minimized.[47]Voglibose, Acarbose and Miglitol are commercially available AGIs for treatment of patients with T2DM(Table 4).[3, 75,80-89]These AGIs result in comparable changes in HbA1c levels and body weight in T2DM patients specifically in Asian and Caucasian populations.[90] Altogether, AGIs appear to be a serious therapeutic option in the treatment of T2DM as they have a comparable effect on glycemic control compared to metformin with no harmful adverse events, they decrease body mass index, they possibly reduce the risk for cardiovascular disease and the side-effects may be reduced by administering a lower dose without influencing its effect on glycemic control.

Role of Voglibose in the management of PPHG

Voglibose, as a monotherapy or in combination with other drugs reduces the development of T2DM in high risk individuals with impaired glucose tolerance and thus results in lifestyle modification.[91] By controlling PPHG, Voglibose causes about 34% risk reduction in development of new cases of hypertension and about 49%risk reduction in cardiovascular diseases (Fig 2).[3,92] The inhibitory activity of Voglibose on maltose and sucrose is reported to be 190-270 times higher than that of Acarbose and about 100 times higher than that of Miglitol.[93] Voglibose also has a better safety profile with only ~7% adverse drug reactions compared to Acarbose (~33%), and Miglitol (~17%) in T2DM patients.[89]In elderly patients and in those with hepatic dysfunction or mild to moderate renal impairments with contraindications for other OHAs, Voglibose is deemed helpful.[94] Voglibose is found to reduce the progression of average and maximum carotid intima media thickness (CIMT) to -0.024 and -0.021 mm/year, respectively for 3 years in patients with T2DM treated with Sulfonylueras or Insulin.[95]Overall, Voglibose is an effective, safe and well tolerated treatment for diabetes, which provides cardiovascular benefits to patients with T2DM.

When adequate glycemic control i.e. PPHG level is not achieved by monotherapy with oral hypoglycemic agents (OHAs), Voglibose can be used in combination with Sulfonylurea or Metformin.[94,96,97]Voglibose in combination with Sulfonylureas in patients with T2DM showed effective reduction in HbA1c (-0.8%, $p<0.001$), FPG (-17.5 mg/dL, $p<0.001$) and PPHG (-60.0 mg/dL, from baseline $p<0.001$) levels than placebo over 24 weeks suggesting

that the combination may be effective in controlling plasma glucose, thereby delaying the vascular complications.[98] . Combination of metformin and AGIs are adopted as they delay gastric emptying, early satiety, decrease glucose spikes and lead to lesser weight gain.However, AGIs should be administered as an add-on therapy after exhausting all other oral hypo glycemic agents. Another study reports that the addition of Voglibose to Pioglitazone attenuates the weight gain induced by Pioglitazone.[99]Triple drug combination of Voglibose, Glimepiride and Metformin has been shown to reduce PPHG levels (baseline 264.4mg/dL± 48.84, after 6 months 170.27mg/dL±33.98), FPG (baseline-171.73mg/dL±25.45, after 6 month 150.57mg/dL± 27.14), and HbA1c values, significantly ($p<0.001$) in T2DM patients from Indian Punjabi population. Thus Voglibose is found to be significantly beneficial in T2DM patients having uncontrolled glycemic status with Glimepiride and Metformin.[100]

