International Journal of Scientific Reports
Shah AD et al. Int J Sci Rep. 2025 Nov;11(11):421-429
http://www.sci-rep.com
pISSN 2454-2156 | eISSN 2454-2164
DOI: https://dx.doi.org/10.18203/issn.2454-2156.IntJSciRep20253330

Review Article

Optimizing nutritional strategies in type 2 diabetes patients receiving sodium-glucose cotransporter-2 inhibitor therapy

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Received: 12 August 2025
Accepted: 08 October 2025

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ABSTRACT

Sodium-glucose cotransporter-2 (SGLT2) inhibitors improve glycaemic control via insulin-independent glycosuria and offer cardiovascular and renal benefits. However, their safety and effectiveness are significantly influenced by dietary context, particularly in high-carbohydrate environments like India, where refined grain consumption and periodic fasting are common. This review discusses how SGLT2 inhibitor-induced energy loss, ketone production, and fluid shifts interact with regional dietary patterns. Key nutritional concerns arise in this context, including sarcopenia from caloric and protein deficits, euglycemic diabetic ketoacidosis (euDKA) triggered by carbohydrate restriction, and volume depletion due to inadequate hydration. To manage these risks, targeted nutritional strategies are essential. These include moderating carbohydrate intake (around 45-50% of total energy), maintaining sufficient protein intake (15-20% of energy), maintaining hydration (2.0-2.5 L/day), and avoiding ketogenic or overly carbohydrate restrictive diets. Furthermore, individualized dietary planning during religious fasts is important for maintaining metabolic balance. In South Asian populations, where dietary habits are deeply rooted in culture, nutrition counseling is not merely supportive but holds a central role in optimizing therapeutic outcomes with SGLT2 inhibitors.

Keywords: Sodium-glucose cotransporter-2 inhibitors, Carbohydrate metabolism, Glycosuria, Sarcopenia, Medical nutritional therapy

INTRODUCTION

Type 2 diabetes mellitus (T2DM) represents a rapidly increasing global health challenge. As of 2024, approximately 589 million adults are estimated to be living with the condition, and projections indicate this figure may increase to 853 million by the year 2050.[1] India accounts for over 101 million individuals living with diabetes, positioning it as a major epicenter of the epidemic. This burden is intensified by the country's ongoing nutritional transition, urbanization, and increasingly sedentary lifestyles.[2]

Dietary patterns in India remain predominantly high in carbohydrates, contributing approximately 65-70% of total daily energy intake.[3,4] These carbohydrates are largely derived from high-glycemic sources such as refined wheat flour and polished white rice, foods that promote rapid postprandial hyperglycemia, insulin resistance, and atherogenic dyslipidemia, all of which compound the challenges of glycemic management in T2DM.[5]

Against this background, SGLT2 inhibitors have emerged and established as a vital therapeutic option for managing T2DM. By inhibiting renal glucose reabsorption, these agents enhance urinary glucose excretion (UGE), thereby lowering plasma glucose levels without relying on insulin action. Beyond glycemic control, SGLT2 inhibitors also contribute to modest weight reduction, lower blood pressure, and offer significant cardiovascular and renal benefits in individuals with T2DM.[6] However, the

pharmacodynamic profile of SGLT2 inhibitors, marked by glycosuria, osmotic diuresis (natriuresis), and increased glucagon secretion, introduces certain metabolic risks that are closely influenced by nutritional factors. Sustained glycosuria results in continuous loss of calories and electrolytes through urine. If not compensated by adequate dietary intake, this can increase the risk of energy deficits, dehydration, and electrolyte disturbances.[7,8]

Despite these important considerations, existing clinical guidelines provide limited direction on nutritional recommendations specifically adapted to SGLT2 inhibitor therapy. In particular, there is a lack of structured guidance regarding carbohydrate quality, protein intake, hydration, and culturally specific meal patterns. This gap is concerning in populations such as South Asians, where high-carbohydrate diets and routine fasting practices can exacerbate therapy-related metabolic risks.[9,10]

