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Targeting Postprandial Hyperglycaemia: Current Insights on Acarbose and Its Therapeutic Role

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Abstract

This in-depth review explores the contribution of acarbose to blood glucose regulation, with a focus on its application in type 1 diabetes, type 2 diabetes mellitus (T2DM), and gestational diabetes mellitus (GDM). As an alpha-glucosidase inhibitor, acarbose functions by slowing the breakdown of carbohydrates in the small intestine, thus avoiding abrupt rises in blood glucose after meals. This mode of action plays an essential role in achieving steady glucose levels and lowering HbA1c, which helps prevent chronic complications of diabetes. The review presents evidence from recent clinical trials confirming the drug's effectiveness and favourable safety characteristics, including low systemic uptake and good tolerance in various patient groups. It also considers the benefits of using acarbose alongside other antidiabetic medications, highlighting its additive role in improving overall glucose management. Moreover, the paper discusses current developments, active investigations, and prospective applications of acarbose in diabetes care, reinforcing its importance as an effective option for strategies designed to optimise patient results.

Keywords: Acarbose, Glycaemic control, Type 2 diabetes mellitus, Postprandial hyperglycaemia, Alpha-glucosidase inhibitor, HbA1c

Introduction

Diabetes represents a major global health issue in the 21st century, with the International Diabetes Federation estimating an increase in affected individuals from 366 million in 2011 to 552 million by 2030, driven mainly by T2DM, which constitutes 90% of all cases [1]. Timely detection is critical to avert complications, and optimal blood glucose management is fundamental. Studies show that tight glucose control markedly decreases the risk of complications, with advantages persisting even after intensive therapy ends [2]. Post-meal glucose concentrations contribute significantly to overall hyperglycaemia and raised HbA1c, both linked to higher mortality rates. Diabetes encompasses multiple forms: type 1, resulting from autoimmune destruction leading to absolute insulin lack; type 2, characterised by insulin resistance and frequently associated with modifiable lifestyle factors; and gestational diabetes mellitus, developing during pregnancy due to hormonal effects on insulin sensitivity. Acarbose serves as an important agent for glucose regulation across these categories. By inhibiting alpha-glucosidase, it delays carbohydrate digestion in the small intestine, postponing glucose uptake and blunting postprandial glucose excursions—key to sustained glycaemic stability. Acarbose is particularly useful in T2DM, providing a distinct mechanism compared with other glucose-lowering drugs. Administered at the start of meals, it binds to alpha-glucosidase enzymes, decelerating carbohydrate hydrolysis and limiting sharp post-meal glucose elevations. It preferentially targets the digestion of starches and disaccharides, enabling precise glucose

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control without broadly impairing nutrient absorption [3]. In summary, acarbose continues to be a useful therapy for addressing postprandial hyperglycaemia in T2DM, supporting better clinical outcomes. The present review evaluates the efficacy and safety of acarbose in treating post-meal hyperglycaemia in patients with T2DM, while examining its effects on overall glycaemic control, HbA1c values, and patient prognosis. It further investigates the possible advantages of acarbose in combined regimens to improve glucose handling and reduce the likelihood of diabetes-associated complications.

Acarbose: structure, mechanism of action and glycaemic management

Acarbose, an oral antidiabetic agent that does not stimulate insulin release, exerts a unique effect on blood glucose by interfering with carbohydrate digestion in the small intestine **(Figure 1)**. This complex oligosaccharide acts as a competitive and reversible inhibitor of pancreatic alpha-amylase and brush-border intestinal alpha-glucosidase enzymes [4]. Pancreatic alpha-amylase breaks down complex carbohydrates into oligosaccharides within the intestinal lumen, while intestinal alpha-glucosidases then hydrolyse these oligosaccharides, trisaccharides, and disaccharides (such as sucrose and maltose) into absorbable monosaccharides including glucose and fructose. Through inhibition of these enzymes, acarbose retards carbohydrate digestion and absorption, thereby attenuating the postprandial increase in blood glucose.

