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Review

Nutritional strategies in managing postmeal glucose for type 2 diabetes: A narrative review

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ABSTRACT

Medical Nutrition Therapy (MNT) plays an essential role in overall glycemic management. Less focus is given on managing postmeal hyperglycemia despite the facts that, it is a common feature of Type 2 Diabetes (T2D). The purpose of this narrative review is to provide a comprehensive understanding of the existing literature on the nutritional approaches to improve postmeal hyperglycemia in patients with T2D. We searched multiple databases for the studies examining the nutritional approaches to manage postmeal glucose in patients with T2D. We included studies that involve human trials that were published in English for the past 10 years. Our review of the current literature indicates that the postmeal hyperglycemia can be improved with four nutritional approaches. These approaches include (i) utilizing the appropriate amount and selecting the right type of carbohydrates, (ii) using specific types of dietary protein, (iii) manipulating the meal timing and orders and (iv) others (promoting postmeal physical activity, incorporating diabetes-specific formula and certain functional foods). The potential mechanisms underlying these approaches are discussed and the identified gaps warranted further research. This array of nutritional strategies provide a set of options for healthcare professionals to facilitate patients with T2D in achieving the optimal level of postmeal glucose.

1. Introduction

Medical Nutrition Therapy (MNT) is a key component in diabetes management. While the role of MNT to improve the overall glycemic level as assessed using HbA1c level has been established, the specific strategies to achieve optimal postmeal glycemic level is uncertain. The postmeal or postprandial hyperglycemia is a common feature of Type 2 Diabetes (T2D) with the majority of the patients with T2D have not achieved the postmeal target of <7.8 mmol/L at 2 h following meals ingestion [1]. The fact that humans spend much of their time in the postprandial state rather than at the fasting state and therefore, nutritional strategies which designed to improve postmeal glycemia deserves clear attention.

Nevertheless, postmeal hyperglycemia occurs frequently among T2D patients. A comparative study in 60 male T2D patients found that postmeal hyperglycemia is 38 ± 4% more prevalent in T2D patients throughout the day as compared to normal glucose tolerant individuals. Even those T2D patients with HbA1c level of below 7.0%, they still experienced glycemic surge for nearly 6 h per day as assessed by 24 h glycemic profiles [2]. In the South East Asia region, Malaysian T2D patients shown the highest concentration of elevated meal postmeal glycemic level of 12.7 mmol/L [3].

Patients with T2D were advised on lifestyle management such as nutritional therapy and increase physical activity before they were administered on pharmacologic treatment. Current diabetes meal-plan recommends portion control and healthful food choices [4], yet the glycemic control of Malaysian T2D patients were unsatisfactory [3] with worsening HbA1c level from 8.0% in 2003 to 8.9% in 2018. In addition [5], reported that 426 (39.6%) out of 1077 T2D patients in Malaysia did not achieve the optimal postmeal

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glycemic level of <10 mmol/L. Hence, the current state of the art in diabetes-nutrition does not support the improvement in postmeal glycemia.

Excessive rise in postmeal glucose increases the risk of cardiovascular disease (CVD) in T2D patients [6]. The physiological influence that occurs in T2D patients during acute glycemic spikes is oxidative stress [1] and deleterious effect on endothelial function [7]. Eventually, these will lead to the development of atherosclerosis [8].

Most of the previous reviews discussed the clinical aspect of the management of postmeal glucose [1,9–11] tended to focus on the role of pharmacologic therapies in managing postmeal glycemic level of T2D patients rather than nutritional approach. On the other hand [12], summed up the strategies to improve postmeal hyperglycemia by looking into the American culture [13]; discussed the impact of dietary components on postmeal glycemic level [14] summarised the dietary approaches and its relation with oxidative stress and CVD risk, indirectly focusing on the postmeal state; whereas [15,16] viewed the management of postmeal glycemia from the point of exercise. In this review, we aim to identify specific nutritional approaches that optimise the postmeal glycemia for patients with T2D. Identification of optimal nutritional approaches may guide healthcare professionals in their recommendation to lead T2D patients in achieving optimal postmeal glucose.

2. Methods

The literature search was conducted using multiple databases including Science Direct and PubMed for studies examining the nutritional strategies to manage postmeal glycemia in patients with T2D. We utilised the MeSH (Medical Subject Headings) terms of "postprandial glycemia" OR "postmeal glucose" AND "Type 2 Diabetes Mellitus" AND "nutritional approaches" OR "dietary strategies". We restricted our search to study related only to humans that were published in English from the year 2009–2019. Studies that were included in the review followed the study designs: randomized clinical trials, prospective observational studies and review papers that are specifically looking into the nutritional approaches to manage postmeal glycemic level. Studies were excluded due to not fulfilling the selection criteria, not applicable to research questions and duplicate publications.

3. Results

The flowchart of literature selection is presented in Fig. 1.

A total of 61 papers were included in this review. All the information retrieved from the literature search were organized in common themes together, meanwhile the results of studies were reported as postmeal glycemic or insulinemic responses as assessed by incremental area under the curve (iAUC). In sum, our review of current literature indicates that postmeal hyperglycemia can be modulated with four nutritional approaches. It includes utilizing the appropriate amount and selecting the right type of carbohydrate, using specific dietary protein, manipulating meal timing and orders, and others (e.g. promoting postmeal physical activity, incorporating diabetes-specific formula and certain functional foods).

4. Utilizing the appropriate amount and selecting right type of carbohydrate

Dietary carbohydrate is the main macronutrient influencing the postmeal glycemic level, especially in patients with T2D. There are three main types of carbohydrate which are starches, sugars and fiber. Upon digestion and absorption of carbohydrate, the elevation of blood glucose in the bloodstream will trigger the pancreatic β-cell to produce insulin. However, patients with T2D commonly had insulin resistance or insulin insensitivity whereby the insulin is not effective to lower blood glucose, causing the blood glucose to consistently rise in the postmeal state, resulting in postmeal hyperglycemia.

Dietitian will advise patients with T2D to control the portion size of carbohydrate as the first step to manage their blood glucose level. T2D patients will be educated on the plate method, carbohydrate exchange system or carbohydrate counting that is practical for patients to adhere with. In order to further optimize the glycemic control, the type of carbohydrate which is assessed by the Glycemic Index (GI) and Glycemic Load (GL) can be implemented.

