ISSN 2320-9968

Vol. 14, No.1 January-March 2026

Journal of Diabetes Education

To Dispel Darkness Of Diabetes

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Tresiba® aPI

Generic Name: Insulin degludec (Monocomponent, biosynthetic r-DNA origin) **Brand Name:** Tresiba® Penfill®, Tresiba® FlexTouch®, 100 units/ml, solution for subcutaneous injection. **Presentation:** Tresiba® Penfill®, Tresiba® FlexTouch® - 1 ml solution contains 100 units insulin degludec (equivalent to 3.66 mg insulin degludec). One cartridge/pre-filled pen contains 300 units of insulin degludec in 3 ml solution. **Indication:** Treatment of diabetes mellitus in adults, adolescents and children from the age of 1 year. **Dosing and administration:** Tresiba® is a basal insulin for once-daily subcutaneous administration at any time of the day, preferably at the same time every day. Initiation: Patients with type 2 diabetes mellitus: The recommended daily starting dose is 10 units followed by individual dosage adjustments. Patients with type 1 diabetes mellitus: Tresiba® is to be used once daily with mealtime insulin and requires subsequent individual dosage adjustments. Tresiba® is for subcutaneous use only. Tresiba® must not be administered intravenously as it may result in severe hypoglycaemia. Tresiba® must not be administered intramuscularly as it may change the absorption. Tresiba® must not be used in insulin infusion pumps. Patients should be instructed to always use a new needle. Injection sites should always be rotated within the same region in order to reduce the risk of lipodystrophy and cutaneous amyloidosis. **Children:** Tresiba® can be used in adolescents and children from the age of 1 year **Pregnancy and lactation:** The treatment with Tresiba® may be considered during pregnancy, if clinically needed. Careful monitoring of glucose control is recommended and the insulin dose adjusted on an individual basis. There is no clinical experience with Tresiba® during breast-feeding. **Elderly:** Tresiba® can be used in elderly. **Special populations and conditions:** Tresiba® can be used in renal and hepatic impaired patients. **Contraindications:** Hypersensitivity to the active substance or to any of the excipients **Special warnings and precautions:** Hypoglycaemia - Omission of a meal or unplanned strenuous physical exercise may lead to hypoglycaemia. Hypoglycaemia may occur if the insulin dose is too high in relation to the insulin requirement. Hyperglycaemia - Administration of rapid-acting insulin is recommended in situations with severe hyperglycaemia. Inadequate dosing and/or discontinuation of treatment in patients requiring insulin may lead to hyperglycaemia and potentially to diabetic ketoacidosis. Skin and subcutaneous tissue disorders - Patients must be instructed to perform continuous rotation of the injection site to reduce the risk of developing lipodystrophy and cutaneous amyloidosis. There is a potential risk of delayed insulin absorption and worsened glycaemic control following insulin injections at sites with these reactions. A sudden change in the injection site to an unaffected area has been reported to result in hypoglycaemia. **Undesirable effect:** With insulin preparations, allergic reactions may occur. Lipodystrophy and cutaneous amyloidosis may occur at the injection site and delay local insulin absorption. Continuous rotation of the injection site may help to reduce or prevent these reactions. Injection site reactions occurred in patients treated with Tresiba®. These reactions are usually mild and transitory and they normally disappear during continued treatment. **Shelf Life:** The shelf life of the drug product is 30 months when stored at 2°C-8°C. **Storage:** Before first use: Store in a refrigerator (2°C – 8°C). Keep away from the freezing element. Do not freeze. Protect from light. After first opening or carried as a spare: product may be stored for a maximum of 8 weeks. Do not store above 30°C. Tresiba® FlexTouch® can be stored in a refrigerator (2°C – 8°C). Tresiba® Penfill® should not be refrigerated. Protect from light. **Disclaimer:** The abbreviated package insert is updated from the CDSCO approved package insert dated 05 Aug 2024 for Tresiba® Penfill® and Tresiba® FlexTouch®. Tresiba®, Penfill®, FlexTouch®, NovoFine® and NovoTwist® are trademarks owned by Novo Nordisk A/S, Denmark. Imported by: Novo Nordisk India Private Limited, Bangalore Note: For detailed information on this product, please refer to full package insert* * The full prescribing information can be obtained at no cost from Novo Nordisk. For full prescribing information please contact +91-080-40303200 or write to us at INAgree@novonordisk.com or reach us at Novo Nordisk India Private Limited, Nxt Tower-2, Floor 1 & 2, Embassy Manyata Business Park, Nagavara Village, Kasaba Hobli, Bangalore-560 045, India.

Fiasp® aPI

Generic Name: Insulin aspart **Presentation:** Fiasp® FlexTouch® 3 ml, Fiasp® Penfill® 3 ml, Fiasp® Vial 10 ml. All presentations contain 100 units/ml insulin aspart. **Indications:** Treatment of diabetes mellitus in adults, adolescents and children from the age of 1 year. There is no clinical experience with the use of Fiasp® in children below the age of 1 year. **Dosage and administration:** Fiasp® is a mealtime insulin for subcutaneous administration up to 2 minutes before the start of the meal, with the option to administer up to 20 minutes after starting the meal. Dosing with Fiasp® is individual and determined in accordance with the needs of the patient. Fiasp® given by subcutaneous injection should be used in combination with intermediate-acting or long-acting insulin given at least once a day. In a basal-bolus treatment regimen approximately 50% of this requirement may be provided by Fiasp® and the remaining by intermediate-acting or long-acting insulin. The individual total daily insulin requirement in adults, adolescents and children may vary and is usually between 0.5 and 1 unit/kg/day. Blood glucose monitoring and insulin dose adjustment are recommended to achieve optimal glycaemic control. **Initiation:** The recommended starting dose in insulin naïve patients with type 1 diabetes is approximately 50% of the total daily insulin dose and should be divided between the meals based on the size and composition of the meals. As a general rule, 0.2 to 0.4 units of insulin per kilogram of body weight can be used to calculate the initial total daily insulin dose in insulin naïve patients with type 1 diabetes. In type 2 diabetes mellitus patients, the suggested initial dose is 4 units at one or more meals. The number of injections and subsequent titration will depend on the individual glycaemic target and the size and composition of the meals. Dose adjustment may be considered daily based on self-measured plasma glucose (SMPG) on the previous day(s) according to Table 1. • Pre-breakfast dose should be adjusted according to the pre-lunch SMPG the previous day • Pre-lunch dose should be adjusted according to the pre-dinner SMPG the previous day • Pre-dinner dose should be adjusted according to the bedtime SMPG the previous day 'No dose adjustment of faster aspart is required if the patient's SMPG falls in the range of 71-108 mg/dL. Alternatively, if the patient's SMPG falls below 71 mg/dL, reduce the dose of faster aspart by 1 unit and if the patient's SMPG falls above 108 mg/dL, increase the dose of faster aspart by 1 unit.' **Paediatric population:** Fiasp® can be used in adolescents and children from the age of 1 year. **Pregnancy and lactation:** Fiasp® can be used in pregnancy. No restrictions on use during breast-feeding. Elderly (≥65 years old): The safety and efficacy of Fiasp® have been established in elderly patients aged 65 to 75 years. The therapeutic experience in patients ≥ 75 years of age is limited. Special populations and conditions: Renal impairment may reduce the patient's insulin requirements. In patients with renal impairment, glucose monitoring should be intensified and the dose adjusted on an individual basis. Hepatic impairment may reduce the patient's insulin requirements. In patients with hepatic impairment, glucose monitoring should be intensified and the dose adjusted on an individual basis. **Contraindications:** Hypersensitivity to the active substance or to any of the excipients **Special warnings and precaution:** Hypoglycaemia: Omission of a meal or unplanned, strenuous physical exercise may lead to hypoglycaemia. Hypoglycaemia may occur if the insulin dose is too high in relation to the insulin requirement. Inadequate dosing or discontinuation of treatment may lead to hyperglycaemia and diabetic ketoacidosis. Patients must be instructed to perform continuous rotation of the injection site to reduce the risk of developing lipodystrophy and cutaneous amyloidosis. There is a potential risk of delayed insulin absorption and worsened glycaemic control following insulin injections at sites with these reactions. A sudden change in the injection site to an unaffected area has been reported to result in hypoglycaemia. Transferring a patient to another type or brand of insulin should be done under strict medical supervision. **Undesirable effect:** Lipodystrophy and cutaneous amyloidosis may occur at the injection site and delay local insulin absorption. Continuous rotation of the injection site within the given injection area may help to reduce or prevent these reactions **Shelf-life:** The shelf life of the drug product is 30 months when stored at 2-8°C. The expiry date of the drug product is indicated on the carton/label. Special precautions for storage: Store in a refrigerator (2°C–8°C). Do not freeze. Keep away from the freezing element. **Disclaimer:** The abbreviated package insert is updated from the CDSCO approved package insert for Fiasp® FlexTouch® dated 22 Mar 2024, Fiasp® Penfill® dated 27 May 2024 and Fiasp® Vial 10 ml dated 10 Jun 2024. Fiasp® and Apis bull logo is a registered trademark owned by Novo Nordisk A/S and registered in Denmark. Imported by: Novo Nordisk India Private Limited, Bangalore *For full prescribing information, please contact +91-080-40303200 or write to us at INAgree@novonordisk.com or reach us at Novo Nordisk India Private Limited, Nxt Tower-2, Floor 1 & 2, Embassy Manyata Business Park, Nagavara Village, Kasaba Hobli, Bangalore-560045, India. Note: For detailed information on this product, please refer to full package insert*.