This review explores how SGLT2 inhibitors interact with the unique dietary practices of South Asian populations, especially in India, and outlines practical strategies to optimize therapy, prevent adverse effects, and align therapy with everyday eating habits. The discussion includes physiological adaptations to SGLT2 inhibitors, analysis of regional dietary patterns that affect treatment response, and emphasis on three emerging clinical concerns: (1) risk of sarcopenia due to deficits in caloric and protein intake; (2) the occurrence of euDKA triggered by carbohydrate restriction or fasting; and (3) the safe use of SGLT2 inhibitors during cultural and religious fasts in Indian settings. A comprehensive framework of nutritional strategies tailored to South Asian dietary patterns is presented to support the safe and effective use of SGLT2 inhibitors in clinical practice.

MECHANISM OF ACTION OF SGLT2 INHIBITORS: NUTRITIONAL AND HEMO-DYNAMIC PERSPECTIVES

Glycosuria-induced energy deficit and risk of catabolism

SGLT2 inhibitors exert their antihyperglycemic effects by selectively inhibiting glucose reabsorption in the renal proximal tubules. This insulin-independent mechanism leads to enhanced UGE, resulting in an estimated daily loss of 60-80 grams of glucose, equivalent to an energy deficit of approximately 240-320 kilocalories.[11] Although this supports glycemic control and modest weight loss, it also initiates a complex set of carbohydrate-dependent metabolic adaptations with important nutritional implications.

In specific patient groups, particularly those who are lean, elderly, or already sarcopenic, this pharmacologically induced energy loss may exceed physiological compensatory capacity, especially when dietary intake fails to meet increased energy and protein demands. In such cases, the body initiates catabolic responses to meet energy demands by mobilizing endogenous substrates such as adipose tissue and skeletal muscle.

If dietary protein intake is inadequate, a negative nitrogen balance may ensue, accelerating the loss of lean body mass. This can lead to symptoms including fatigue, weakness, and a gradual decline in functional status. Over time, persistent energy-protein imbalance may result in clinically significant malnutrition, which increases morbidity and undermines therapeutic outcomes.[12,13]

Figure 1 summarizes these nutritional and hemodynamic consequences. It illustrates how SGLT2 inhibitor-induced glycosuria and natriuresis contribute to energy depletion, muscle catabolism, dehydration, and electrolyte imbalances. Addressing these risks requires timely nutritional assessment and intervention, with particular attention to ensuring adequate caloric intake and protein of high biological value, especially in individuals at increased risk of undernutrition.[14]

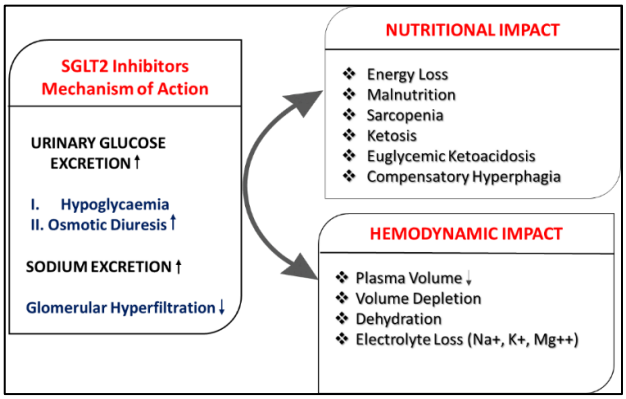

Figure 1: Nutritional and hemodynamic consideration in patients receiving SGLT2 inhibitor therapy.