In contrast to many other antidiabetic agents, acarbose limits the absorption of dietary glucose, thereby curbing post-meal glucose surges. It competitively blocks alpha-glucosidase activity responsible for converting oligosaccharides to monosaccharides, as depicted in **Figure 2**. This action decreases the demand for insulin release and promotes more stable daily glucose patterns. Furthermore, acarbose diminishes hyperinsulinaemia and reduces pancreatic beta-cell burden [5]. Prolonged administration not only controls postprandial glucose but also lowers fasting levels, rendering it valuable for comprehensive glycaemic management in diabetes.

For dietary carbohydrates to enter the circulation as an energy substrate, starches and oligosaccharides must be converted to monosaccharides—the sole form capable of intestinal absorption. This conversion depends on alpha-glucosidase enzymes located on the small intestine's brush border. Structurally similar to oligosaccharides, acarbose exhibits approximately 100,000-fold greater affinity for these enzymes. By blocking the degradation of complex carbohydrates in the small intestine, it postpones glucose uptake and minimises postprandial hyperglycaemia, as illustrated in **Figure 3**. Although its benefits are well established, acarbose remains underused worldwide, especially in early diabetes and prediabetes. It has been extensively employed in nations such as China for more than 20 years yet is often neglected elsewhere [6]. Consequently, acarbose competitively suppresses enzyme activity, resulting in reduced monosaccharide production, lessened insulin requirements, and lower post-meal glucose and insulin concentrations.

Different from conventional treatments that reduce glucose via enhanced insulin output, acarbose prevents postprandial elevations, producing an anti-hyperglycaemic rather than hypoglycaemic effect. As glucose rises are blunted, insulin secretion declines, alleviating the hyperinsulinaemia typical of prediabetes and early diabetes [6]. With continued use, acarbose eases beta-cell strain, improving insulin secretory efficiency, as indicated by reduced proinsulin levels relative to sulfonylurea therapy. Given the association of proinsulin with cardiovascular risk, acarbose may favourably affect long-term outcomes in T2DM. The slowed carbohydrate digestion also stimulates release of glucagon-like peptide-1 (GLP-1), an incretin hormone that delays gastric emptying, suppresses glucagon, and modulates insulin secretion according to glucose levels [6]. This pathway likely accounts for the observed reductions in both postprandial and fasting glucose during extended treatment. Notably, acarbose can be safely combined with other antidiabetic drugs, augmenting treatment effectiveness without added tolerability issues.

Pharmacokinetics

Acarbose functions primarily within the gastrointestinal (GI) tract, exhibiting very limited systemic absorption. Only under 2% of the intact compound enters the bloodstream, while roughly 35% is taken up as breakdown products. Intestinal bacteria and enzymes metabolize the drug, with the majority—about 51% of an ingested dose—being excreted via the faeces [7].

Metabolism

The intestinal flora plays a central role in breaking down acarbose. Bacterial enzymes convert it into derivatives

such as acarbose-glucose and glucose. Although human enzymes can partially process the drug, gut microbes further degrade it through hydrolysis, which may contribute to reduced effectiveness in certain individuals.

Administration and dosage

Acarbose is available in oral tablets of 25 mg, 50 mg, and 100 mg. The standard regimen involves taking it three times daily at the onset of each main meal. Treatment typically begins at 25 mg per dose, with gradual increases every 4-8 weeks to reach effective glycaemic management while keeping gastrointestinal adverse effects to a minimum. In patients below 60 kg body weight, the upper limit should remain at 50 mg three times daily. Safety data are lacking for children, pregnant or breastfeeding women, and those with significant renal impairment (serum creatinine exceeding 2.0 mg/dL) [8].

Drug interactions

Acarbose can decrease the absorption of certain medications, such as digoxin and valproic acid. When combined with insulin or sulfonylureas, it may heighten the risk of low blood glucose episodes. Moreover, pancreatic enzyme supplements (amylase, lipase, protease) can impair acarbose's activity.