A considerable amount of reviews had been published on the effectiveness of reducing the amount of carbohydrate consumed and selection of low GI food in managing postmeal glycemic level [11,13,17,18]. While the proper amount of carbohydrate becomes questionable, there were studies demonstrated on the different amount of carbohydrate and their effect on postmeal glucose.

In Sweden, a randomized crossover trial carried out by [19] among 19 in patients with T2D found that low carbohydrate diet (16–24% from total energy) induced lower insulin and glucose excursions throughout the day compared to low-fat diet comprised of 45–56% carbohydrate. However, this study was a short term that does not necessarily show subtle changes over time. Besides, a total of 104 T2D patients were randomly provided with (i) high carbohydrate/high GI, (ii) high carbohydrate/low GI or (iii) low carbohydrate/high MUFA meals for one year. The macronutrient distributions of the diet were ~55% carbohydrate, ~15% protein and ~30% fat of total energy intake. In order to investigate the acute effects of meals on postmeal responses, after the patients complied to their respective study diet for 1 year, half of them were assigned to the test meals of their study diet; the other half were required to take baseline meal which is high carbohydrate/high GI. The results indicated that a high CHO/low GI meal exert both acute and chronic effects on postmeal glucose fluctuations compared to the other groups. Nonetheless, this study was not able to demonstrate differences in total iAUC among groups possibly due to metabolic adaptation over time.

Dietary fiber had been illustrated to have distinct properties in lowering postmeal glucose level [20]. Dietary fiber can be found in whole grain foods, legumes and oatmeal. A systematic review and meta-analysis of 9 crossover studies demonstrated that oatmeal with β-glucan significantly reduced acute postmeal glucose and insulin responses [21]. In China, compared to low fat and high fiber diet, an improvement in postmeal glucose were found in overweight T2D patients when they replaced cereals with either 50 g or 100 g of whole grain oats for 30 days. This study was followed up to 1 year and the subject group consuming 100 g oats had a bigger reduction in postmeal glucose level [22]. On the other hand, when substituting cereals with 60–100 g of oats per day for 8 weeks, it did not show any effect on postmeal glycemic or insulinemic response [23]. The contrast of both findings showed that substitution of carbohydrate foods without lowering the GI or GL would not modulate postmeal hyperglycemia.

GI provides ranking to a specific food based on the postmeal blood glucose response after ingestion of 50 g available carbohydrate [24]; whereas GL combines both amount and GI of carbohydrate foods. There are three categories to group the GI values of carbohydrate food (i) low GI: <55, (ii) intermediate GI: 56–69 and (iii) high GI: >70; while (i) low GL: <10, (ii) intermediate GL: 11–19 and (iii) high GI: >20. The GI and GL value of carbohydrate foods were influenced by several factors such as the natural characteristics of the starch (ratio of amylose to amylopectin), type of rice, post-harvest treatment, consumer preparation and consumption

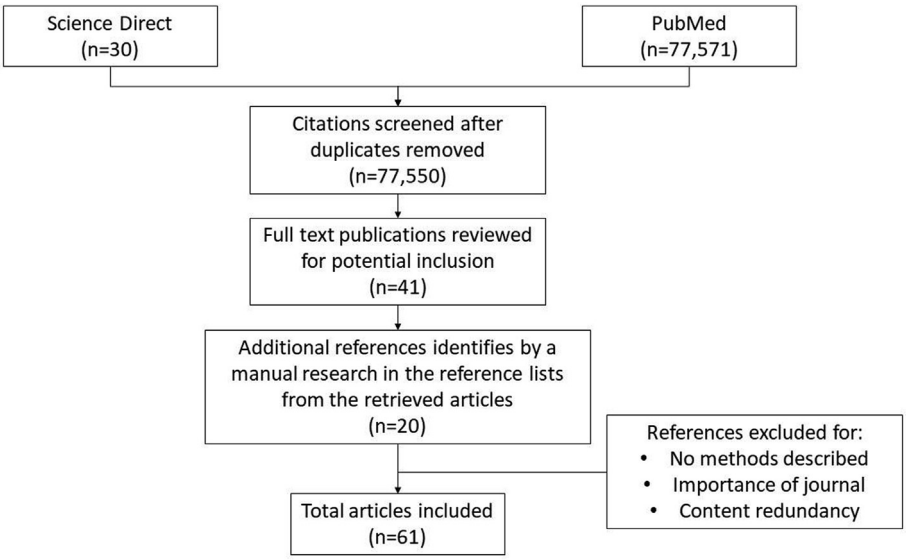

Fig. 1.

[25] as well as amount and type of dietary fibre.

Numerous studies had demonstrated the effectiveness of a low GI diet in improving postmeal glycemic response among T2D patients, in line with the recommendation from the International Diabetes Federation [11,17,26,27]. In a randomized crossover study of 14 T2D patients of mean HbA1c 6.6%, breakfast that is high GI (60.9 ± 1.7) and low fiber ($2.5\,g \pm 0.4$) reported less favourable to postmeal glycemic and insulinemic responses compared to other meals with different GI or fiber [28].

Rice is the typical staple source of carbohydrate for the Asian population, so it mainly contributes to the dietary GI and GL. Due to the differing country origin, there are different varieties of rice. In India, the researcher studied on the foxtail millet dosa [29]; whereas in Japan, the researcher studied on glutinous brown rice [30], both types of rice had a lower GI value compared to white rice. These two randomized crossover studies illustrated a significant reduction in the postmeal glucose level in patients with T2D. Not only that, a combination of bean (e.g., pinto beans, black beans or red kidney beans) with long grain rice also lowered the iAUC of glucose [31]. In Malaysia, acute crossover study among 41 T2D patients with mean HbA1c of 7.8% reported that low GI meal (GI:37, GL:13) significantly reduced postmeal glycemic level by 39% and insulin responses by 26% [32]. This study was extended to a 3-month period in order to evaluate the effect of low GI meal on the postmeal glycemic responses. Comparing to isocaloric conventional carbohydrate exchange diet, low GI diet (GI value from 63 to 57) induced lower postmeal glycemic excursions at 0, 60, 150 and 180 min [33].