Hormonal adaptations: glucagon-mediated gluconeogenesis and protein breakdown

Beyond glycosuria-induced energy loss, SGLT2 inhibitors also influence metabolic pathways through distinct hormonal mechanisms. These agents consistently reduce circulating insulin levels while elevating glucagon secretion, leading to a shift in the insulin to glucagon ratio. This altered hormonal environment favors hepatic gluconeogenesis, glycogenolysis, and lipolysis-processes that help maintain euglycemia, particularly during fasting or carbohydrate-restricted states.[15]

These adaptations, while physiologically compensatory, may contribute to unintended protein loss, especially in vulnerable individuals. Elevated glucagon levels stimulate the mobilization of amino acids for gluconeogenesis, increasing protein turnover and placing additional demands on lean tissue stores. Notably, this catabolic effect can occur independently of a caloric

deficit and may present even in nutritionally replete individuals. Such observations have been reported in clinical settings, highlighting the complexity of metabolic responses under SGLT2 inhibition.

Therefore, effective nutritional management must address not only energy replacement but also hormonal modulation. Maintaining a balanced glucagon to insulin ratio through moderate carbohydrate intake can help preserve basal insulin secretion, thereby reducing excessive reliance on amino acid catabolism. Simultaneously, ensuring sufficient intake of high biological value protein is critical for minimizing muscle breakdown and preserving lean body mass.

By targeting both energy metabolism and hormonal regulation, these strategies contribute to improved metabolic stability, reduced sarcopenia risk, and optimized therapeutic outcomes in patients receiving SGLT2 inhibitor therapy.[16]

Hepatic ketogenesis and risk of euDKA

Alongside its effects on gluconeogenesis and lipolysis, SGLT2 inhibition enhances hepatic β-oxidation and ketone body production, indicating a metabolic shift toward increased lipid utilization. While ketone bodies serve as efficient energy substrates for the brain and myocardium, excessive accumulation, particularly under conditions of carbohydrate restriction, can precipitate euDKA. Case reports have linked the use of SGLT2 inhibitors to euDKA in patients following very low-carbohydrate or ketogenic diets.[17,18]

Maintaining a moderate carbohydrate intake plays a critical role in mitigating this risk by supporting basal insulin secretion and limiting excessive lipolysis. As such, dietary strategies that avoid extreme carbohydrate restriction are essential to reducing the likelihood of euDKA in patients undergoing SGLT2 inhibitor therapy.[19,20]

Renal and hemodynamic adaptations: diuresis, volume depletion, and electrolyte imbalance

SGLT2 inhibitors promote both glycosuria and natriuresis, causing osmotic diuresis and a decrease in intravascular volume. These effects contribute to small yet clinically helpful reductions in systolic and diastolic blood pressure, particularly in patients with hypertension or fluid overload.[21] However, these same hemodynamic adaptations may also result in unintended adverse effects.

Hypovolemia, defined by decreased circulating plasma volume, can impair tissue perfusion and increase the risk of fatigue, dizziness, or circulatory instability. Orthostatic hypotension, a sudden drop in blood pressure upon standing, is more common in elderly or volume-depleted patients and may lead to dizziness or syncope. Furthermore, acute kidney injury (AKI) may occur due to reduced renal perfusion, especially in patients using diuretics, experiencing dehydration, or undergoing prolonged fasting. Electrolyte imbalances, including mild hypokalemia, hypomagnesemia, and hyponatremia, may occurs due to enhanced renal excretion. These disturbances can impair neuromuscular function and compromise cardiovascular stability.[22]

The nutritional implications of these fluid shifts are significant. In hot and extremely hot climates or during periods of religious fasting, both common in South Asian contexts, patients may be at higher risk of volume and mineral depletion. As such, individualized hydration strategies, adequate intake of electrolytes, and cautious sodium restriction (only when clinically warranted) are critical to ensuring treatment safety and continuity during SGLT2 inhibitor therapy.[23]

Appetite regulation and compensatory hyperphagia

Although SGLT2 inhibitors cause a net caloric loss through glycosuria, emerging research suggests that some patients may exhibit compensatory hyperphagia, an increased appetite, especially craving carbohydrate-rich or sweet foods. This response appears to be partially mediated by fibroblast growth factor 21 (FGF21), a hormone involved in energy regulation and adaptive feeding behavior.[24] Elevated levels of FGF21 in response to energy loss may help explain this shift in food preference.