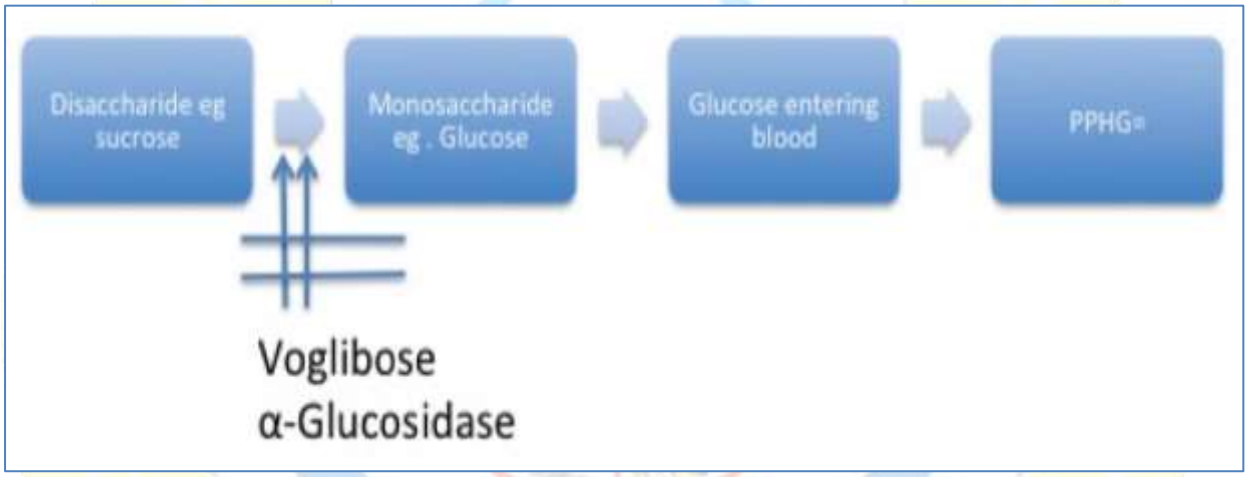

Figure 2. Mechanism of action of Voglibose

Pharmacokinetic characteristics of Voglibose

It has been observed that Voglibose is poorly absorbed after oral dose(<6%) with undetectable plasma concentrations. It is distributed in the lumen of gastrointestinal tract in unchanged form with variable protein binding affinity and is negligibly metabolized by intestinal enzymes and microbial flora.5% is excreted through urine suggesting no dose requirement and 98% is excreted via faeces.[81]Evidence from a study suggest that, Voglibose in combination with Dapagliflozin in Japanese T2DM patients, depictes no difference in C_{max}, $AUC_{0\text{-}inf}$, t_{max} and plasma clearance suggesting the combination to be administered without dose adjustment. .Also, Voglibose aids in increasing half-life of Dapagliflozin from 13.9 hours to 17.9 hours.[101]

Regulatory status of Voglibose

Voglibose is commercially available in Japan since 1994 and is approved in three countries. However it is not approved by US Foods and Drug Administration (USFDA) and European

Medicine Agency (EMA).[3, 102,103]

Objective

Whether used alone or in combination with other blood glucose lowering interventions, the increased number of choices available to practitioners and patients has heightened uncertainty regarding the most appropriate means of treating this widespread condition. Therefore, the consensus statement attempts to provide evidence to establish relationship between regional food variants, post prandial hyperglycemia and AGIs in its management with special focus on Voglibose for curbing glycemic excursions in T2DM patients.

Methodology - Development of the Consensus Protocol

Over 30 national and 74 regional medical experts across 26 cities in India belonging to the specialty of general medicine, internal medicine, and diabetology participated in forty-nine confluence meetings followed by CME attended by 2500 regional health care professionals. The regional committee prepared the context of post prandial hyperglycemia and the national medical expert's sought the consensus statement. The historical evidences and data from the clinical trials were discussed and the necessary amendments were incorporated into the final consensus statement.

Recommendations from the Consensus Meeting

In accordance with the consensus meeting which was conducted with medical experts to understand the role of Voglibose in the management of PPHG, we have outlined the key findings and recommendations that were made by participating physicians.

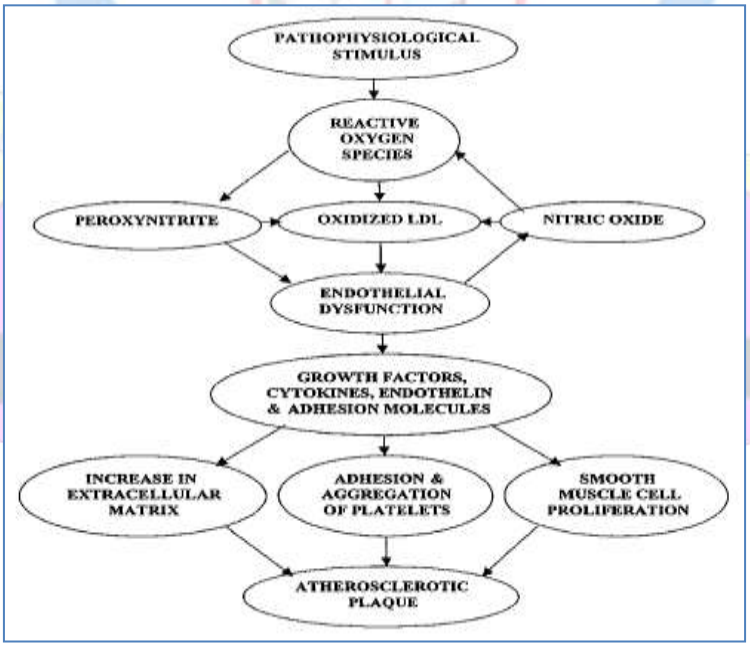

Figure 3: Schematic diagram depicting the involvement of reactive oxygen species, endothelial dysfunction, growth factors, cytokines, and adhesion molecules in the genesis of atherosclerosis.[104]

Importance of PPHG

PPHG is an independent risk factor associated with cardiovascular conditions and is reported to be associated with several cardiovascular complications including postprandial blood pressure, myocardial infarction (MI), CIMT and cardiovascular mortality suggesting that PPHG may be a better predictor of cardiovascular risk than is FPG or HbA1c alone. Furthermore, glycemic spikes associated with PPHG can lead to an increase in oxidative stress, endothelial dysfunction, and inflammatory processes that can together facilitate the development of atherosclerosis (Figure 3). However, PPHG is often underscored and neglected in clinical practice even though it is a more important risk factor in determining cardiovascular risks than FPG or HbA1c alone.