In sum, this combination of findings may help us to understand the effect of modulating amount and type of carbohydrate in order to achieve optimal postmeal glycemic control in patients with T2D. Further research should be undertaken to establish the underlying metabolism of carbohydrate or glucose towards the pathophysiology of T2D, while this can be accomplished by analysing the metabolites perturbation through metabolomics approach.

5. Specific dietary protein

Moving on, co-ingested macronutrient (i.e., dietary protein) has been shown to influence the magnitude of the glycemic excursion at postmeal state other than carbohydrate ingestion. This effect is also known as a mixed meal effect, whereby this macronutrient lengthens the time for digestion. In addition to the amount of protein, the quality and timing of protein ingestion are important factors affecting postmeal glycemic excursions.

In terms of the amount of protein, a randomized crossover trial of 12 T2D patients with mean HbA1c of 7.1% illustrated that high-protein breakfast (35% protein, 45% carbohydrate) reduced 17% of postmeal glucose level and iAUC of the glucose-dependent insulinotropic peptide (GIP). GIP is an incretin hormone secreted from the intestine to stimulate pancreatic insulin secretion. Meanwhile, a 23% reduction in both parameters was observed when the patients ingested isocaloric high carbohydrate meal (15% protein and 65% carbohydrate) [34]. This study revealed the need for further investigation of the quantity of protein that should be prescribed to optimize the postmeal glycemic control of T2D patients.

There were few reviews highlighted on the provision of type protein, specifically whey protein, in the management of postmeal glucose level in patients with T2D [35,36]. Turning now to the experimental evidence, a 12-week randomized clinical trial conducted in 48 Israel patients with T2D, demonstrated that high energy breakfast based on whey protein diet (28 g) reduced 19% of iAUC glucose compared to protein sources from eggs, tuna and soy which reduced 12% of iAUC glucose levels. Also, the overall iAUC for insulin and glucagon-like-peptide (GLP-1) were greater in the whey protein diet group [37].

Regarding the timing of whey protein ingestion, [38] reported that 25 g whey preloads before each of three main meals significantly lowered 4 h postmeal glycemia and slowed gastric emptying rate. This effect persisted up to 4 weeks of exposure. Moreover, premeal consumption of protein-enriched (9.3 g of whey protein) bar significantly lowered iAUC of plasma glucose from 0 to 180min than with postmeal consumption in 15 Korean T2D patients. At the same time, the insulinogenic index and total GLP-1 levels were higher, indicating early phase insulin secretion. Another significant finding by [39], they observed 48% reductions in peak glucose, 28% reduction in iAUC glucose and 22% increment of insulin secretion after a protein and lipid preload of 50 g Parmesan cheese and one boiled egg. In contrast with the previous studies mentioned, this study assessed the effect by measuring the oral glucose tolerance test (OGTT) level.

To conclude, the literature identified the role of dietary protein

in managing the postmeal glycemic excursions in patients with T2D. Not only that, the type and timing of protein ingestion need to be taken into consideration when prescribing dietary advice to the patient in order to achieve optimal glycemic control.

6. Manipulating meal timing and orders

Most of the metabolic pathway involved in glucose homeostasis is controlled by the circadian clock, whereby the circadian clock is synchronized with the meal timing [40]. Hence, it is necessary to address the meal timing as it affects the whole day glycemic excursions instead of the solely postmeal state.

More recent attention has focused on the importance of breakfast consumption because skipping breakfast leads to a significant increase in HbA1c level and 24 h postmeal hyperglycemia even without overeating in the previous evening [41]. With respect to this statement, T2D patients who did not take breakfast illustrated 37% and 15% higher iAUC for plasma glucose and glucagon, while 17% and 19% lower iAUC for insulin and GLP-1, respectively during lunch [42,43]). Then, in a randomized crossover trial of 18 patients with T2D where mean HbA1c is 7.6%, it was reported that high energy breakfast lowered 24% $iAUC_{0-180min}$ for postmeal glucose and increased 11% $iAUC_{0-180min}$ for insulin. This glucose lowering effect was persisted throughout the day indicated that meal timing is a crucial factor in the improvement of overall glycemia [42,43]). These findings were in line with a similar study performed by [34].

Interestingly, several studies looked into the temporal sequence of meal consumption, which found an effect on postmeal glycemia in patients with T2D. A pilot study was conducted among 11 T2D patients using a typical Western meal to determine the impact of food order on postmeal glycemic surge. When the patients consumed vegetables and protein before carbohydrate food, the mean postmeal glucose levels were decreased by 28.6%, 36.7% and 16.8% at 30, 60 and 120min, respectively. Not only that, insulin excursions were also significantly reduced, likely to show improvement in insulin sensitivity through this meal pattern [44]. Then, this study was extended to confirm the effectiveness of consuming carbohydrate last in attenuating postmeal glucose level, compared to taking carbohydrate first or all components together without sequencing [45].

In Japan, an acute study of ingesting fish or meat before rice significantly improved postmeal glucose excursions compared to ingesting rice first. Furthermore, this study found delayed gastric emptying rate and enhanced incretin (GLP-1) secretion when patients consumed fish or meat before rice, suggesting the possible explanations on the mechanisms [46]. Another long-term study recruited 17 T2D patients under free-living conditions, they were instructed to consume fat and protein before carbohydrate during lunch and dinner. After 8 weeks, there was a significant reduction in mean 2 h postmeal glucose excursion as assessed by self-monitoring blood glucose (SMBG) system.

The previous research had mainly discussed the sequence of carbohydrate, protein and fat in ameliorating postmeal glycemic response. There were relatively few studies examining the sequence of vegetable intake in a meal. Acute study in Japan demonstrated a beneficial reduction in postmeal glucose and insulin in patients with T2D when they consumed vegetables before carbohydrate [47]. This simple-to-follow advice was implemented and studied for 2 years period, where T2D patients were encouraged to ingest green vegetables at least once per day and eat them slowly. The similar findings were also reported [48]. The beneficial effect of this advice can be attributed to increase dietary fiber intake as described previously on the role of dietary fiber in managing postmeal glycemic level.

An implication of these findings raises the possibility to emphasize on proper meal timing and meal sequence to patients with T2D during diet consultation by the healthcare professional. It was evidenced that by adopting an appropriate dietary habit and meal pattern consistently, T2D patients are able to optimize their postmeal glycemic control.