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Reference: 1. Kalra S et al. Advances in Therapy. 2018 Jul;35:928-36. 2. Tresiba® pack insert as per CDSCO approval r-DNA-1101(12)/48/2024-e-office dated 22 Jul 2024. 3. Fiasp® CDSCO approved pack insert version dated 10 Jun 2024. 4. Heise T et al. Clin Pharmacokinet 2017;56:551-559.

IN25FSP00057 | Review Date: 09 June 2025

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JOURNAL OF DIABETES EDUCATION

To Dispel Darkness of Diabetes

Vol. 14 Number 1 January-March 2026

CONTENTS

1. **Anemia in Diabetic Kidney Disease, a Nephrologist's Perspective** 02
Rushi Deshpande, Ashitosh Patil

2. **Fuel Metabolism in Intermittent Fasting** 09
Meenakshi Bajaj

3. **Non-Weight-Bearing Exercise for Diabetes Management** 14
Shreyas Katharani

4. **Questions and Answers** 18

5. **Recipes** 20

6. **How Knowledgeable Are You?** 22

7. **Myths and Facts** 23

ANEMIA IN DIABETIC KIDNEY DISEASE, A NEPHROLOGIST'S PERSPECTIVE

PATHOPHYSIOLOGY, DIAGNOSTIC APPROACH AND THERAPEUTIC STRATEGIES

Rushi Deshpande*, Ashitosh Patil**

INTRODUCTION

Diabetes mellitus is the leading cause of chronic kidney disease worldwide. Anemia frequently develops in diabetic kidney disease (DKD), even in early stages and is associated with higher cardiovascular morbidity, lower quality of life and faster progression to end-stage kidney disease (ESKD) (1,2). Patients with DKD often present with anemia earlier and more severely than non-diabetic CKD populations (3,4). Early recognition and intervention are crucial.

EPIDEMIOLOGY

Global anemia prevalence in diabetic kidney disease averages 27.0%, with dramatic variation from 3.8% (Iceland) to 84.3% (Nepal). India shows high burden with 70.3% prevalence in Delhi and 60.0% in Chennai, ranking among the most affected countries globally. South-East Asia region including India, demonstrates the highest mean regional prevalence at 50.2%, significantly exceeding the global average (5).

FIGURE 1

PREVALENCE OF ANEMIA IN DIABETIC KIDNEY DISEASE (5)

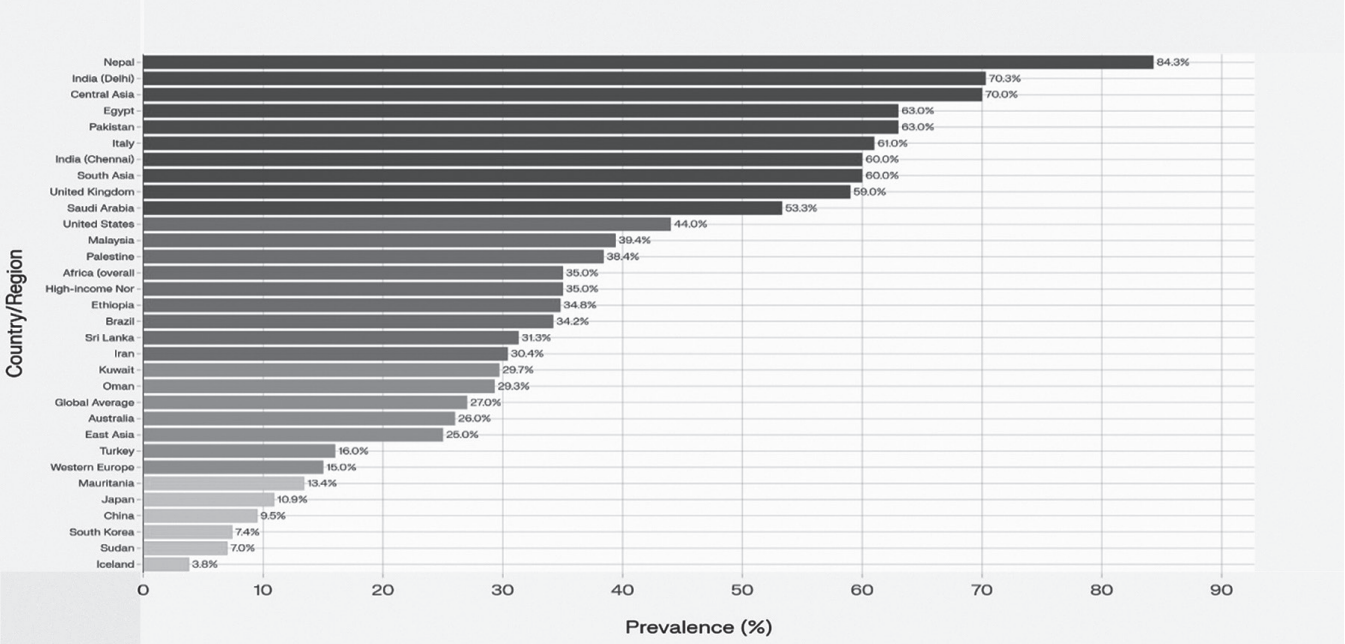

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TABLE 1

PATHOPHYSIOLOGIC MECHANISMS OF ANEMIA IN DIABETIC KIDNEY DISEASE (2,8)

Mechanism	Pathophysiology	Clinical Implication
Erythropoietin deficiency	Loss of EPO-producing renal fibroblasts due to fibrosis, ischemia, hyperglycemia-induced injury	Inappropriately low erythropoiesis; early anemia
Iron dysregulation	Hepcidin-mediated functional iron deficiency from chronic inflammation; poor absorption	Microcytic anemia, erythropoiesis-stimulating agents (ESA) hypo responsiveness
Chronic inflammation	IL-6, TNF-α, IL-1β impair erythropoiesis; oxidative stress shortens RBC lifespan	ESA resistance, fatigue, cardiovascular stress
Uremic toxins	Guanidines, indoxyl sulfate suppress bone marrow, increase RBC destruction	Reduced reticulocyte response; severe anemia in advanced CKD
Vitamin deficiency	B12/folate deficiency from malnutrition or drugs	Megaloblastic features; worsens anemia
Medications and comorbidities	ACE inhibitors, ARBs, antidiabetic drugs; proteinuria, neuropathy	Contribute to anemia severity and ESA resistance

PATHOPHYSIOLOGY

Anemia in DKD is multifactorial, involving endocrine, inflammatory, hematologic and metabolic mechanisms. Table 1 above summarizes the main pathophysiologic contributors.