This compensatory drive may diminish the expected weight loss benefits of SGLT2 inhibitors and contribute to glycemic variability. Importantly, this behavior is not solely hormonal; central nervous system adaptations and behavioral compensation in response to perceived energy loss may also play a role.

These findings underscore the importance of integrating nutritional counseling into clinical care. Guiding patients to maintain an energy balance without overcompensating for glycosuric losses can help sustain the metabolic benefits of therapy. Moreover, aligning pharmacologic interventions with behavioral support is essential to prevent overeating and preserve therapeutic outcomes.[25,26]

REGIONAL DIETARY PATTERNS AND CLINICAL NUTRITION IMPLICATIONS

The clinical expression of SGLT2 inhibitor therapy in India is strongly shaped by the country's carbohydrate-dominant diets, meal composition, and fasting practices. While the pharmacologic actions remain constant, their metabolic consequences may be intensified or attenuated depending on regional food habits and nutritional adequacy.

In most Indian populations, carbohydrates account for approximately 65-70% of daily caloric intake, primarily

from refined grains (e.g., polished rice, Maida), starchy vegetables, and sugar-laden dishes.[4] These dietary structures, often lacking adequate fiber and protein, are metabolically imbalanced and may exaggerate glycemic fluctuations, increase insulin demand, and amplify the osmotic effects of glycosuria under SGLT2i therapy.

When combined with inadequate hydration or protein consumption, such dietary exposures can exacerbate therapy-induced alterations in fluid equilibrium, electrolyte levels, and ketone metabolism. Importantly, these risks are modulated by region-specific dietary patterns, religious rituals, and socioeconomic access to diverse food groups. Regionally informed nutritional strategies are thus essential to optimize therapy outcomes.[27]

High glycaemic foods and macronutrient disparities in Indian diets

Indian meal patterns frequently include high glycaemic index staples, which contribute to significant postprandial glucose spikes. These foods are frequently taken without sufficient dietary fibre, protein-rich foods, or healthy fat sources (e.g., nuts, seeds, or plant oils), all of which aid with moderate glycaemic control. Such rapid glucose elevations increase insulin demand and intensify SGLT2 inhibitor induced osmotic diuresis, thereby raising the risk of dehydration and electrolyte imbalance.

This is compounded by widespread protein insufficiency, particularly among vegetarians, lower socioeconomic groups and those adhering to religious dietary restrictions. Many individuals fall short of the recommended 0.8-1.0 g/kg/day intake, limiting the body's capacity to preserve lean mass, sustain gluconeogenesis, and adapt to glycosuria-related energy deficiency. In elderly or metabolically fragile patients, this contributes to sarcopenia and blunted metabolic compensation.[10]

Together, the combination of high glycemic carbohydrate intake and suboptimal protein consumption creates a nutritionally vulnerable state that can compromise both the safety and therapeutic efficacy of SGLT2 inhibitors in the absence of targeted dietary support.

Regional profiles: dietary staples and metabolic risk

Although India's regional diets vary in ingredients and preparation styles, they often converge in promoting nutritional patterns linked to elevated metabolic risk. The scale and expression of this risk vary with local food traditions, portion sizes, and the frequency of high-carbohydrate meals, particularly during the festive occasions.

In each region of India, staple foods offer inherent nutritional value but are frequently consumed in forms or quantities that compromise metabolic control. In North India, diets include whole wheat flatbreads and legumes, which provide fiber and micronutrients. However, these are commonly paired with refined flour (Maida) based fried items like puri and bhature, and ghee-laden sweets such as halwa, jalebi, and kheer, contributing to postprandial hyperglycemia and excess caloric intake. In Western India, diverse forms of bhakri made from jowar, bajra, or rice are staples. Although millets like jowar and bajra have a low glycemic index, their metabolic advantage is often offset by large portion sizes, concurrent white rice consumption, and frequent intake of calorie-dense sweets like shrikhand, ladoo, and sheera, especially during festivals. In Eastern and Northeastern India, diets center on white rice, puffed grains, tubers, and fish or vegetable curries. While these offer essential nutrients, their benefits may be diluted by frequent consumption of syrup-soaked sweets like payesh, rasgulla, and sandesh.