7. Others (promoting postmeal physical activity, incorporating diabetes-specific formula and certain functional foods)

Despite focusing on nutritional approaches to lower postmeal hyperglycemia, the postmeal walk is reported as an effective strategy in managing the glycemic level at postmeal state. There were three latest studies retrieved. Firstly, a randomized crossover trial was conducted in 41 elderly T2D patients with mean HbA1c of 7.5%. This study found that advice to walk 10min after each main meals shown a 12% improvement in iAUC of glucose than when the timing of physical activity was not specified [49]. Besides, a crossover trial in China sought to determine the effect of a post-dinner walk in ameliorating the postmeal glycemic response until the next morning (12 h) by using a continuous glucose monitoring (CGM) system. Although there were no differences during the exercise session, the analysis revealed that after 1 h exercise, the total iAUC glucose was significantly reduced, then the 2 h postmeal glucose spike, peak glucose and mean glucose were also reduced when patients exercise after dinner. No difference was observed in the 12 h glycemic excursions in this study [50]). An interesting result was demonstrated by a randomized controlled trial conducted in 11 Canadian T2D patients. When the strategies of postmeal walking after each meal and a low carbohydrate diet (10% from total energy) were combined, the glucose iAUC as assessed by CGM system was significantly reduced by 94% compared to 86% reduction in only low carbohydrate diet or low fat control diet (55% carbohydrate from total energy) without postmeal walking [51].

In recent years, there has been an increasing interest in determining the effect of diabetes-specific formula (DSF) on the postmeal glucose management in patients with T2D. First of all, looking into some acute studies, a randomized crossover study in India found that replacing breakfast with DSF was able to lower postmeal iAUC of glucose and insulin in 40 T2D patients with moderate glycemic control (HbA1c <9%), as compared with isocaloric breakfast comprised of cornflakes and milk [52]. Next, reduction in postmeal iAUC of glucose was observed in a group of elderly T2D patients who ingested DSF in comparison to isocaloric oatmeal. At the same time, this study illustrated higher postmeal GLP-1 secretion, which reflected insulin stimulation by the pancreatic cell [53].

On the other hand, a 4-week study in the Netherlands compared two formulas which were DSF vs standard, fiber-containing formula. Results showed that DSF lowered only the postmeal glucose response but not other parameters such as postmeal insulin and insulin resistance [54]. Furthermore, a similar result was portrayed in a 3-month study on 40 elderly T2D patients [55]. Despite these inconsistent findings with the previous acute studies, it is important to take note these studies recruited T2D patients from older age group, and they were on insulin therapy. It is possible that the deterioration of pancreatic β-cell is more extensive in this group than those who were on oral antidiabetic agents. Moving on, researchers attempted to evaluate the postmeal glucose excursions over a 3-month period by using a CGM system. In T2D patients who replaced breakfast with DSF, there was a 37% reduction in the postmeal glucose surge [56].

There is a growing body of literature that recognises the importance of functional food ingredient in managing the postmeal

glycemic spike in patients with T2D. A review conducted by [57] examined the effect of berries and anthocyanins in lowering postmeal glycemia response by explaining the possible mechanism involved in carbohydrate digestion. Addition of 40 g dried cranberries in breakfast resulted in a significant reduction of postmeal glycemic excursions in 25 American T2D patients, but no difference was found regarding insulin resistance [58]. Nopal (*Cactus Opuntia*) is a traditional medicine use by the Mexican to treat diabetes. When patients with T2D consumed 300 g steamed nopal together with usual breakfast, the iAUC for glucose and insulin were significantly reduced [59]. Functional food ingredient varies depending on origins, ethnic and cultural practice. In Malaysia, the medicinal plants *Cosmos Caudatus* (*Ulam raja*) was studied on its anti-diabetic properties. Consumption of *Ulam Raja* for 8 weeks significantly improved HbA1c level, insulin resistance and insulin sensitivity [60]. A further study with more focus on postmeal hyperglycemia is therefore recommended.

Overall, these studies indicated that promoting postmeal physical activity, incorporating diabetes-specific formula and certain functional foods had shown a profound effect on postmeal glycemic response. It is crucial for the healthcare professional to consider the sociodemographic characteristics (i.e., age, family support, socioeconomic status, etc.), motivation level and other comorbidities when suggesting these approaches to the patient. For instance, although high-intensity interval exercise was reported to lower postmeal glycemic increment compared to moderate intensity continuous exercise [61], this should be individualised based on patient's age, strength and flexibility, and other clinical complications.

8. Discussion

Postmeal glycemia, also known as glycemic variability, can provide information on the amplitude, duration and frequency of glycemic excursions neither postmeal nor the whole day [27]. The development of postmeal hyperglycemia is linked to the pathophysiology of T2D, where there is deterioration in the pancreatic β-cell function [62] and deficiency in the incretin hormones (GLP-1 and GIP) secreted from the gastrointestinal tract (GIT) [63]. Both of these conditions progressively decline the insulin stimulation and secretion, resulting in elevated glucose level in the bloodstream. Moreover, lack of insulin sensitivity or insulin resistance causes delayed uptake of glucose in the peripheral tissues; meanwhile triggered hepatic gluconeogenesis [62]. To develop a full picture of optimal glycemic control in T2D patients, it is necessary to include postmeal hyperglycemia into the T2D management, despite the routine assessment of fasting plasma glucose and HbA1c level.

Altering the amount and type of carbohydrate are well established as the mainstay of nutritional strategy to manage postmeal hyperglycemia. A diet that is moderate in the amount of carbohydrate, low in GI and GL, and high in soluble dietary fiber are promising to improve postmeal glycemia in T2D patients. The mechanism underlying this diet can be explained by a slower rate of gastric emptying [64], lead to a slower rate of carbohydrate absorption in the GIT, thereby compensating the delay in insulin secretion and insulin resistance [65], at last, exerted a positive impact on postmeal glycemic response [66].

Although this strategy was reported to be effective, implementing it may be challenging issue in Malaysia. The self-care practices among T2D patients were not satisfying. Among 126 T2D patients with suboptimal glycemic control, 80% consumed more than four meals per day. Meanwhile, 60% of them consumed more than two exchanges of carbohydrate per snack, which is more than the recommended daily allowance [67]. Besides, almost 84% of the patients did not adhere to the dietary recommendations provided by dietitians. Hence, in order to achieve the target postmeal glucose, their dietary challenges need to be understood.