DIAGNOSTIC APPROACH

Diagnosis requires a systematic approach to determine the primary cause and guide therapy. Table 2 outlines the recommended diagnostic work-up.

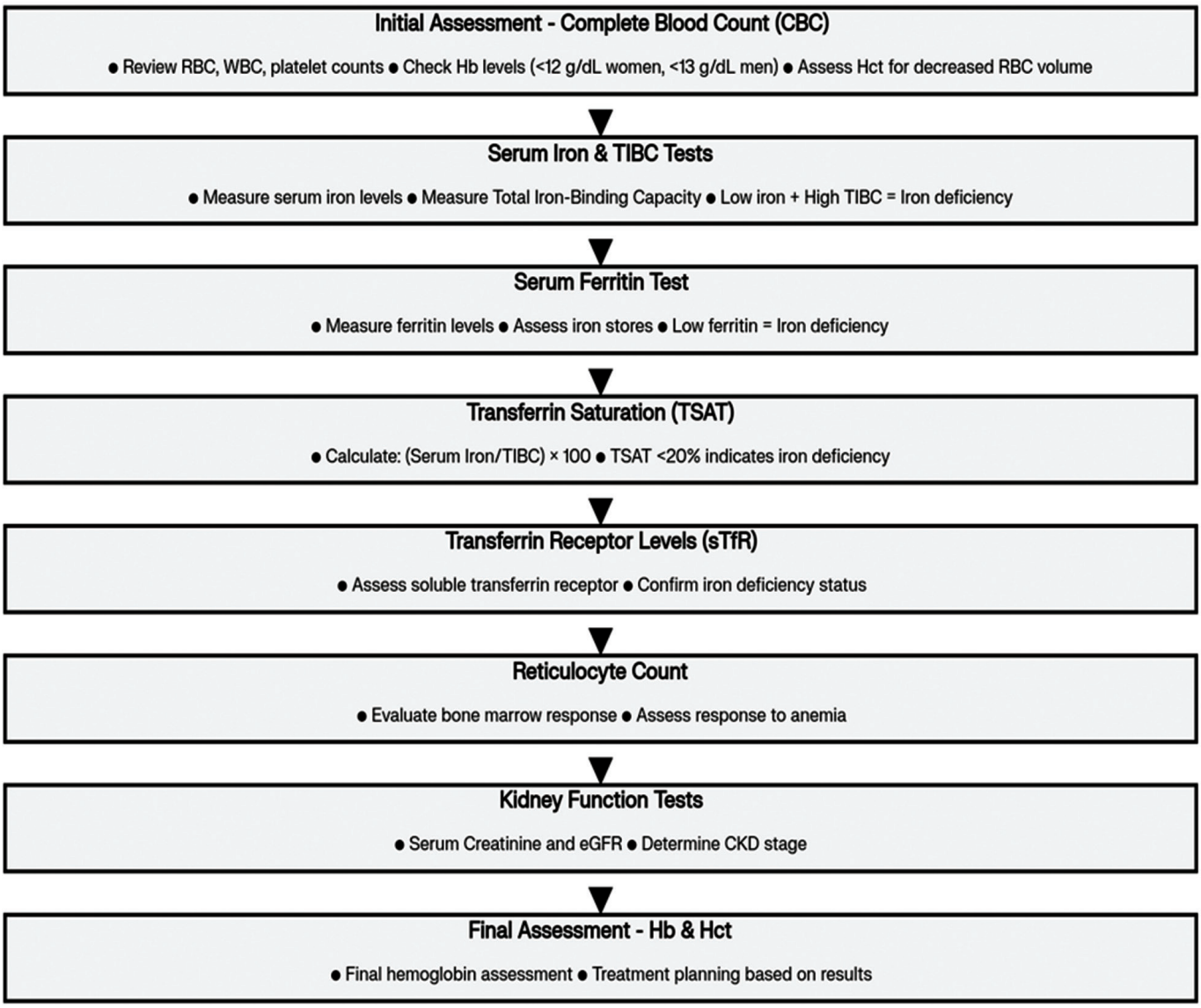
TABLE 2

DIAGNOSTIC WORK-UP ALGORITHM FOR ANEMIA IN DKD (2,8)

Test	Purpose	Interpretation / Threshold
CBC and hemoglobin	Identify anemia, RBC indices	Hemoglobin <13 g/dL (men) <12 g/dL (women)
Reticulocyte count	Assess marrow response	Low: production defect; High: blood loss/ hemolysis
Peripheral smear	Assess Morphology	abnormalities in size, shape and color
Ferritin and TSAT	Evaluate iron stores	TSAT <20–30% Ferritin <100 ng/mL (non-dialysis) Ferritin <500 ng/mL (dialysis)
Serum iron / TIBC	Complement iron assessment	Low iron, high TIBC: iron deficiency
sTfR	Assess iron status and erythropoiesis; differentiates iron deficiency anemia (IDA) from anemia of chronic disease (ACD)	Normal: 0.8–2.5 mg/L IDA: ↑ (>2.5 mg/L) ACD: Normal/slightly ↑
CRP / IL-6	Detect inflammation	Elevated: functional iron deficiency likely
B12 / folate	Identify megaloblastic causes	Low: correct vitamin deficiency
Renal function and urine	Stage CKD, detect proteinuria	Declining eGFR, proteinuria correlates with anemia severity
Occult blood / GI evaluation	Rule out blood loss	Positive: treat source of bleeding
Serum EPO	Rarely needed; severe unexplained anemia	Low EPO suggests EPO deficiency
Bone marrow biopsy	Reserved for unexplained/ refractory anemia	Abnormal: consider marrow disorder

FIGURE 2

DIAGNOSTIC FLOWCHART FOR IRON-DEFICIENCY ANEMIA IN CKD (2,8)



MANAGEMENT STRATEGIES

Therapy targets underlying deficiencies, optimizes hemoglobin and reduces complications. (Figure 2)

Iron Therapy

- ***Rationale and Indications:*** Iron repletion is foundational (6,7). Both absolute and functional iron deficiency impair erythropoiesis and reduce ESA responsiveness. Indications for iron therapy include TSAT ≤20% and/or ferritin below context-specific thresholds (eg. ferritin <100 ng/mL in non-dialysis CKD, <200 ng/mL in dialysis).
- ***Oral Versus Intravenous Iron:*** Oral iron is inexpensive and accessible but often limited by poor absorption and gastrointestinal adverse effects. Hepcidin-mediated blockade in CKD reduces oral iron efficacy, particularly in inflammatory states and advanced CKD. Intravenous iron (formulations include iron sucrose, ferric carboxymaltose, iron dextran, ferric gluconate) reliably repletes iron stores and improves Hemoglobin and ESA responsiveness. Real-world practice favors intravenous iron in CKD stage G4–G5, ESA-

treated patients and those with intolerance or inadequate response to oral iron.

- ***Safety Considerations:*** Intravenous iron is generally safe when administered using recommended dosing and monitoring protocols. Transient post-infusion reactions and rare anaphylaxis (mainly with older dextran preparations) are concerns. Emerging safety data do not show clear excess risk of infection with modern intravenous iron when used appropriately, but vigilance is warranted.