Clinical efficacy of acarbose in type 1, type 2, and gestational diabetes

Acarbose provides notable advantages for individuals diagnosed with type 2 diabetes (T2D). Numerous randomized trials and systematic reviews have confirmed its ability to decrease post-meal blood glucose peaks and lower HbA1c values. One systematic review encompassing seven studies found a 35% reduction in cardiovascular event risk in T2D patients treated with acarbose. Long-term administration has further been associated with decreased fasting glucose concentrations, thereby enhancing comprehensive glucose regulation.

While its main indication is T2D, acarbose has proven effective in type 1 diabetes as an adjunct to insulin treatment. It attenuates postprandial glucose fluctuations and supports improved overall glucose management. Research in type 1 patients has documented declines in both HbA1c and post-meal glucose levels, establishing acarbose as a useful complementary option.

The drug has additionally been evaluated for gestational diabetes mellitus (GDM). It is recognized as a secure and reliable alternative to insulin or other oral glucose-lowering medications for addressing postprandial hyperglycaemia during pregnancy. Clinical investigations revealed no meaningful differences in newborn outcomes or birth weights among infants of mothers receiving acarbose versus those treated with insulin or glyburide, supporting its role as a feasible choice in GDM care. As monotherapy, acarbose exhibits substantial effectiveness in maintaining glucose control in T2DM cases.

Multiple controlled studies performed in China have validated its performance, especially when matched against alternative antidiabetic drugs such as vildagliptin, nateglinide, and metformin. Results indicated that acarbose comparably reduced HbA1c, fasting plasma glucose, and postprandial glucose levels. Moreover, it showed greater weight-lowering benefits relative to vildagliptin and metformin in Chinese individuals with T2DM [9]. In a 24-week trial by Usman *et al*. that enrolled 661 participants, acarbose (100 mg three times daily) was evaluated against vildagliptin (50 mg twice daily). Comparable declines were observed in fasting glucose (1.5±0.2 mmol/L with acarbose versus 1.2±0.1 mmol/L with vildagliptin) and HbA1c (1.4%±0.1% versus 1.3%±0.1%). Acarbose, however, achieved a more pronounced weight decrease (1.7±0.2 kg) compared to the near-neutral effect of vildagliptin (0.4±0.1 kg). Likewise, a 48-week investigation by Joshi *et al.* [10] involving 784 Chinese patients demonstrated equivalent HbA1c reductions with acarbose and metformin, accompanied by superior weight loss in the acarbose arm (2.52 kg versus 1.89 kg). Short-duration trials (2–9 weeks) comparing acarbose to nateglinide indicated equivalent control of postprandial glucose.

Nateglinide, which promotes insulin release in the early phase, operates differently from acarbose, the latter acting via slowed carbohydrate breakdown and uptake. Although both agents lowered post-meal glucose, nateglinide uniquely affected free fatty acids and insulin concentrations—outcomes not seen with acarbose [11]. Acarbose has similarly displayed positive results in subjects with impaired glucose tolerance (IGT).

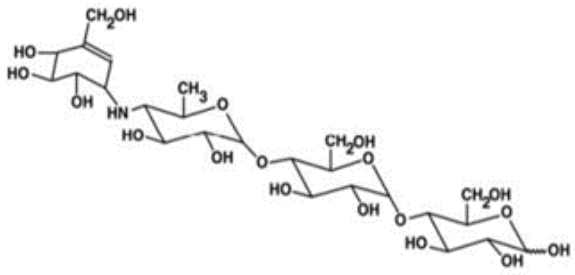

Figure 1. Chemical structure of acarbose [4]