Furthermore, dietary protein plays a role in modulating postmeal hyperglycemia. There are two phases of insulin secretion: first phase insulin happened 10 min after meal consumption, followed by second phase that lasted for 3–4 h. These macronutrients were digested later compared to carbohydrate. Thus, they are able to delay the peak glucose response and sustain late postmeal hyperglycemia [18]. On top of that, whey protein had been studied on its insulinotropic effect in attenuating postmeal glycemia surges [68]. The bioactive peptides or amino acids generated during digestion of whey protein may inhibit the enzyme dipeptidyl-peptidase-4 (DPP-4) and enhance incretin effect, leading to pancreatic insulin secretion and improving postmeal glycemic control [37]. Besides, whey protein is a rapid-acting protein and contains higher amount of branched chain amino acids, which also support the insulinotropic effect to reduce postmeal glucose level [37].

Consistent meal timing, in a particular breakfast consumption, can affect the glycemic fluctuation not only at the postmeal state but the subsequent meal of the day. This can be explained by a phenomenon called “second meal phenomenon” [69]. The first meal of the day, which leads to glucose elevation, can enhance the responsiveness and memory of pancreatic β-cell to work effectively in stimulating the incretin and insulinotropic effects [42,43]). Moreover, manipulating meal sequence can play a role in postmeal glycemic control by delaying gastric emptying rate and enhancing insulin secretion through incretin effect. Not only that, altering meal sequence can be attributed to increasing dietary fiber intake or satiating effect stimulated by protein and fat, lead to the overall reduction in total energy intake and improvement in postmeal glycemic control [48].

To date, the replacement of a meal with DSF has been gaining attention from the researchers and the public. The DSF is specially formulated to suit the nutritional requirement of T2D patients, mainly targeting to achieve optimal postmeal glycemic control. Several properties of the DSF contributes to better glycemic control which include low GI, high fiber, blend of high-quality protein (50% whey and 50% casein) and a special fat blend of monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA). Thus, the DSF can be recommended as a meal replacement for T2D patients.

Patients with T2D are always be reminded on the importance of physical activity in mitigating postmeal glycemic excursions. Exercise is clinically evidenced to improve insulin sensitivity, increase glucose uptake in skeletal muscle and decrease hepatic glucose production [15]. The current guideline did not specify the timing of physical activity, where there had been some evidence reporting on the benefit of implementing exercise after each of the main meal. When prescribing the level of exercise, it should be started with the modest level, so that the results apply to a broad group of patients, and they are motivated to participate [49]. Also, it is essential for the healthcare professionals to educate and monitor the risk of hypoglycemia when a patient performs physical activity.

Various studies had assessed the efficacy of functional food in lowering postmeal glycemic response of T2D patients. The functional food is usually identified as a traditional medicine where their magnitude of effect on postprandial glycemic excursions is comparable to that observed with pharmacological agents that target on postmeal glucose. This positive impact observed was likely to be related to the bioactive compound (e.g., anthocyanins, proanthocyanidins) that inhibit the activity of carbohydrate digestive enzyme, lead to a slower absorption of glucose in the GIT and modulate postmeal glucose metabolism [57,58].

The combinationof findings provide some support for developing an integrated approach targeting specifically on the postmeal

glycemia in patients with T2D. However, the precise mechanism of implying these strategies on glucose metabolism remains to be elucidated and warrants further investigation. The metabolomics technique is a newly emerging tool to study the metabolic perturbation of bioactive metabolites in human body. The identified metabolites can be mapped onto the metabolic pathway to seek association and explanation on relevance metabolites with the postmeal glucose metabolism. Future research should discover the postmeal metabolites that contribute to glycemic spike as it allows comprehensive understanding on the interaction between nutrient and metabolism.

9. Conclusion

In conclusion, postmeal hyperglycemia warrants to be highlighted in the management of patients with T2D as it is evidenced to be the independent risk factor of CVD, which eventually leads to complications and mortality. Before proceeding to pharmacologic treatment such as Acarbose, improvement in several nutritional aspects shown to exert immediate favourable changes in postmeal hyperglycemia. In sum, the postmeal glycemic response can be modulated by these four factors which includes utilizing the appropriate amount and selecting the right type of carbohydrate, using specific dietary protein, manipulating meal timing and orders, and others (e.g. promoting postmeal physical activity, incorporating diabetes-specific formula and certain functional foods). The evidence focused here are mainly dealing with certain kind of nutritional strategy, but the effect of combined strategies remains unanswered. In addition, it is recommended to conduct a long term study and assess the glycemic variability by using the CGM system. Nonetheless, this narrative reviews provide a suite of intervention options for the management of postmeal hyperglycemia in patients with T2D by the healthcare professionals. Not only that, intensive education and empowerment on self-management have to be emphasized in order to increase awareness and exert more profound efforts about optimal glucose profiling.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.dsx.2019.05.026.