In Southern India, rice is dominant staple and is consumed in substantial quantities, often multiple times daily, in forms like idli, dosa, and pongal. These are frequently paired with milk-based desserts such as payasam, contributing to consistently high glycemic load.[29,30]

Across the diverse regions of India, refined rice, potatoes, and cooking oils are common dietary staples. These foods tend to be high in calories and are rapidly absorbed by the body. National dietary surveys show that rice remains the most widely consumed cereal, while potatoes frequently appear among the top three vegetables consumed across the country. Additionally, oil intake has risen substantially across both rural and urban settings. These eating patterns, though rooted in tradition and convenience, often increase the risk of high blood sugar and cholesterol.

When protein diversity is limited and hydration awareness is low, these regional dietary patterns may increase susceptibility to therapy-related metabolic complications and accelerate insulin resistance, particularly during medical therapy/disease management.[30]

Table 1: Regional Indian diets and high glycemic load contributors.

India-regions	Staples	High glycemic load contributors	Metabolic risk
North	Whole wheat (chapati), legumes, seasonal vegetables, dairy.	Refined flour breads (puri, bhature), sweetened desserts (halwa, kheer, jalebi), potato-rich curries.	Postprandial hyperglycaemia

Continued.

India-regions	Staples	High glycemic load contributors	Metabolic risk
West	Jowar, bajra, wheat (bhakri), pulses, greens	Overconsumption of millet/ rice breads (bhakri)/ white rice in one meal, ghee-heavy sweets. (churma, sheera, laddoo) Continued.	Central obesity Insulin resistance
East/ Northeast	White rice, puffed rice (muri), tubers, fish/ vegetable curries.	Deep-fried breads (luchi), sweet potatoes, sugar syrups in sweets. (payesh, rasgulla, sandesh)	Dyslipidaemia Hypertension
South	Parboiled/white rice, dosa, idli, sambhar, coconut-based curries.	Large portions of white rice, semolina (rava) / fermented rice batter dishes, fried snacks, sweet pongal, payasam. (milk desserts)	NAFLD/ MASH

***Sources**: National institute of nutrition (2020); ICMR-NIN (2023). Abbreviations: NAFLD-Non-alcoholic fatty liver disease; MASH-Metabolic dysfunction-associated steatohepatitis.

Festivals, fasts, and the dual challenge of dietary extremes

Fasting and feasting cycles associated with cultural and religious observances-such as Ramadan, Navratri, and Jain fast (including prolonged or extreme fasting), pose unique metabolic challenges for individuals with diabetes. These practices often involve prolonged food and fluid restriction, followed by refeeding with energy-dense, high-glycemic meals that can disrupt metabolic stability.

During Ramadan, daily fasting from dawn to dusk is followed by iftar and suhoor meals rich in sweets (e.g., dates, sherbets), fried foods (samosas, pakoras), and calorie-dense main courses like biryani. This pattern can result in daytime dehydration and ketosis, followed by postprandial glycemic surges. In Navratri, fasting often excludes grains and pulses, relying instead on starchy tubers (e.g., potato, yam), tapioca, nuts, and sweetened dishes like halwa and laddoo. Jain fasting may involve extended periods of complete food and water abstinence, sometimes with minimal or delayed refeeding, raising risks of severe dehydration, electrolyte imbalance, and catabolic stress.[31]

These shifts between caloric deficits and postprandial glycaemic surges lead to significant glycaemic variability, hormonal imbalances, and osmotic stress, especially in patients with T2DM. For those on SGLT2 inhibitors, the compounded effects of glycosuria-induced volume loss, enhanced ketone production, and electrolyte depletion can increase the risk of orthostatic hypotension, dizziness, or syncope, especially in elderly or frail patients. These risks underscore the importance of personalized dietary planning, hydration strategies, and close medical supervision during religious fasting in patients receiving SGLT2 inhibitors.