Role of Indian dietary and lifestyle habits on PPHG

- As the Indian diet is rich in carbohydrate, the incidence of PPHG is reported to be greater in the Indian population than the Western world. Traditional Indian diets are carbohydrate-rich; sometimes, as high as 80% of the macronutrient composition. Wheat is largely consumed in Maharashtra and Northern India, while rice and its products predominates the South Indian meal. Furthermore, Indians have sweet tooth resulting in glycemic spikes (Table 5).[102]
- The higher glucose load in the Indian diet leads to greater prandial glycemic excursion, increased glucosidase and incretin in activity in the gut which leads to higher lipemic peaks and associated cardiovascular disease.
- Additionally, consuming large portions and more frequent meals is a common habit observed in Indians thus contributing to higher PPHG levels.
- Lack of exercise is also considered one of the major reasons behind the increased prevalence of diabetes in Indians.

Table 5.Regional variation in food constituents

Region	Meal Time	Typical Meal	Food Constituents
North	Breakfast	Potato parathas with ghee	Rich in carbohydrates and fat (Diet rich in
	Lunch	Puri, chapattis	Carbohydrates and fats raises the glycemic load.
	Dinner[105]	Paratha	High glycemic load results in high PPHG
West[105]	Breakfast	Theplas, poha	Diet high in carbohydrates providing 60 %
	Lunch	Rotis with ghee and white rice, sweets	calories of the total dietary intake
	Dinner	White rice, paratha, roti	
East[105]	Breakfast	Puffed rice, upma	High in calories, total fat and carbohydrates
	Lunch	Cooked rice, sweets	
	Dinner	Cooked rice	
South[105]	Breakfast	Idli, upma	Greater intake of rice which leads to an increase in
	Lunch	White rice	carbohydrate consumption
	Dinner	White rice	

Overall achieving optimal PPHG control is of prime importance as Indians have inherent higher insulin resistance and are more prone to dyslipidemia than Caucasians. Additionally, visceral obesity, metabolic syndrome, and the PPR-γ gene issues are other commonly occurring conditions which contribute to the development of diabetes and increase the risk of early onset of cardiovascular disease. Furthermore, as opposed to Caucasians, Indian diabetic patients have higher PPHG at lower and higher HbA1c values. India is a country with several weddings and festive seasons occurring around the year, in such situations the consumption of fat increases in the form of sweets and oil rich food resulting in increased glycemic load.

Management of PPHG

- Both pharmacological and non-pharmacological interventions are required to manage PPHG. Targets for glycemic control should be individualized (between 140 to 180 mg/dL) based on each patient's clinical status.
- PPHG should be routinely screened in clinical practice and primary care settings given the ease of availability of various glucometers and glucose testing strips. 2-hour PPHG measurements are more commonly used in routine clinical practice and should be the choice for screening post prandial glycemia based on the presence of risk factors.However, in case of pregnancy/gestational diabetes mellitus, a one hour post breakfast/meal is considered important.
- Continuous glucose monitoring (CGM) and meal-based self-monitoring of blood glucose (SMBG) with adequate patient education can help measure the glycemic variability and can thus help monitor blood glucose levels in primary care settings.
- Meal-based SMBG may help patients to monitor their meal choices and portion sizes according to the effect their food has on their glucose levels. Postprandial SMBG values often yield the highest glucose readings of the day and may motivate patients to avoid foods with high glycemic levels, encourage them for optimal physical activity to manage hyperglycemic excursions, or evaluate and adjust insulin doses. Frequency of meal-based monitoring in patients with diabetes should be individualized based upon the PPHG control.

Role of Alpha Glucose Inhibitors

Drugs targeting PPHG are essential to control the post prandial glucose level. These drugs may be used in combination or as a monotherapy. Overall, AGIs are considered effective for treating diabetes in the Indian population,given the predominance of a high carbohydrate diet. AGIs delay the absorption of carbohydrates from the gastrointestinal tract, thereby limiting PPHG excursions.