References

[1] Gerich J. Pathogenesis and management of postprandial hyperglycemia: role of incretin-based therapies. Int J Gen Med 2013. https://doi.org/10.2147/IJGM.S51665.
[2] van Dijk JW, Manders RJF, Hartgens F, Stehouwer CD, Praet SFE, van Loon LJC. Postprandial hyperglycemia is highly prevalent throughout the day in type 2 diabetes patients. Diabetes research and clinical practice. 2011. https://doi.org/10.1016/j.diabres.2011.03.021.
[3] Mohamed M. An audit on diabetes management in Asian patients treated by specialists: the Diabcare-Asia 1998 and 2003 studies. Curr Med Res Opin 2008;24(2):507–14. https://doi.org/10.1185/030079908X261131.
[4] Medical Nutrition Therapy for Type 2 Diabetes Mellitus. Medical nutrition therapy for type 2 diabetes Mellitus. 2012.
[5] Abougalambou SSI, Ahmed NO, Abougalambou AS. A study evaluating the postprandial plasma glucose control among type 2 diabetes patients attending a teaching hospital in Malaysia. Diabetes, Metab Syndrome: Clin Res Rev 2017;11:S445–9. https://doi.org/10.1016/j.dsx.2017.03.033.
[6] Cavalot F, Petrelli A, Traversa M, Bonomo K, Fiora E, Conti M, et al. Postprandial blood glucose is a stronger predictor of cardiovascular events than fasting blood glucose in type 2 diabetes mellitus, particularly in women: lessons from the San Luigi Gonzaga diabetes study. J Clin Endocrinol Metab 2006. https://doi.org/10.1210/jc.2005-1005.
[7] Watanabe K, Suzuki T, Ouchi M, Suzuki K, Ohara M, Hashimoto M, et al. Relationship between postprandial glucose level and carotid artery stiffness in patients without diabetes or cardiovascular disease. BMC Cardiovasc Disord 2013. https://doi.org/10.1186/1471-2261-13-11.
[8] Node K, Inoue T. Postprandial hyperglycemia as an etiological factor in vascular failure. Cardiovasc Diabetol 2009;8:1–10. https://doi.org/10.1186/1475-2840-8-23.
[9] Kota S, Modi K, Satya Krishna S. Glycemic variability: clinical implications. Indian J Endocrinol Metabol 2013;17(4):611. https://doi.org/10.4103/2230-8210.113751.
[10] Sheu WHH, Rosman A, Mithal A, Chung N, Lim YT, Deerochanawong C, et al. Addressing the burden of type 2 diabetes and cardiovascular disease through the management of postprandial hyperglycaemia: an Asian-Pacific perspective and expert recommendations. Diabetes Res Clin Pract 2011;92(3):312–21. https://doi.org/10.1016/j.diabres.2011.04.019.
[11] Zenari L, Marangoni A. What are the preferred strategies for control of glycaemic variability in patients with type 2 diabetes mellitus? Diabetes Obes Metab 2013;15(S2):17–25. https://doi.org/10.1111/dom.12143.
[12] O'Keefe JOJH, Gheewala NM, O'Keefe JOJH. Dietary strategies for improving post-prandial glucose, lipids, inflammation, and cardiovascular health. J Am Coll Cardiol 2008;51(3):249–55. https://doi.org/10.1016/j.jacc.2007.10.016.
[13] Gao D, Vinoy S, östman E, Lahtinen S, Griffiths HR, Lansink M, et al. Impact of diet composition on blood glucose regulation. Crit Rev Food Sci Nutr 2013;56(4):541–90. https://doi.org/10.1080/10408398.2013.792772.
[14] Sottero B, Gargiulo S, Russo I, Barale C, Poli G, Cavalot F. Postprandial dysmetabolism and oxidative stress in type 2 diabetes: pathogenetic mechanisms and therapeutic strategies. Med Res Rev 2015. https://doi.org/10.1002/med.21349.
[15] Kearney ML, Thyfault JP. Exercise and postprandial glycemic control in type 2 diabetes. Curr Diabetes Rev 2016;12(3):199–210. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/26073704.
[16] Stephenson EJ, Smiles W, Hawley JA. The relationship between exercise, nutrition and type 2 diabetes. Diabetes Phys Act 2014;60:1–10. https://doi.org/10.1159/000357331.
[17] Ceriello A, Barakat M, Bahendeka S, Colagiuri S, Gerich J, Hanefeld M, et al. Guideline for management of postmeal glucose in diabetes. Diabetes Res Clin Pract 2014;103(2):256–68. https://doi.org/10.1016/j.diabres.2012.08.002.
[18] Hamdy O, Barakatun-Nisak MY. Nutrition in diabetes. Endocrinol Metab Clin N Am 2016;45(4):799–817. https://doi.org/10.1016/j.ecl.2016.06.010.
[19] Nystrom FH, Guldbrand H, Jaredsson C, Bunjaku B, Rosenqvist U, Fernemark H. A randomized cross-over trial of the postprandial effects of three different diets in patients with type 2 diabetes. PLoS One 2013;8(11):e79324. https://doi.org/10.1371/journal.pone.0079324.
[20] Tessari P, Lante A. A multifunctional bread rich in beta glucans and low in starch improves metabolic control in type 2 diabetes: a controlled trial. Nutrients 2017;9(3). https://doi.org/10.3390/nu9030297.
[21] Hou Q, Li Y, Li L, Cheng G, Sun X, Li S, Tian H. The metabolic effects of oats intake in patients with Type 2 diabetes: a systematic review and meta-analysis. Nutrients 2015;7(12):10369–87. https://doi.org/10.3390/nu7125536.
[22] Zhang Z, Li X, Jing L, Bao L, Li Y, Ma X, et al. Short- and long-term effects of wholegrain oat intake on weight management and glucolipid metabolism in overweight type-2 diabetics: a randomized control trial. Nutrients 2016;8(9):549. https://doi.org/10.3390/nu8090549.
[23] Fyfe C, Johnstone AM, Hickson K, McGeoch SC, Megson IL, Clark LF, et al. A randomized crossover study to assess the effect of an oat-rich diet on glycaemic control, plasma lipids and postprandial glycaemia, inflammation and oxidative stress in Type 2 diabetes. Diabet Med 2013;30(11):1314–23. https://doi.org/10.1111/dme.12228.
[24] Higgins JA. Whole grains, legumes, and the subsequent meal effect: implications for blood glucose control and the role of fermentation. J Nutr Metab 2012. https://doi.org/10.1155/2012/829238.
[25] Boers HM, Seijen Ten Hoorn J, Mela DJ. A systematic review of the influence of rice characteristics and processing methods on postprandial glycaemic and insulinaemic responses. Br J Nutr 2015. https://doi.org/10.1017/S0007114515001841.
[26] Ajala O, English P, Pinkney J. Systematic review and meta-analysis of different dietary approaches to the management of type 2 diabetes. Am J Clin Nutr 2013. https://doi.org/10.3945/ajcn.112.042457.Am.
[27] Tay J, Thompson CH, Brinkworth GD. Glycemic variability: assessing glycemia differently and the implications for dietary management of diabetes. Annu Rev Nutr 2015;35(1):389–424. https://doi.org/10.1146/annurev-nutr-121214-104422.
[28] Silva FM, Kramer CK, Crispim D, Azevedo MJ. A high–glycemic index, low-fiber breakfast affects the postprandial plasma glucose, insulin, and ghrelin responses of patients with type 2 diabetes in a randomized clinical trial. J Nutr 2015;145(4):736–41. https://doi.org/10.3945/jn.114.195339.
[29] Narayanan J, Sanjeevi V, Rohini U, Trueman P, Viswanathan V. Postprandial glycaemic response of foxtail millet dosa in comparison to a rice dosa in patients with type 2 diabetes. Indian J Med Res 2016. https://doi.org/10.4103/ijmr.IJMR_551_15.
[30] Nakayama T, Nagai Y, Uehara Y, Nakamura Y, Ishii S, Kato H, Tanaka Y. Eating glutinous brown rice twice a day for 8 weeks improves glycemic control in Japanese patients with diabetes mellitus. Nutr Diabetes 2017;7(5):e273–6. https://doi.org/10.1038/nutd.2017.26.
[31] SV T, DM W, AM H. Bean and rice meals reduce postprandial glycemic