NUTRITION STRATEGIES FOR SAFE AND EFFECTIVE SGLT2 INHIBITOR THERAPY

Building on the cultural and regional diversity of Indian foods addressed in earlier section regional dietary patterns and clinical nutrition implications, this section applies those insights to practical, evidence-based nutrition strategies for patients receiving SGLT2 inhibitor medication. While the pharmacological effects of SGLT2 inhibitors are consistent across populations, nutritional context-including the quantity, quality, and timing of macronutrient intake, plays a pivotal role in modulating therapeutic outcomes and adverse event risks.

Indian diets often deliver a high quantity of carbohydrates, frequently exceeding 250-350 grams/day, with low fiber density and limited protein diversity. National nutrition surveys indicate that total carbohydrate consumption is high across both vegetarian and non-vegetarian groups in India, primarily from cereals and starchy vegetables.[30]

This creates a metabolic imbalance with the glycosuria-induced energy loss, catabolic stress, and osmotic diuresis triggered by SGLT2 inhibitors. Therefore, tailoring nutritional guidance to match regional staples, meal size, fasting customs, and protein adequacy is essential to preserve safety and optimize efficacy.

Carbohydrate management

Moderating both the **quantity** and **quality** of carbohydrate intake is critical for patients undergoing SGLT2 inhibitor therapy. Aim for 45-50% of total daily calories from carbohydrates, as opposed to the ~ 65-70% typically seen in Indian diets. For an average adult (2000 kcal/day), this corresponds to roughly 225-250 g of carbs daily, though many patients may benefit from further reduction to ~130-180 g/ day depending on their baseline intake and tolerance.[32]

Optimal sources include whole grains (brown rice, whole wheat with bran), millets [such as ragi (finger millet), jowar (sorghum), bajra (pearl millet)], legumes (dal, chickpeas, beans), and non-starchy vegetables. These foods attenuate postprandial glucose spikes and provide more sustained energy release, which can reduce the osmotic diuresis and glucagon swings provoked by high-GI foods. For example, substituting white rice with a mix of millets or adding barley to rice can lower meal glycemic index.

Additionally, spreading carbohydrate intake evenly through the day (e.g. three smaller meals and snacks if needed) is preferable to consuming very large carbohydrate loads in one sitting. Large bolus loads can overwhelm the SGLT2-mediated glycosuria capacity and cause marked hyperglycemia and diuresis before excess glucose is excreted. Educating patients on approximate carbohydrate content of common foods through methods like carbohydrate counting or using plate models can empower them to moderate portion sizes. For instance, one bowl of cooked rice (~30 g carbs) might be a reasonable portion, but traditional servings can be 2-3 times that; counseling to cut back can significantly reduce daily carb excess.[33]

Hydration and fluid maintenance

SGLT2 inhibitors promote osmotic diuresis and natriuresis, leading to mild intravascular volume contraction. While this effect may be therapeutically beneficial in patients with hypertension or fluid overload, it can also precipitate adverse outcomes such as hypotension, fatigue, orthostatic dizziness, or even AKI particularly among older adults, those exposed to extremely hot or humid climate that cause excessive sweating, or during fasting states.

Patients should be encouraged to maintain a daily fluid intake of 2.0-2.5 liters, unless otherwise restricted for medical reasons. In individuals with comorbid conditions such as heart failure or chronic kidney disease (CKD), fluid recommendations must be carefully individualized based on volume status, ejection fraction, and renal function markers. In these populations, close clinical monitoring is essential to avoid both dehydration and fluid overload.