Erythropoiesis-Stimulating Agents (ESAs)

- ***Erythropoiesis-Stimulating Agents (ESAs) Indications and Dosing Principles***

  ESAs (epoetin alfa, darbepoetin alfa, methoxy polyethylene glycol-epoetin beta) are effective in increasing hemoglobin and reducing transfusion needs. Initiation typically follows failure of iron repletion to correct hemoglobin below prespecified thresholds (commonly ≤10 g/dL), with individualized decision-making. Dosing is weight-based and titrated to response, avoiding rapid hemoglobin rises and high maintained hemoglobin targets.

- ***Risks and Evidence Large trials***

  These trials have demonstrated symptomatic benefit but have raised safety concerns when aiming for near-normal hemoglobin levels. Studies showed increased risk of cardiovascular events, thrombosis and stroke when hemoglobin targets exceeded 13 g/dL or when ESAs were used aggressively; thus, moderating targets to approximately 10–11.5 g/dL is widely recommended (8,9,10).

Hypoxia-Inducible Factor Prolyl-Hydroxylase Inhibitors (HIF-PHIs) (11,12)

- Hypoxia-Inducible Factor Prolyl Hydroxylase Inhibitors (HIF-PHIs) HIF-PHIs (roxadustat, daprodustat, vadadustat, desidustat) (13,14) are oral agents that stabilize HIF, increasing endogenous EPO synthesis and improving iron metabolism via hepcidin suppression. Clinical trials indicate non-inferiority to ESAs for hemoglobin correction and improved iron utilization, with potential advantages in ESA-hyporesponsive patients. Long-term cardiovascular and malignancy safety data are maturing; vigilance and post-marketing surveillance continue. In India, some HIF-PHIs are approved and being adopted, particularly where oral administration and ESA-sparing effects are desirable.
- Effective in both dialysis and non-dialysis CKD.

Supportive Measures

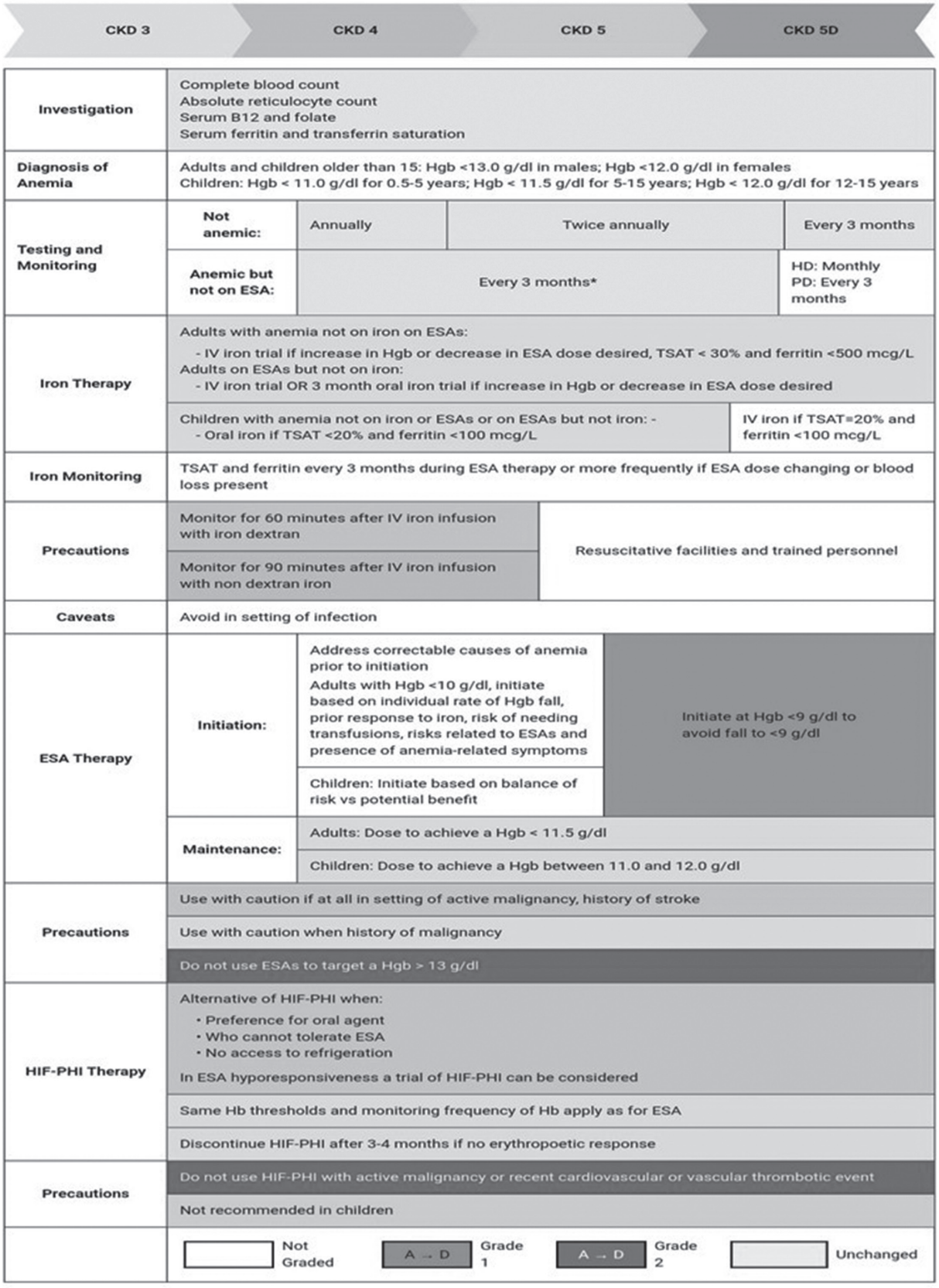
- Transfusion is reserved for severe or symptomatic anemia. In transplant candidates, transfusion is minimized to avoid alloimmunization. Addressing secondary hyperparathyroidism, optimizing nutrition and treating occult bleeding sources are important adjunctive measures
- SGLT2 Inhibitors and Ancillary Therapies: SGLT2 inhibitors (eg., empagliflozin, canagliflozin, dapagliflozin) have consistently increased haemoglobin and haematocrit across trials in CKD and diabetes populations, likely through modest plasma volume contraction and improved renal oxygenation and erythropoietin dynamics. These effects may complement direct anemia-targeted therapies and are integrated into contemporary DKD management (15)

Monitoring and Targets

- Targets: Consensus recommendations favor conservative hemoglobin targets to balance symptomatic benefit and safety: typically, 10–11.5 g/dL in most CKD patients receiving ESA therapy. Iron indices targets include TSAT 20–30% and ferritin thresholds individualized by context (eg., ferritin <500–800 ng/mL as an upper safety limit during iron repletion).
- Monitoring Schedule After initiating therapy, check hemoglobin every 2–4 weeks until stable, then every 1–3 months depending on CKD stage and therapy. Monitor TSAT and ferritin at least every 3 months in treated patients and more often when dosing intravenous iron or changing ESA therapy. (Figure 3)

FIGURE 3

MANAGEMENT ALGORITHM FOR ANEMIA IN DKD (16)



COMPLICATIONS

Anemia in DKD is associated with:

- Increased cardiovascular morbidity (heart failure, left ventricular hypertrophy)
- Accelerated CKD progression
- Fatigue and reduced quality of life
- Higher hospitalization and mortality rates (1,3,6,8)

INDIAN CONTEXT

Challenges and Opportunities: In India, barriers to optimal anemia care in DKD include delayed CKD diagnosis, financial constraints limiting ESA and intravenous iron use, variable availability of HIF-PHIs and high burden of nutritional anaemia and infectious comorbidity. Practical adaptations include using oral or low-cost intravenous iron when feasible, biosimilar ESAs, task-shifting for screening and integrating anemia care into diabetes and CKD public health programs. Capacity-building for dialysis centres to offer safe intravenous iron and ESA therapy is a priority. (Table 3)