response in adults with type 2 diabetes: a cross-over study. Nutr J 2012.
[32] Barakatun Nisak MY, Ruzita AT, Norimah AK, Nor Azmi K, Fatimah A. Acute effect of low and high glycemic index meals on post-prandial glycemia and insulin responses in patients with type 2 diabetes mellitus. Malays J Med Health Sci 2009;5(1):11–20.
[33] Yusof BNM, Talib RA, Kamaruddin NA, Karim NA, Chinna K, Gilbertson H. A low-GI diet is associated with a short-term improvement of glycaemic control in Asian patients with type 2 diabetes. Diabetes Obes Metab 2009. https://doi.org/10.1111/j.1463-1326.2008.00984.x.
[34] Thyfault JP, Liu Y, Nyhoff LM, Leidy HJ, Kanaley JA, Heden TD, Park Y-M. A high-protein breakfast induces greater insulin and glucose-dependent insulinotropic peptide responses to a subsequent lunch meal in individuals with type 2 diabetes. J Nutr 2014;145(3):452–8. https://doi.org/10.3945/jn.114.202549.
[35] Mignone LE. Whey protein: the "whey" forward for treatment of type 2 diabetes? World J Diabetes 2015;6(14):1274. https://doi.org/10.4239/wjd.v6.i14.1274.
[36] Patil P, Mandal S, Tomar SK, Anand S. Food protein-derived bioactive peptides in management of type 2 diabetes. Eur J Nutr 2015;54(6):863–80. https://doi.org/10.1007/s00394-015-0974-2.
[37] Jakubowicz D, Wainstein J, Landau Z, Ahren B, Barnea M, Bar-Dayan Y, Froy O. High-energy breakfast based on whey protein reduces body weight, postprandial glycemia and HbA1C in Type 2 diabetes. JNB (J Nutr Biochem) 2017. https://doi.org/10.1016/j.jnutbio.2017.07.005.
[38] Ma Jing, Jesudason DR, Stevens JE, Keogh JB, Jones KL, Clifton PM, et al. Sustained effects of a protein "preload" on glycaemia and gastric emptying over 4 weeks in patients with type 2 diabetes: a randomized clinical trial. Diabetes Res Clin Pract 2015;108(2):e31–4. https://doi.org/10.1016/j.diabres.2015.02.019.
[39] Tricò D, Filice E, Baldi S, Frascerra S, Mari A, Natali A. Sustained effects of a protein and lipid preload on glucose tolerance in type 2 diabetes patients. Diabetes Metab 2016;42(4):242–8. https://doi.org/10.1016/j.diabet.2016.03.004.
[40] Froy O. Metabolism and circadian rhythms - implications for obesity. Endocr Rev 2010. https://doi.org/10.1210/er.2009-0014.
[41] Bi H, Gan Y, Yang C, Chen Y, Tong X, Lu Z. Breakfast skipping and the risk of type 2 diabetes: a meta-analysis of observational studies. Publ Health Nutr 2015. https://doi.org/10.1017/S1368980015000257.
[42] Jakubowicz D, Wainstein J, Ahrén B, Bar-Dayan Y, Landau Z, Rabinovitz HR, Froy O. High-energy breakfast with low-energy dinner decreases overall daily hyperglycaemia in type 2 diabetic patients: a randomised clinical trial. Diabetologia 2015. https://doi.org/10.1007/s00125-015-3524-9.
[43] Jakubowicz D, Wainstein J, Ahren B, Landau Z, Bar-Dayan Y, Froy O. Fasting until noon triggers increased postprandial hyperglycemia and impaired insulin response after lunch and dinner in individuals with type 2 Diabetes: a randomized clinical trial. Diabetes Care 2015. https://doi.org/10.2337/dc15-0761.
[44] Shukla AP, Iliescu RG, Thomas CE, Aronne LJ. Food order has a significant impact on postprandial glucose and insulin levels. Diabetes Care 2015. https://doi.org/10.2337/dc15-0429.
[45] Shukla AP, Andono J, Touhamy SH, Casper A, Iliescu RG, Mauer E, et al. Carbohydrate-last meal pattern lowers postprandial glucose and insulin excursions in type 2 diabetes. BMJ Open Diabetes Res Care 2017;5(1):6–11. https://doi.org/10.1136/bmjdrc-2017-000440.
[46] Kurose T, Seino S, Seino Y, Minami K, Murotani K, Nosaka C, et al. Meal sequence and glucose excursion, gastric emptying and incretin secretion in type 2 diabetes: a randomised, controlled crossover, exploratory trial. Diabetologia 2015;59(3):453–61. https://doi.org/10.1007/s00125-015-3841-z.
[47] Imai S, Matsuda M, Fujimoto S, Miyatani S, Hasegawa G, Fukui M, et al. 糖尿病患者における食品の摂取順序による食後血糖上昇抑制効果Crossover study of the effect of "vegetables before carbohydrates" on reducing postprandial glucose and insulin in Japanese SubjectswithType 2 DiabetesMellitus. J Jpn Diabetes Soc 2010.
[48] Imai S, Matsuda M, Hasegawa G, Fukui M, Obayashi H, Ozasa N, Kajiyama S. A simple meal plan of "eating vegetables before carbohydrate" was more effective for achieving glycemic control than an exchange-based meal plan in Japanese patients with type 2 diabetes. Asia Pac J Clin Nutr 2011.
[49] Reynolds AN, Mann JI, Williams S, Venn BJ. Advice to walk after meals is more effective for lowering postprandial glycaemia in type 2 diabetes mellitus than advice that does not specify timing: a randomised crossover study. Diabetologia 2016. https://doi.org/10.1007/s00125-016-4085-2.
[50] Ma Jianhua, Li F, Li H, Hu Y, Su X, Zhang D, et al. Twenty minute moderate-intensity post-dinner exercise reduces the postprandial glucose response in Chinese patients with type 2 diabetes. Med Sci Monit 2018;24:7170–7. https://doi.org/10.12659/msm.910827.