To maintain electrolyte balance, especially in high-temperature environments or during extended fasting periods, electrolyte-rich fluids such as diluted buttermilk, coconut water, clear soups, or oral rehydration solutions (ORS) can be incorporated into the daily diet. Patients should also be counseled to recognize early signs of dehydration, including orthostatic hypotension, dark-colored urine, very low urine volume, dry mouth, and fatigue, and to adjust their fluid intake accordingly.[34]

Avoidance of ketogenic and very low-carbohydrate diets

Ketogenic diets, which typically provide less than 10% of total daily energy from carbohydrates (under 50 grams per day), and very low-carbohydrate diets, providing less than 26% of total energy (under 130 grams per day), are medically contraindicated in individuals taking SGLT2 inhibitors due to an increased risk of euDKA.[35] SGLT2 inhibitors promote ketogenesis by increasing glucagon secretion and suppressing insulin levels. When carbohydrate intake is severely restricted, ketone production by the liver may become dangerously high, even if blood glucose remains normal or only mildly elevated.

Studies have shown that insulin suppression and elevated glucagon under SGLT2 inhibition amplify hepatic ketone generation. There have been documented cases of euDKA developing in patients on SGLT2 inhibitors who follow ketogenic diets, where normal glucose levels masked severe acidosis, complicating timely diagnosis. Excessive alcohol intake should also be avoided, as it can impair gluconeogenesis, worsen ketosis, and contribute to dehydration. These factors further increase the risk of euDKA in patients using SGLT2 inhibitors.[18]

Clinicians should routinely screen for such dietary behaviours and educate patients to avoid aggressive carbohydrate elimination and excessive alcohol consumption, particularly during periods of caloric restriction and metabolic fasting associated with certain diets and illnesses.[35]

Protein intake and lean mass preservation

To offset the catabolic risk posed by glycosuria-induced energy loss (~240-320 kcal/day), a daily protein intake of 1.0-1.2 g/kg body weight (15-20% of total energy) is advisable, especially in elderly or sarcopenic individuals.

High biological value protein sources such as dairy (milk, curd, paneer), soy, pulses, and eggs should be emphasized in meal planning. During religious or cultural fasts where animal protein may be excluded, milk proteins and legumes become critical to prevent protein-energy malnutrition.[34]

Fat intake for cardiometabolic synergy

Given the cardioprotective profile of SGLT2 inhibitors, optimizing dietary fat quality can further improve metabolic outcomes. Monounsaturated fatty acids (MUFA), found in groundnut, mustard, sesame, and olive oils, as well as in nuts like peanuts and almonds, should be prioritized for their lipid-lowering and insulin-sensitizing effects. Omega-3 polyunsaturated fatty acids (PUFA), available in flaxseeds, chia seeds, walnuts, and oily fish such as mackerel (bangda), sardines, and Indian salmon (rawas), are also beneficial in lowering triglycerides level and improving cardiometabolic health.[37]

In contrast, saturated fats found in in ghee, full-fat dairy, and red meat should be limited. Trans fats, often present in Vanaspati, deep-fried snacks (e.g., samosas, pakoras), and traditional sweets, should be avoided for their proatherogenic properties.

Safe adaptations during religious or cultural fasting

Religious fasts (e.g., Ramadan, Navratri, Jain fasting) may increase the risk of hypovolemia, ketogenesis, and

euDKA in patients receiving SGLT2 inhibitor therapy. Patient counseling should include education on early warning signs such as dizziness, nausea, fruity breath (indicative of ketosis), dry mouth or dark urine (suggestive of dehydration), and confusion or sweating (which may indicate hypoglycemia). Patients should be advised to break the fast immediately if any of these symptoms occur.

Pre- and post-fast meal planning should focus on incorporating complex carbohydrates, adequate protein, and fluid-rich foods to maintain metabolic and hemodynamic stability. It is also important to assess renal function and metabolic status before initiating fasting, especially in high-risk individuals or those extending the fasting duration.

These strategies can help reduce fasting-related complications and support safe continuation of SGLT2 inhibitor therapy in a culturally sensitive manner.[29,38]

Culturally adapted regional meal strategies

Personalizing dietary advice to regional food habits and preferences improves adherence and safety. The table below outlines culturally sensitive refinements linked with SGLT2 inhibitors therapy:

These strategies must remain sensitive to seasonal availability, food access, and cultural acceptance. Where possible, visual meal planning tools, cooking demonstrations, or community-based education can support patient engagement and sustainability.[39]

Table 2: Zone-specific culturally adapted dietary recommendation for optimizing SGLT2 inhibitor therapy.