TABLE 3

PRACTICAL RECOMMENDATIONS
(Nephrologist Perspective) (1)

Recommendation	Details / Practical Notes
Early Screening	Screen for anemia early in DKD; repeat periodically according to CKD stage
Address Reversible Causes	Correct iron, B12, folate deficiencies; investigate occult blood loss
Iron Therapy	Prefer intravenous iron for moderate-to-severe CKD or functional iron deficiency; use oral iron selectively in early CKD or resource-limited settings with close monitoring
ESA Therapy	Initiate when hemoglobin ≤10 g/dL after iron repletion; individualize targets (~10–11.5 g/dL); titrate slowly to minimize adverse events
HIF-PHI Therapy	Consider for oral therapy or ESA-hyporesponsive; discuss evolving safety data with patients
SGLT2 Inhibitors	Integrate into DKD care to improve cardiorenal outcomes; use as adjunct to optimize hemoglobin dynamics
Transfusion Management	Minimize transfusions in transplant candidates; coordinate with transplant teams
Practical Considerations in India	Adapt guideline-driven care to local resources; prioritize capacity building for intravenous iron and ESA availability

CONCLUSION

Anemia is a common complication of DKD, contributing to cardiovascular morbidity, reduced quality of life and accelerated progression to ESKD. Its pathophysiology is multifactorial, involving erythropoietin deficiency, iron dysregulation due to chronic inflammation and hepcidin elevation, shortened red blood cell lifespan and bone marrow suppression. Accurate diagnosis requires systematic screening with hemoglobin assessment, iron studies, reticulocyte counts, inflammatory markers and evaluation for alternative causes. Management should be individualized and evidence-based which

includes iron supplementation, erythropoiesis-stimulating agents (ESAs), hypoxia-inducible factor prolyl-hydroxylase inhibitors (HIF-PHIs), disease-modifying agents like SGLT2 inhibitors into a coordinated care plan and supportive measures. Strategies must be adapted to local resource settings, particularly in low and middle-income countries such as India. Early detection and individualized therapy, supported by continued research and strengthened public health initiatives, are essential to mitigate complications, reduce the global burden of anemia in DKD and improve patient outcomes.

FUTURE DIRECTIONS AND RESEARCH PRIORITIES

- Address long-term cardiovascular and malignancy safety data for HIF-PHIs.
- Develop strategies to modulate hepcidin therapeutically.
- Create scalable models for anemia care in LMICs (low and middle-income countries).
- Conduct trials in diverse populations, including Indian cohorts, to guide guideline adaptation and ensure equity of care.

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FUEL METABOLISM IN INTERMITTENT FASTING

Meenakshi Bajaj*

INTRODUCTION

The metabolic function in humans is influenced not only by the type of food we consume but also by the time we consume it. Chrononutrition is an emerging field of research that investigates the association between the timing of food intake and endogenous circadian rhythm and how it influences metabolic regulation. It plays a key role in managing the daily shifts of glycemic control, insulin resistance, lipid metabolism and hormonal regulation, thereby modifying the efficacy of nutrition absorption, ingestion, digestion and storage throughout the day. The core aspects of nutrition, such as total calorie intake and nutrient composition, remain fundamental in overall health. However, recent studies suggest that the timing of meals can also have a significant impact on metabolic outcomes (2).

Based on these concepts, intermittent fasting (IF) is the current popular dietary strategy, which promotes a shift in the body's energy use from relying on glucose to utilising fat and ketones, rather than slowing metabolic rate. Fasting durations typically range from 12 to 48 hours and include approaches such as 1–2 days per week or limiting daily intake to a 4–12-hour window. This daily strategy, known as time-restricted feeding (TRF) or time-restricted eating (TRE), is classified as early (eTRF), delayed (dTRF) or late (lTRF) depending on the eating window (3). Modified fasting regimens limit caloric intake on designated "fasting" days to ~20–25% of usual intake (≈400–800 kcal), while other days are unrestricted. This creates a weekly negative energy balance and triggers fasting-like metabolic responses (ketosis, lowered insulin, altered IGF-1 and mTOR), but with better adherence and tolerability than complete fasts (4).

CIRCADIAN RHYTHM & METABOLIC REGULATION

The circadian rhythms are the body's natural biological clock that regulates the metabolic homeostasis, sleep-wake cycles and various body functions for around 24 hours in response to the light and darkness of the environment. The central clock and several peripheral clocks work together to develop a coordinated, hierarchical circadian system that regulates circadian rhythms. The central biological clock is the suprachiasmatic nucleus (SCN) of the hypothalamus. The SCN regulates the body's functions by aligning body processes and behaviors with the rotation of day and night on Earth. Whereas, peripheral clocks, or circadian rhythms, are also seen in organs and tissues, including the liver, pancreas, gut, skeletal muscle and fat. The body requires the biological clock for memory conservation, repair of body tissue, defence against infections and the regulation of body metabolism. Through sleep-wake cycles, the biological clock influences metabolism and nutrition (1).

Disruption of the circadian rhythm increases the risk of obesity, insulin resistance, fatty liver disease and metabolic syndrome through its adverse impact on fat metabolism control, glucose homeostasis and energy balance within several organs. The body clock can also reflect when the body has irregular increases or decreases in body temperature. For about twenty-four hours, the biological clock regulates the body when functioning normally.

HORMONAL RHYTHMS

Metabolic activities are closely linked to endogenous hormonal cycles. Cortisol peaks around 8 am, preparing the body for daytime energy requirements. Ghrelin, the appetite

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hormone, rises around 8 am, 1 pm and 6 pm in a day to stimulate hunger. Adiponectin peaks around 11 am, thereby improving insulin sensitivity and fat metabolism. Insulin levels peak in the afternoon, around 5 pm, when insulin sensitivity is already lower and leptin rises around evening at 7 pm, signaling satiety and helping reduce fat storage in the body. Aligning meals with these natural rhythms, especially eating earlier in the day, helps in better insulin response, glucose tolerance and lipid metabolism (13,15).

Circadian rhythms are associated with the suprachiasmatic nucleus (SCN) and each organ's function is at its highest potential when it is properly aligned (15).

Adipocytes normally store fat at night and release it during the day, but with a disrupted clock, they store too much fat and fail to mobilize it, increasing the risk of obesity and insulin resistance. The liver, under normal rhythms, manages glycogen, glucose, lipids and proteins in sync with feeding and fasting. When disrupted, it produces glucose at the wrong time and poorly handles lipids, which contributes to high blood glucose and fatty liver disease (15).

The pancreas is equally affected; usually, α-cells release glucagon and β-cells secrete insulin with greater sensitivity throughout the day. Disturbed rhythms cause tissues to become less sensitive and insulin production to be slowed, which raises fasting glucose levels. Nutrient absorption and microbial activity in the gut and microbiota follow day-night cycles; however, misalignment results in dysbiosis, reduced synthesis of bile acid and short chain fatty acids and disturbed metabolism (15).

Similarly, during the day, skeletal muscle is often very responsive to insulin and effectively absorbs fat and glucose. Because muscles absorb less glucose when this sensitivity is disrupted, blood glucose levels rise (15).

Adiponectin also plays a significant role in regulating energy balance. Several studies have highlighted the importance of adiponectin, which is produced by fat cells and has demonstrated significant reduction in diabetes, inflammation and atherosclerosis. Lower levels of adiponectin hormone are associated with metabolic disorders such as insulin resistance, obesity, Type 2 diabetes, elevated blood pressure and cardiovascular disease. However, adiponectin levels can be increased by calorie restriction and exercise for weight loss, which in turn improves insulin resistance. This shows the importance of managing the adiponectin levels and lifestyle interventions in preventing and managing metabolic health and associated diseases (15).