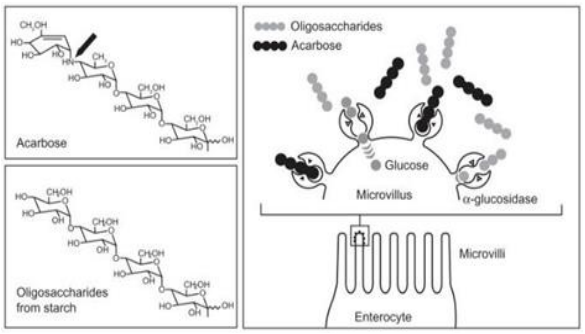

Figure 2: Mechanism of acarbose: competitive inhibition of intestinal enzymes responsible for oligosaccharide hydrolysis [5]

A trial with 252 Chinese participants revealed that acarbose markedly decreased post-meal glucose, serum insulin, and triglyceride concentrations, while achieving more substantial weight reduction than placebo [12]. Additionally, acarbose elevates both fasting and postprandial GLP-1 concentrations, contributing to enhanced glucose regulation. In general, acarbose performs especially well in individuals following Asian dietary patterns rich in carbohydrates, thereby amplifying its glucose-lowering impact. Its performance in managing glycaemia matches that of several common antidiabetic medications, and it provides extra advantages through weight loss, positioning it as an important choice for T2DM therapy.

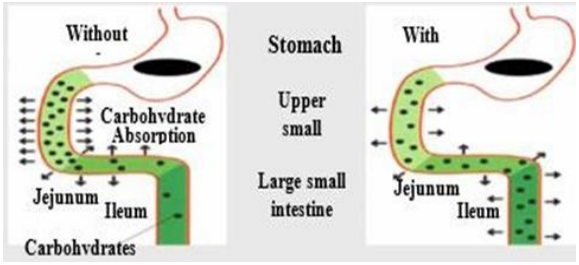

Figure 3: Non-systemic action of acarbose in postponing carbohydrate uptake [6]

Acarbose vs other antidiabetic agents: a comparative analysis

When assessing the role of any diabetes treatment, direct comparisons with alternative options for the same disorder are essential **(Table 1)**. Agents that stimulate insulin release, especially sulfonylureas, represent a widely adopted and cost-effective approach in global diabetes care. They function by attaching to the sulfonylurea receptor 1 on the ATP-sensitive potassium channel (KIR 6.2) in pancreatic beta cells. This binding blocks potassium efflux, depolarises the membrane, and activates voltage-gated calcium channels.

Calcium influx into the cell then triggers insulin release in a dose-related manner, independent of prevailing blood glucose levels—occurring during high, normal, or low glucose states. As a result, sulfonylureas carry a notable risk of hypoglycaemia, particularly in susceptible groups such as older adults, those with near-normal glucose, or renal impairment [12]. Hypoglycaemic episodes provoke counter-regulatory hormone surges (adrenaline, noradrenaline, glucagon), potentially causing severe events like arrhythmias, myocardial infarction, hypertensive emergencies, or stroke. These risks are heightened in elderly patients and those with longstanding diabetes, accounting for higher rates of unrecognised cardiovascular fatalities associated with sulfonylureas, glinides, and insulin.

Furthermore, sulfonylureas may exert direct cardiovascular harm, including arrhythmias from interactions with cardiac and vascular smooth muscle. Existing data, including from the UKPDS trial, suggest no long-term decrease in major cardiovascular outcomes, with benefits limited primarily to retinopathy [11]. By contrast, acarbose operates through a unique pathway, blocking digestive enzymes in the small intestine to limit carbohydrate uptake. As outlined in **Table 1**, acarbose presents distinct strengths and limitations relative to other antidiabetic drugs like sulfonylureas and metformin. For example, whereas sulfonylureas frequently cause weight increase and higher hypoglycaemia rates, acarbose avoids weight gain and poses minimal hypoglycaemic risk when used alone.