- AGIs are affordable and have a better safety profile compared to other oral antidiabetics as they do not give rise to hypoglycemia and exhibit no major side-effects except for some gastrointestinal disturbances, the incidence of which can be reduced by increasing the dose gradually and by making a few dietary changes.
- Combination of metformin and AGIs are adopted as they delay gastric emptying, early satiety, decrease glucose spikes and lead to lesser weight gain.However, AGIs should be administered as an add-on therapy after exhausting all other oral hypo glycemic agents.
- Voglibose is preferred amongst the currently available AGI variants as it is better tolerated than Acarbose as produces lesser GI upset. Voglibose delays the absorption of the complex sugars and results in reduction of PPHG. Voglibose increases the release of GLP-1, which enhances the insulin secretion from the pancreatic beta cells and also increases the sensitivity of insulin. Additionally, Voglibose in combination with diet and other drugs reduces visceral fat deposition and ameliorates insulin resistance and hence, is preferred in obese patients uncontrolled on metformin and sulfonylureas.
- Voglibose can also be considered as a first-line drug in patients who exhibit a striking disparity between their fasting and postprandial plasma glucose levels. In western populations Voglibose reduces the HbA1c levels around 0.6%-0.8% but in Indians it might be $\geq$1% as carbohydrate consumption in west is 30%-40% as opposed to 70% in Indians 70%.
- Voglibose in combination with metformin is ideal for obese diabetics. When voglibose is used along with insulin, the dose of the latter can be reduced, thereby reducing the risk of hypoglycemia significantly
- Voglibose-based triple drug therapy, i.e. Voglibose in combination with Metformin and Sulfonylurea, is suitable for patients with deranged fasting blood glucose, higher PPHG levels, patients with HbA1c ranging between 7%-7.5%, and in cases where blood glucose
- levels remain uncontrolled or where aggressive blood glucose control is expected, as well as in chronic diabetic patients taking these medications as separate drugs.
- Combination of Voglibose, Metformin and Glimepiride is suitable for the management of reactive hyperglycemia, considering that it's unlikely to increase the endogenous secretion of insulin in the second phase after a meal.
- The combination of Metformin, Sulphonylurea, and Voglibose can be considered for newly diagnosed patients as well as for patients with existing diabetes depending on the HbA1c value and meal patterns. However, triple-drug therapy containing Voglibose should be started only when monotherapy and dual-drug therapy have failed to produce adequate glycemic control.

Concerns with the use of Voglibose in managing PPHG

- The only concern with the use of Voglibose is gastrointestinal suffering such as bloating, flatulence, steatorrhea, and diarrhea which can be avoided by starting with a low dose and gradually elevating it.
- Dual drug therapy may cause hypoglycemia. Thus it is advisable to carry glucose sachet or hypotablets.
- In cases of nephropathy, Voglibose can be used only in cases of mild and moderate renal failure and not in more severe cases.

Dietary recommendations

- High fiber and low-carbohydrate diet comprising largely of fruits, vegetables, and proteins should be included in day to day living. Patients should try to decrease carbohydrate intake to 40-50%.
- Consumption of refined carbohydrates should be avoided.
- Brown rice should be preferred over polished white rice.
- Four to five small meals containing food that are low in their GI are recommended compared to two moderately heavy meals.

Lifestyle recommendations

- Patients should be advised to walk for few minutes (15-30minutes) after every meal, and must be coaxed into indulging in muscle-strengthening exercises on a regular basis.
- Lastly, measures should be taken to improve awareness, regarding CVD risk with PPHG, among other practicing clinicians who treat diabetes patients as well as in diabetic patients.

CONCLUSION

PPHG is an independent risk factor of cardiovascular complications in diabetes patients and a better predictor of glycemic control than the FPG. Patients who are seemingly well controlled with diet, exercise, and medical therapy, even those with normal FPG and HbA1c could have uncontrolled PPHG. The combination of improved detection and monitoring of PPHG along with effective medications may help to establish optimal glycemic control thereby reducing diabetic complications. The ideal treatment for patients with T2DM should include a combination of agents that lower basal plasma glucose levels and agents that control meal-related glucose excursions. The success in maintaining the glycemic and perhaps, the lipid excursion must be acknowledged as the therapy target. Most available treatments reduce fasting glycemia, but have a lesser effect on the PPHG excursions. Considering high carbohydrate content in the Indian diet, AGIs are effective in treating Indian diabetics patients especially Voglibose as it is effective in controlling PPHG in Indian population when

administered as monotherapy or in combination with other drugs. The availability of new agents directed at the postprandial state as the aforementioned ones, leads to the possibility of better long-term management of patients with T2DM, aiding in the prevention of the high morbidity-mortality associated to diabetes, especially cardiovascular disease.

Key points

•PPHG plays a vital role in glycemic control thus controlling PPHG, can reduce the risk of cardiovascular diseases

•AGIs have definitive role in the management of PPHG with no major side effects except for the gastrointestinal symptoms

•AGIs are cost effective, and have less hepatic side effect

•Voglibose is one of the most preferred commercially available AGI

•It can be preferred as a monotherapy in thin diabetics and its combination with metformin would be ideal for obese diabetics

For the Indian population, Voglibose in combination with Metformin or Sulfonylurea, is suitable when fasting blood glucose, PPHG levels and HbA1c are suboptimal

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