PHASES OF FUEL USE DURING INTERMITTENT FASTING

Fed State (0–4 hours after eating)

The body changes from the fed state to the early fasting state within the first several hours (approximately 0–4 hours) following your previous meal. Insulin levels remain elevated during this time as it helps in the uptake of glucose into cells and the storage of excess nutrients as fat and glycogen, while circulating glucose from the most recent meal remains the primary fuel for tissues. As post-meal digestion concludes, the liver releases glucose into the bloodstream to maintain normal blood glucose levels by breaking down glycogen in response to a decreasing insulin-to-glucagon ratio. Before significant activation of fat oxidation and gluconeogenesis occurs with longer fasting periods, this reliance on glycogenolysis is typical in the early post-absorptive period (9).

Early Fasting State (4–12 hours)

At this point, the stored glucose serves as the energy fuel instead of the nutrients from the previous meal. The liver begins releasing glucose by breaking down its stored glycogen (glycogenolysis) when insulin levels fall and glucagon rises at the same time. This helps maintain normal blood glucose levels when no meal is consumed. The primary source of glucose during this period is liver glycogen; these stores gradually decrease and are mostly exhausted by 12 hours. Although glucose is still the body's main fuel at this early stage of fasting, as glycogen levels drop, the body gradually

increases the production of glucose from other sources and starts to rely more on fat breakdown, with fatty acids beginning to contribute to energy needs (10,11).

Fat Oxidation Phase (12–24 hours)

The body starts burning fat rather than carbs after 12 to 24 hours of fasting. The body relies on enhanced fat breakdown, which is stored in adipose tissue, as the stored liver glycogen is severely depleted. Fatty acids produced by adipose tissue are beta-oxidised and used by the liver and other tissues to produce energy. The body uses fat to meet its energy needs because fewer carbohydrates are consumed. This is a normal physiological response to extended fasting. The body may produce small amounts of ketones during this time and it may increase considerably in a prolonged fasting state. This metabolic shift from glucose to fat as the primary energy source is critical throughout the 12- to 24-hour fasting window.

Ketogenic Phase (24–72 hours or with repeated IF)

After approximately 24 hours of fasting, hepatic glycogen stores become largely depleted, prompting the body to rely increasingly on energy derived from adipose tissue and to a lesser extent, protein stores. This metabolic shift is driven mainly by the mobilisation of triglycerides from adipose tissue, which are broken down into free fatty acids and glycerol. In the liver, glucose is produced by glycerol via gluconeogenesis and simultaneously, ketone bodies are produced in the conversion of free fatty acids via ketogenesis. furthermore, the produced ketone bodies are released into the bloodstream and taken up by peripheral tissues, where the produced energy is converted back into acetyl-CoA to fulfil the energy needs. In the early stages of prolonged fasting, along with increased lipid metabolism, amino acid production from gluconeogenesis continues to supply glucose to tissues with inefficiency of ketone utilisation. Further prolonging fasting could significantly increase the production of ketones, which are utilised in tissues and lowers decreases glucose uptake (10,12).

BREAKFAST AND CIRCADIAN ALIGNMENT

There is evidence that the best metabolic circumstances for eating occur in the morning. In particular, the first meal of the day, early breakfast, consumed at the start of the activity phase, is a potent “zeitgeber” or initiator of peripheral clock gene activation. It improves hormones and enzymes that control hunger, muscle synthesis, total glycemia and body weight. Additionally, compared to an isocaloric dinner, the diet-induced thermogenesis is substantially stronger following a high-energy breakfast, underscoring the significance of the first meal of the day in reaching metabolic homeostasis (11).

The “lack of breakfast” or delaying breakfast until noon, also referred to as the “extended postabsorptive state, the shift from an overnight fast to a fed state has the potential to desynchronise clock gene expression and metabolism regulation, despite numerous studies demonstrating the metabolic benefits of eating breakfast early. High BMI, hyperglycemia, insulin resistance and the risk of Type 2 diabetes have all been closely linked to skipping breakfast (11).

Research comparing a high-calorie breakfast to a high-calorie dinner revealed that the breakfast group lost more weight, reduced their waist size and improved their insulin and fasting glucose levels. The benefits of early TRE are generally greater than those of delayed TRE. Eating earlier in the day has been shown to increase insulin sensitivity, muscle glucose absorption and lipid metabolism in both human and animal studies. For 12 weeks, an 8-hour eating window from 10 am to 6 pm was seen in one clinical experiment, including obese patients. There was better circadian alignment, which is believed to benefit long-term metabolic health, even though there were no significant lipid changes (15).

DISCUSSION

Recent studies suggest that time-restricted eating and 5:2 have been shown to enhance glycemic management, triglycerides and LDL cholesterol

in recent trials, although the findings were inconsistent. Alternate day fasting (ADF) and modified ADF have been shown in several trials to significantly improve blood pressure, BMI and waist circumference (5-8). These results demonstrated that intermittent fasting may be advantageous in the short term, especially for lowering body weight and waist circumference. Individuals adhere to time-restricted eating than to more restrictive patterns like the 5:2 diet or alternate-day fasting; it appears to be more effective than many of the techniques.

Although IF might help with weight control and metabolic health in the short run, there are still issues with extremely limited eating periods. Zhong et al. (2025) reported that eating for <8 hours per day was associated with a 135% higher risk of cardiovascular death and decreased muscle mass. The risk of cardiovascular death increased by 224% among people who already had cardiovascular disease or diabetes (14).

On the contrary, prolonged intermittent fasting may raise the risk of protein and micronutrient deficiencies as well as insufficient fluid intake, necessitating supplements and careful medication adjustments, including diuretics, antihypertensive drugs and SGLT-2 inhibitor (15).

CONCLUSION

Intermittent fasting facilitates shift from glucose to fat oxidation and helps in ketone production. When matched with the biological clock, especially through early restricted eating, it supports and help in maintaining the blood glucose level, improve insulin sensitivity and weight management, However, its advantages rely upon the meal timing, fasting duration and individual health status. Prolonged or overly restrictive fasting may increase the risk of lean mass loss, nutrient deficiencies and cardiovascular complications in vulnerable populations. Therefore, intermittent fasting should be individualised, with emphasis on circadian alignment, nutritional adequacy and long-term sustainability.

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NON-WEIGHT-BEARING EXERCISE FOR DIABETES MANAGEMENT

A COMPREHENSIVE GUIDE

Shreyas Katharani*

INTRODUCTION

Managing diabetes effectively requires a multifaceted approach that includes proper nutrition, medication adherence and regular physical activity. For individuals with diabetes, particularly those with complications such as diabetic neuropathy, foot ulcers or joint problems, non- weight-bearing exercises offer a safe and effective way to maintain fitness while minimizing injury risk (1).

UNDERSTANDING NON-WEIGHT-BEARING EXERCISE

Definition and Core Principles

Non-weight-bearing exercises are physical activities performed without placing the full body weight on the feet or joints. These exercises reduce stress on the lower extremities while still providing cardiovascular, muscular and metabolic benefits essential for diabetes management (2).

Non-Weight-Bearing Exercise for People with Diabetes (PWD)

Non-weight-bearing exercise is especially beneficial for PWD because it reduces the risk of injury in individuals with peripheral neuropathy by limiting pressure on insensitive areas (3). It protects joints in those with arthritis or joint-related complications by minimizing impact on the knees, hips and ankles (4). It enhances foot safety for individuals with diabetic foot conditions by lowering the risk of trauma that can lead to ulcers or infections (1). These exercises provide important cardiovascular benefits without exposing the body to high-impact stress It also support blood sugar control by improving insulin sensitivity and promoting glucose uptake through regular physical activity (5).