Table 1. Comparative summary of antidiabetic treatments [12]

Treatment	Site of Action	Body Weight	Hypoglycaemia (Monotherapy)	Long-term Efficacy	Gastrointestinal Side Effects	Safety (Related Diseases)	Mean Reduction in HbA1c
Acarbose	CH-digestive enzymes,	− (↓)	−	+	+	−	−1%

	small intestine						
Sulfonylurea	Beta cells	↑	↑	−	−	−	−1.25%
Glinides	Beta cells	↑	↑	−	−	?	−0.75%
Metformin	Insulin sensitivity, liver, peripheral tissues	−	−	−	+	−	−1%
Gliptins	DPP-4 enzymes, plasma, peripheral tissues	− (↓)	−	?	(+)	(+)	−0.75%
Pioglitazone	PPAR-gamma receptor, cell nucleus	↑	−	(+)	−	+	−1%

Table 2. Comparison of effects of acarbose and other anti-diabetic agents.

Study	Duration (weeks)	Drugs / Comparators	Patients (N)	Main Outcomes	Other Outcomes
Zhang *et al*. 13	24	Acarbose / Vildagliptin	441 / 220	Both groups showed a similar reduction in HbA1c and fasting plasma glucose	Body weight reduction was greater in the acarbose group compared to vildagliptin
Gu *et al*. 9	9	Acarbose / Nateglinide	16 / 16	Postprandial glucose decreased to a similar extent in both groups	Nateglinide significantly increased postprandial insulin release and reduced FFA concentrations
Moelansa *et al*. 15	2	Acarbose / Nateglinide	52 / 51	AUCpp and IGP decreased similarly in both groups	Both treatments significantly improved intra- and inter-day glycaemic fluctuations
Hedrington *et al*. 16	48	Acarbose / Metformin	391 / 393	HbA1c decreased to a similar degree in both groups	Body weight decreased more in the acarbose group compared to metformin

However, acarbose can cause gastrointestinal side effects that may affect adherence. Studies also indicate that DPP-4 inhibitors such as sitagliptin and vildagliptin provide similar efficacy and safety to acarbose, though they are associated with their own specific adverse reactions. In summary, while acarbose and other antidiabetic agents effectively manage blood glucose in T2DM, treatment selection must balance their mechanisms of action, side-effect profiles, and long-term health implications.

Combination therapy

Using acarbose in combination regimens has become a valuable approach for controlling type 2 diabetes, especially as an add-on to metformin, insulin, or other agents. As an alpha-glucosidase inhibitor, acarbose delays intestinal carbohydrate absorption, thereby reducing post-meal glucose excursions [13]. This mechanism makes it an effective complementary therapy across multiple treatment plans. **Table 2** illustrates the effects of acarbose compared with other antidiabetic agents used for glycaemic control.

Acarbose vs. vildagliptin: comparison in diabetes management

Acarbose, an alpha-glucosidase inhibitor, delays carbohydrate digestion in the small intestine, slowing glucose absorption and attenuating postprandial glucose spikes without stimulating insulin release. In contrast, vildagliptin, a DPP-4 inhibitor, enhances incretin

hormones (GLP-1 and GIP), thereby increasing glucose-dependent insulin secretion and suppressing glucagon release. This dual action of vildagliptin improves both fasting and postprandial glucose control. Acarbose primarily reduces post-meal glucose rises, leading to gradual HbA1c reductions, but its impact on fasting glucose is limited [14].

Monotherapy or combination studies typically show HbA1c reductions of 0.5% to 1.0% with acarbose. Vildagliptin, by targeting both fasting and postprandial glucose, usually achieves HbA1c reductions of 0.7% to 1.0%. Acarbose is frequently associated with gastrointestinal side effects such as diarrhoea, abdominal discomfort, flatulence, and nausea, resulting from undigested carbohydrates reaching the colon. These symptoms are generally transient and diminish with gradual dose titration. Vildagliptin, by comparison, has a lower incidence of gastrointestinal complaints and is generally better tolerated, though rare side effects include mild headache, dizziness, or arthralgia. Rare cases of pancreatitis have also been reported with DPP-4 inhibitors, including vildagliptin [15].