[51] Francois ME, Myette-Cote E, Bammert TD, Durrer C, Neudorf H, DeSouza CA, Little JP. Carbohydrate-restriction with postmeal walking effectively mitigates postprandial hyperglycemia and improves endothelial function in type 2 diabetes. Am J Physiol Heart Circ Physiol 2017. ajpheart.00524.2017, https://doi.org/10.1152/ajpheart.00524.2017.
[52] Gulati S, Misra A, Nanda K, Pandey RM, Garg V, Ganguly S, Cheung L. Efficacy and tolerance of a diabetes specific formula in patients with type 2 diabetes mellitus: an open label, randomized, crossover study. Diabetes, Metab Syndrome 2015. https://doi.org/10.1016/j.dsx.2014.10.001.
[53] Mottalib A, Mohd-Yusof BN, Shehabeldin M, Pober DM, Mitri J, Hamdy O. Impact of diabetes-specific nutritional formulas versus oatmeal on postprandial glucose, insulin, GLP-1 and postprandial Lipidemia. Nutrients 2016;8(7):1–11. https://doi.org/10.3390/nu8070443.
[54] Lansink M, van Laere KMJ, Vendrig L, Rutten GEHM. Lower postprandial glucose responses at baseline and after 4 weeks use of a diabetes-specific formula in diabetes type 2 patients. Diabetes Res Clin Pract 2011. https://doi.org/10.1016/j.diabres.2011.05.019.
[55] Mayr P, Kuhn KS, Klein P, Stover JF, Pestana EA. A diabetes-specific oral nutritional supplement improves glycaemic control in type 2 diabetes patients. Exp Clin Endocrinol Diabetes 2016;124(7):401–9. https://doi.org/10.1055/s-0042-100909.
[56] Stenvers DJ, Schouten LJ, Jurgens J, Endert E, Kalsbeek A, Fliers E, Bisschop PH. Breakfast replacement with a low-glycaemic response liquid formula in patients with type 2 diabetes: a randomised clinical trial. Br J Nutr 2014;112(4):504–12. https://doi.org/10.1017/S0007114514001123.
[57] Castro-Acosta ML, Lenihan-Geels GN, Corpe CP, Hall WL. Berries and anthocyanins: promising functional food ingredients with postprandial glycaemia-lowering effects. Proc Nutr Soc 2016;75(03):342–55. https://doi.org/10.1017/s0029665116000240.
[58] Schell J, Betts NM, Foster M, Scofield RH, Basu A. Cranberries improve postprandial glucose excursions in type 2 diabetes. Food Funct 2017;8(9):3083–90. https://doi.org/10.1039/c7fo00900c.
[59] Pichardo-Ontiveros E, Pedraza-Chaverri J, Tovar AR, Vázquez-Manjarrez N, Avila-Nava A, Torres N, López-Romero P. The effect of nopal (Opuntia Ficus indica) on postprandial blood glucose, incretins, and antioxidant activity in Mexican patients with type 2 diabetes after consumption of two different composition breakfasts. J Acad Nutr Diet 2014;114(11):1811–8. https://doi.org/10.1016/j.jand.2014.06.352.
[60] Cheng S, Ismail A, Anthony J, Ng OC, Hamid AA, Barakatun-nisak MY. Eight weeks of Cosmos caudatus (Ulam Raja) supplementation improves glycemic status in patients with type 2 Diabetes . A Randomized Controlled Trial; 2015. 2015.
[61] Terada T, Wilson BJ, Myette-Côté E, Kuzik N, Bell GJ, McCargar LJ, Boulé NG. Targeting specific interstitial glycemic parameters with high-intensity interval exercise and fasted-state exercise in type 2 diabetes. Metab Clin Exp 2016;65(5):599–608. https://doi.org/10.1016/j.metabol.2016.01.003.
[62] Pratley RE, Weyer C. The role of impaired early insulin secretion in the pathogenesis of Type II diabetes mellitus. Diabetologia 2001. https://doi.org/10.1007/s001250100580.
[63] Holst JJ, Gromada J. Role of incretin hormones in the regulation of insulin secretion in diabetic and nondiabetic humans. Am J Physiol Endocrinol Metab 2004. https://doi.org/10.1152/ajpendo.00545.2003.
[64] Yu K, Ke MY, Li WH, Zhang SQ, Fang XC. The impact of soluble dietary fibre on gastric emptying, postprandial blood glucose and insulin in patients with type 2 diabetes. Asia Pac J Clin Nutr 2014;23(2):210–8. https://doi.org/10.6133/apjcn.2014.23.2.01.
[65] Marathe CS, Rayner CK, Jones KL, Horowitz M. Relationships between gastric emptying, postprandial glycemia, and incretin hormones. Diabetes Care 2013. https://doi.org/10.2337/dc12-1609.
[66] Chang J, Rayner CK, Jones KL, Horowitz M. Diabetic gastroparesis and its impact on glycemia. Endocrinol Metab Clin N Am 2010. https://doi.org/10.1016/j.ecl.2010.08.007.
[67] Tan MY, Magarey J. Self-care practices of Malaysian adults with diabetes and sub-optimal glycaemic control. Patient Educ Counsel 2008;72(2):252–67. https://doi.org/10.1016/j.pec.2008.03.017.
[68] Azzout-Marniche D, Gaudichon C, Tomé D. Dietary protein and blood glucose control. Curr Opin Clin Nutr Metab Care 2014;17(4):349–54. https://doi.org/10.1097/MCO.0000000000000062.
[69] Lee S-H, Tura A, Mari A, Ko S-H, Kwon H-S, Song K-H, et al. Potentiation of the early-phase insulin response by a prior meal contributes to the second-meal phenomenon in type 2 diabetes. Aust J Pharm: Endocrinol Metabol 2011. https://doi.org/10.1152/ajpendo.00244.2011.