KEY BENEFITS FOR DIABETES MANAGEMENT

Glycemic Control Benefits (1)

Regular participation in non-weight-bearing exercise improves insulin sensitivity through enhanced glucose uptake by skeletal muscles, leading to better blood sugar regulation (2). Consistent glucose utilization contributes to reduced HbA1c levels and improved long-term diabetes control (6). Post-exercise glucose lowering occurs as muscles replenish glycogen stores, resulting in immediate blood glucose benefits (5).

Cardiovascular Health Improvements

Non-weight-bearing exercises provide significant cardiovascular benefits by improving heart function and circulation, lowering blood pressure in hypertensive individuals with diabetes, enhancing the lipid profile including HDL cholesterol and reducing the overall risk of cardiovascular disease (1).

Metabolic Advantages

These benefits include an increased metabolic rate during and after exercise, enhanced fat oxidation for effective weight management, improved preservation of muscle mass and more efficient overall energy metabolism (4).

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SPECIFIC TYPES OF NON-WEIGHT-BEARING EXERCISES

Aquatic Exercises

Swimming

It is a low-impact, full-body exercise performed for 20–45 minutes at a moderate to vigorous intensity, with precautions that include monitoring blood glucose levels before and after activity and ensuring proper foot care (2).

Water Aerobics

Water aerobics is a low-impact cardiovascular and resistance workout suitable for all fitness levels, offering social motivation in group classes, with warm water (83–88°F) being ideal for people with diabetes (4).

Water Walking/Jogging

Water walking or jogging offers a natural, low-impact gait, with progression from shallow to deeper water and water shoes recommended for people with diabetes (3).

UPPER BODY EXERCISES

Chair-Based Workouts

It includes seated arm circles for shoulder mobility and circulation, chair boxing for cardiovascular fitness and upper-body strength, resistance band exercises to strengthen arms, shoulders and core and seated rowing motions to work back muscles and improve posture (7).

Wheelchair Sports and Activities

These sports and activities include basketball for cardiovascular fitness, tennis for individual or doubles play and racing or pushing exercises to strengthen the upper body and core (4).

CYCLING VARIATIONS

Stationary Cycling

This kind of cycling offers a controlled, adjustable-intensity workout with easy blood glucose monitoring, starting at 10–15 minutes and progressing to 30–60 minutes, while recumbent bikes provide additional back support (2).

Hand-Cycling

Hand-cycling provides a cardiovascular workout for the upper body using hand cycles or arm ergometers and is especially suitable for individuals with lower limb limitations (4).

RESISTANCE TRAINING (SEATED/ SUPPORTED)

Machine-Based Exercises

Exercises such as lat pulldowns strengthen the back, chest presses build chest and arm strength and seated leg extensions and leg curls target the quadriceps and hamstrings safely.

Free Weight Exercises

Seated dumbbell exercises like bicep curls and shoulder presses, resistance band workouts for portability and versatility and medicine ball exercises for core strength and coordination offer a well-rounded strength routine (4).

EXERCISE PROGRAMMING GUIDELINES

Frequency and Duration

Aquatic exercises are recommended three to five times per week for 20–45 minutes at a moderate intensity. Upper-body resistance exercises should be performed two to three times per week for 20–30 minutes at a moderate to vigorous intensity. Stationary cycling is advised three to five times per week, with sessions lasting 20–60 minutes at a moderate intensity. Chair-based exercises can be done daily for 10–20 minutes at a light to moderate intensity, making them suitable for individuals with limited mobility or balance issues (1).

Progression Strategies

Begin with 10–15 minute sessions, gradually adding 5 minutes weekly as tolerated, increase intensity through resistance or speed and incorporate variety by rotating different exercise types.

SAFETY CONSIDERATIONS AND PRECAUTIONS

Blood Glucose Monitoring

Monitor glucose by testing 30 minutes before exercise, watching for hypoglycemia during

activity and checking again immediately after and two hours post-exercise (1).

Foot Care Guidelines

Maintain foot health by inspecting daily for cuts or irritation, wearing well-fitting supportive shoes, keeping feet dry, especially after water activities and seeing a podiatrist regularly if diabetic (3).

EXERCISE MODIFICATIONS BY COMPLICATION

For Diabetic Neuropathy

Avoid high-impact activities, focus on seated exercises and swimming, use proper padding and support and monitor for temperature sensitivity (3).

For Diabetic Retinopathy

Avoid exercise that cause sudden blood pressure spikes or inverted positions, opt for moderate-intensity activities and consult an ophthalmologist before starting new programs (1).

For Cardiovascular Disease

Begin with low-intensity exercise, progress gradually, monitor heart rate closely and consider participating in cardiac rehabilitation programs (10).

CREATING A BALANCED EXERCISE PROGRAM

Weekly Schedule Example

A sample weekly schedule could include 30 minutes of water aerobics on Monday, 25 minutes of upper-body resistance training on Tuesday, 30 minutes of stationary cycling on Wednesday, 20 minutes of chair exercises and stretching on Thursday, 35 minutes of swimming on Friday, 30 minutes of hand cycling or wheelchair sports on Saturday and 15 minutes of gentle stretching and relaxation on Sunday.

Combining Exercise Types

A balanced exercise routine combines 3–4 weekly cardiovascular sessions, 2–3 strength-training sessions, daily flexibility work and balance training as appropriate and safe.

MONITORING PROGRESS AND OUTCOMES

Key Metrics to Track

HbA1c levels should be monitored every three months, with a target of less than 7% for most adults with diabetes. Blood pressure should be checked weekly and maintained below 130/80 mmHg. Body weight and body mass index (BMI) should also be assessed weekly, with targets individualized based on clinical goals. Exercise duration should be tracked daily, aiming for a gradual increase over time as tolerance improves. Perceived exertion should be assessed during exercise, with an optimal target of 5–7 on the Rate of Perceived Exertion (RPE) scale, indicating moderate to vigorous intensity (1).

Long-term Benefits Assessment

Non-weight-bearing exercises leads to better glucose management hence better diabetes management, enhances quality of life by increasing energy and mobility, reduces the risk of diabetes-related complications and improves medication effectiveness with the potential need for fewer medications (1).

CONCLUSION

Non-weight-bearing exercises offer individuals with diabetes a safe, effective pathway to better health management. They are specifically needed for diabetics with feet problems, severe peripheral neuropathy and pain in lower limb joints. By incorporating aquatic activities, upper body exercises, cycling variations and seated resistance training, diabetics can achieve significant health benefits while minimizing injury risks. The key to success lies in consistent participation, proper monitoring and gradual progression under appropriate medical supervision.

Regular engagement in non-weight-bearing exercise programs can lead to improved glycemic control, enhanced cardiovascular health and better overall quality of life for individuals managing diabetes. With proper planning and

execution, these exercises become powerful tools in comprehensive diabetes care.

Always consult with healthcare providers before beginning any new exercise program, especially in people with diabetes or other chronic conditions of bone, joints and peripheral nerves.

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QUESTIONS AND ANSWERS

Q. What is microalbuminuria and why is it important in people with diabetes?

A. Microalbuminuria refers to a moderate increase in the excretion of albumin, a vital blood protein in the urine. It is defined by an albumin excretion rate of 30–300 mg per 24 hours, 20–200 µg/min, or an albumin-to-creatinine ratio (ACR) of 3–30 mg/mmol in a spot urine sample.

This condition arises when the kidneys begin to leak small amounts of albumin due to increased permeability of the glomerular filtration barrier, which normally prevents albumin from passing through. These ultrastructural changes within the glomerulus, in addition to filtration rate and pressure alone contribute to the development of microalbuminuria.

Clinical Relevance: Microalbuminuria is a well-established clinical marker of early kidney damage, especially in individuals with diabetes or hypertension. It signals a heightened risk for both chronic kidney disease (CKD) and cardiovascular disease (CVD).