Acarbose is weight-neutral and carries no risk of hypoglycaemia when used alone, as it does not increase insulin secretion, making it suitable for patients concerned about weight gain or low glucose episodes. Vildagliptin is similarly weight-neutral and rarely causes hypoglycaemia unless combined with insulin secretagogues such as sulfonylureas. These characteristics make vildagliptin appealing for patients requiring balanced glycaemic control with low systemic risk.

Acarbose's use may be limited by gastrointestinal intolerance in some patients. Vildagliptin, with its broader effect on both fasting and postprandial glucose and better tolerability, is often preferred for patients who cannot tolerate acarbose's side effects. While acarbose targets postprandial hyperglycaemia specifically, vildagliptin provides more comprehensive daily glucose control. The decision between them depends on the patient's glucose profile, side-effect tolerance, and whether fasting or postprandial control is the primary goal.

Indications for acarbose

Acarbose is approved for adults with type 2 diabetes mellitus as an adjunct to diet and exercise when these measures alone are insufficient to achieve adequate glycaemic control [16]. Clinical trials have demonstrated significant HbA1c reductions. Data from six placebo-controlled studies showed the following average HbA1c decreases with three-times-daily dosing:

25 mg: 0.44% reduction
50 mg: 0.77% reduction
100 mg: 0.74% reduction
200 mg: 0.86% reduction
300 mg: 1.00% reduction

Although the 300 mg dose produced the largest reduction, the FDA-approved maximum is 100 mg three times daily. No statistically significant difference in HbA1c lowering was observed between the 50 mg, 100 mg, and 200 mg regimens. Combination with metformin or other antidiabetic agents often yields further glycaemic improvement. Some studies also suggest that patients on metformin plus acarbose experienced lower rates of COVID-19-related hospitalisation.

Off-label uses

Type 1 diabetes

Although not FDA-approved for this indication, acarbose has shown benefit as an adjunct to insulin in clinical studies, significantly reducing postprandial glucose excursions without increasing hypoglycaemic events.

Prediabetes

While not formally indicated, acarbose has been studied for its potential to delay progression from prediabetes to type 2 diabetes, with some trials reporting a substantial reduction in conversion rates.

Weight loss

Studies show that acarbose promotes modest weight reduction regardless of initial body weight, in addition to bettering glucose regulation.

Aging and other conditions

Certain investigations propose that acarbose might exert anti-aging properties and offer advantages in disorders such as polycystic ovary syndrome and ischaemic stroke, though these remain unapproved indications by the FDA.

Contraindications for acarbose

Acarbose is not recommended in certain patient groups because of possible risks and complications linked to its administration [17]. These encompass:

Hypersensitivity

Documented hypersensitivity to acarbose or any excipients.

Diabetic ketoacidosis

Unsuitable for individuals with diabetic ketoacidosis, where prompt glucose lowering is required.

Liver cirrhosis

Patients with cirrhosis should refrain from acarbose, as it could worsen hepatic dysfunction.

Inflammatory bowel disease

Conditions such as Crohn's disease or ulcerative colitis may deteriorate with acarbose therapy.

Colonic ulceration

The drug could aggravate pre-existing ulcers in the colon.

Intestinal obstruction

Acarbose is contraindicated in cases of known, suspected, or predisposition to bowel obstruction.

Chronic intestinal diseases

Individuals with longstanding gut disorders impairing digestion or absorption risk heightened effects from increased intestinal gas.

Safety and side effects profile of acarbose in diabetes management

Data from clinical trials and post-marketing monitoring reveal that serious adverse reactions with acarbose are uncommon. The primary complaints involve mild-to-moderate gastrointestinal issues, such as flatulence, bloating, diarrhoea, and indigestion.