India-regions	Include	Limit
North	Whole wheat chapatis; dal or paneer at meals; fruit or plain yogurt for dessert.	Deep-fried breads (naan, puri); ghee-rich desserts (halwa, jalebi).
West	Mixed-grain bhakri with dal and vegetables; roasted gram sweets with sugar substitutes.	Large portions of bhakri/rice; ghee-heavy sweets (ladoo, sheera, churma).
East/ Northeast	Khichdi (rice and lentils); fish or vegetable curry; soy/legume mixes; fruit or low-sugar mishti doi.	Fried snacks (telebhaja); syrupy sweets (payesh, rasgulla, sandesh).
South	Millets or brown rice for part of meal; lentil-rich dosa/idli; curd for protein and hydration support.	Large portions of white rice; sweet dishes (payasam, sweet pongal).

Table 3: Nutritional considerations in SGLT2 inhibitor therapy.

Nutritional parameters	Recommendation	Preventive measure
Carbohydrate intake	~45-50% of total energy from low-GI, high-fiber sources; distribute intake evenly across meals.	Avoid large portions of refined carbs; monitor postprandial glucose.
Very low carbohydrate or ketogenic diets	Avoid strict very low-carbohydrate (<26% energy) and ketogenic (<10% energy) diets.	Prevent euDKA by maintaining moderate carb intake.
Hydration	Ensure 2-2.5 L/day (unless contraindicated); increase during fasting or extremely hot or humid climate.	Monitor hydration status; include fluids with electrolytes if needed.
Protein intake	1.0-1.2 g/kg/day (15-20% energy); emphasize high biological value protein sources.	Prevent sarcopenia, especially in elderly; avoid excess in CKD.
Dietary fats	Prefer MUFA and omega-3-rich fats; limit saturated fat.	Avoid trans fats and fried food to support cardiovascular health.
Fasting practices	Individualize plan; ensure hydration; break the fast with balanced meals.	Monitor for hypoglycemia, dehydration, and ketone elevation; educate patients accordingly.

By adhering to these practices, patients and providers can work together to fully realize the benefits of SGLT2 inhibitors including improved glycaemic control, weight management, and organ protection, while minimizing preventable adverse effects. [5,15]

CONCLUSION

SGLT2 inhibitors have contributed meaningfully to the management of type 2 diabetes, with added benefits for cardiovascular and renal health. Yet, their overall effectiveness depends not just on the pharmacology but on how well therapy aligns with the patient's everyday nutritional habits and metabolic condition.

In regions such as South Asia, where high-refined-carbohydrate diets and fasting traditions are common, nutritional factors can significantly influence treatment response. Issues like low protein intake, suboptimal hydration, and extremes in carbohydrate consumption may increase the risk of muscle loss, dehydration, or even complications such as euglycemic ketoacidosis.

Conversely, very low-carbohydrate or ketogenic diets, sometimes adopted with the intention of improving control, may counteract the intended metabolic effects of SGLT2 inhibitors.

To improve both safety and outcomes, dietary considerations must be addressed alongside pharmacologic decisions. Practical strategies such as maintaining moderate carbohydrate intake, ensuring adequate protein, and preparing for fasting periods can reduce risks and help patients maintain treatment continuity.

When nutrition is integrated into care planning from the start, especially in culturally distinct dietary environments, SGLT2 inhibitors are more likely to achieve their full therapeutic benefit. This approach supports a more personalized and realistic model of diabetes management, one that balances efficacy with sustainability in everyday life.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Shah AD, Hebbale VA, Sarda PS, Khobragade KJ. Optimizing nutritional strategies in type 2 diabetes patients receiving Sodium-glucose cotransporter-2 inhibitor therapy. Int J Sci Rep 2025;11(11):421-9.