These arise from bacterial fermentation of undigested carbohydrates in the colon and tend to subside over time, potentially due to enhanced α-glucosidase activity in distal small intestinal regions. Starting with low doses and titrating slowly generally makes these effects short-lived and tolerable [18]. Systemic absorption after oral intake is negligible (under 2%), minimising the chance of non-gastrointestinal adverse reactions. Indeed, no systemic toxicities have been identified in trials [19]. Longitudinal monitoring showed no meaningful changes in haematology, urinalysis, or biochemical parameters.

Tolerance to acarbose remains high across age groups, comorbidities, and polypharmacy scenarios. Trials repeatedly confirm its favourable tolerability profile. For example, a 100 mg three-times-daily dose, either as monotherapy or combined with other antidiabetic agents, was safe and well accepted in Chinese individuals with type 2 diabetes [20]. The lack of associated weight increase or hypoglycaemia represents a key advantage in personalised treatment selection.

No evidence of cardiovascular toxicity has emerged with acarbose, and systemic events are exceptionally infrequent, supported by broad clinical datasets and worldwide usage. This strong safety record stems from its predominantly local intestinal action and minimal bioavailability. A prospective 5-year surveillance study in 1,996 patients reported no severe or lethal events. Similarly, a double-blind trial in 1,429 subjects with impaired glucose tolerance treated for 3 to 5 years found comparable adverse event rates between acarbose and placebo groups, without any serious incidents [21].

Rare, reversible elevations in liver enzymes were noted in the first decade post-approval, but these did not exceed placebo rates in controlled studies, amounting to only 19 cases across 500,000 treated patients. No such elevations occurred in impaired glucose tolerance trials.

Evidence from diabetic patients with raised liver enzymes suggests acarbose may even ameliorate chronic hepatic conditions. In summary, results from major randomised trials and real-world data position acarbose as one of the most secure antidiabetic options, suitable for standalone or combination use.

Future directions for acarbose in diabetes treatment: emerging trends and research

With advancing insights into diabetes care, acarbose retains an important position in glucose management, especially for type 2 diabetes patients. Current developments and active studies are uncovering novel applications, emphasising its broader potential and extended clinical utility.

Combination therapies

Investigations are increasingly examining acarbose alongside diverse antidiabetic drugs. Ongoing work evaluates its complementary actions with modern agents, including GLP-1 receptor agonists and sodium-glucose cotransporter-2 (SGLT-2) inhibitors. Such pairings may optimise glycaemic outcomes while reducing drawbacks like weight gain or hypoglycaemia commonly seen with older regimens.

Weight management

Given the increasing rates of obesity in individuals with diabetes, acarbose is receiving growing interest for its potential contributions to body weight regulation. Ongoing studies are evaluating its capacity to facilitate weight reduction or avert weight increase, particularly when paired with dietary and physical activity modifications. This approach may deliver combined advantages of superior glucose regulation and better overall metabolic status.

Long-term outcomes

As additional extended-duration evidence emerges, forthcoming research is expected to emphasise the enduring impact of acarbose treatment on cardiovascular events and total mortality. Owing to its robust safety record and negligible risk of systemic adverse reactions, acarbose could establish itself as an essential element in prolonged diabetes care protocols.

Conclusion

In summary, acarbose exhibits considerable promise as an effective treatment option for regulating blood glucose in patients with type 2 diabetes mellitus. Through blocking carbohydrate digestion in the small intestine, acarbose promotes slower glucose uptake, thereby supporting stable blood glucose concentrations and advancing comprehensive glycaemic management. Data from clinical investigations affirm its value in lowering HbA1c values and optimising patient results, especially in combination with other glucose-lowering agents.

Although transient mild gastrointestinal disturbances may arise, these are typically short-lived and do not overshadow the therapeutic advantages. Considering the escalating prevalence of diabetes and its related complications, integrating acarbose into treatment plans remains highly relevant. Upcoming investigations should further examine its prolonged benefits, ideal pairings with additional medications, and suitability across varied demographic groups.

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