Disease Associations and Prognostic Value: In Type 1 diabetes, microalbuminuria closely correlates with microvascular complications such as retinopathy, whereas in Type 2 diabetes, it often represents broader systemic vascular injury. Persistent microalbuminuria predicts the progression of diabetic nephropathy and is one of the strongest risk markers for advanced kidney disease. It precedes sustained declines in glomerular filtration rate (GFR) and progression to end-stage renal disease.

Additionally, microalbuminuria independently increases the risk of cardiovascular events, including coronary artery disease, heart failure, stroke and cardiovascular mortality. Even small, subclinical increase in the urinary albumin has been associated with rising hazards for CVD reinforcing its prognostic significance.

Importance in Diabetes Management:

Early Indicator of Kidney Damage: Microalbuminuria often marks the onset of diabetic nephropathy before visible symptoms or reductions in kidney function become evident. Early detection enables prompt intervention to prevent or delay progression to overt proteinuria and CKD.

Predictor of Diabetic Nephropathy and Disease Progression: It is the most reliable predictor for developing overt diabetic nephropathy in both Type 1 and Type 2 diabetes. Persistent microalbuminuria is associated with a faster decline in GFR and an increased risk of end-stage renal disease.

Marker of Cardiovascular Risk: The presence of microalbuminuria highlights patients at greater risk for cardiovascular complications, including myocardial infarction, stroke and cardiovascular mortality independent of traditional risk factors.

Indicator of Widespread Microvascular Damage: Microalbuminuria reflects not only renal injury but also generalised endothelial dysfunction and/or other microvascular complications, of diabetic retinopathy and neuropathy.

Potential for Reversal with Early Intervention: Detection of microalbuminuria signals the need for intensified blood pressure as well as glycemic control. With improvement in control of these parameters and appropriate pharmacotherapy, microalbuminuria may sometimes regress.

Prernaa Prabhakar

Q. Can omega-3 fatty acids improve outcomes in cardiovascular and diabetic kidney disease (DKD)?

A. Omega-3 fatty acid belong to the group of polyunsaturated fatty acids (PUFAs) derived primarily from fish oil. The three principal types are α-linolenic acid (ALA), eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). These are essential fatty acids and small dietary amounts are required for normal growth and physiological function. Omega-3 fatty acid is well known for their ability to reduce serum triglyceride levels. Although some findings remain controversial, regular omega-3 fatty acid supplementation has been associated with a reduced risk of myocardial infarction and sudden cardiac death. Evidence also suggests that omega-3 fatty acid may improve blood circulation, enhance fibrinolysis and lower blood pressure and heart rate.

Hypertension is a major risk factor for the development and progression of chronic kidney disease (CKD); therefore, the antihypertensive and anti-inflammatory effects of omega-3 fatty acids may contribute to slowing CKD progression. Numerous studies have investigated the benefits of omega-3 fatty acids in inflammatory, autoimmune and renal disease. Clinical evidence indicates that omega-3 fatty acids can reduce proteinuria in chronic glomerular diseases and slow disease progression in IgA nephropathy. Animal studies further support these findings, demonstrating reduced renal inflammation and fibrosis following omega-3 fatty acids supplementation. Additionally, a large community-based cohort study reported that higher dietary intake of long-chain n-3 PUFAs and fish consumption was associated with a lower prevalence of CKD.

However, results across studies remain inconsistent. While large randomized trials such as VITAL-DKD did not demonstrate significant kidney-protective effects, smaller studies suggest improvements in surrogate markers such as inflammation, pruritus and lipid profiles. These findings indicate that omega-3 fatty acid may play a supportive role, particularly in cardiovascular risk reduction among patients with diabetes, rather than serving as a primary therapy for DKD.

Jayshri Jain

RECIPES

CUCUMBER PANCAKE

INGREDIENTS

60 gm Gram flour (Besan)

50 gm Cucumber (grated)

2 cloves Garlic (chopped)

2 Green chillies (chopped)

Few Coriander leaves (finely chopped)

1 tsp Jeera powder

1 tsp Dhaniya powder

1 tsp Groundnut oil

½ Banana leaf (for shaping of the dough)

METHOD

Pre-Preparation:

Chop and grate all the vegetables as mentioned.

Preparation:

In a bowl, add all the ingredients except the oil and the banana leaf. Mix well.

On a banana leaf, add the pancake dough and shape the dough into a circle.

In a heated pan, add oil. Add the shaped dough (do not add the banana leaf). Let it cook and then flip. Serve hot.

PROVIDES 2 SERVINGS

Nutritional Information Per Serving

Energy (kcals)	Protein (grams)	Carbohydrate (grams)	Fat (grams)	GI
120	3	12	2	Low

SPECIAL FEATURES

- Fibre-rich
- Low Glycemic Index

Harshini Thakur

MATAR TOAST

INGREDIENTS

30 gm Semolina (Rava)
100 gm Peas
50 gm Onion
50 gm Capsicum
50 gm Tomato
50 gm skim Paneer
Few coriander leaves
1 tsp Chilli flakes
2 green Chilli
Salt as per taste

METHOD

Pre-preparation:

Chop onion, capsicum, tomato and potato.

Preparation:

In a grinder, add pea, coriander and green chillies (do not make it too smooth a paste).

Add Rava, salt and ½ cup water. Mix well and keep aside.

For the filling, take onions, tomatoes, capsicum, and paneer in a bowl. Add chilli flakes. Mix well.

In a sandwich griller, add the pea batter, add the filling and cover again with pea batter.

Heat the toaster for 8-10 minutes on low flame. Let it cook. Serve hot

PROVIDES 2 SERVINGS

Energy (kcals)	Protein (grams)	Carbohydrate (grams)	Fat (grams)	GI
150	8	20	2	Low

SPECIAL FEATURES

- High Protein
- Low Glycemic Index

Harshini Thakur

HOW KNOWLEDGEABLE ARE YOU?

1. Which transporter is insulin-dependent?

A. GLUT1

B. GLUT2

C. GLUT4

D. GLUT5

2. Insulin has which effect?

A. Lipolysis

B. Glycogen synthesis

C. Protein breakdown

D. Ketone formation

3. Which hormone opposes insulin action?

A. Thyroxine

B. Aldosterone

C. Glucagon

D. Calcitonin

4. Which organ is the major site for insulin clearance?

A. Kidneys

B. Pancreas

C. Liver

D. Spleen

5. Fasting plasma glucose mainly reflects:

A. Hepatic glucose output

B. Muscle glycogen

C. Intestinal absorption

D. Insulin clearance

6. Ketone bodies are formed during:

A. Hyperinsulinemia

B. Hypoinsulinemia

C. High-carb diet

D. None

7. GLUT2 is located in:

A. Brain

B. Pancreas & liver

C. Muscle

D. Adipose tissue

8. The primary fuel for the brain is:

A. Amino acids

B. Fatty acids

C. Glucose

D. Ketones

9. Insulin secretion is stimulated by:

A. Fasting

B. Exercise

C. Hyperglycemia

D. Cortisol

10. Which hormone increases after meals?

A. Insulin

B. Glucagon

C. Growth hormone

D. Epinephrine

ANSWERS:

1. C 2. B 3. C
4. C 5. A 6. B
7. B 8. C 9. C
10. A

MYTHS AND FACTS

Myth: "All obese people will eventually develop diabetes."

Fact:- Not everyone with obesity is suffering from diabetes. The excess weight might increase the risk, but it does not mean the disease will occur. Many people with obesity can still be metabolically healthy because their bodies have retained the ability to regulate blood sugar efficiently. Some individuals exhibit a favorable pattern of fat distribution, with excess adipose tissue stored predominantly in subcutaneous depots rather than accumulating around vital organs. In addition, robust and well-functioning pancreatic β-cells that continue to produce adequate insulin contribute to metabolic resilience. Collectively, these factors help explain why obesity increases the risk of metabolic disease but does not inevitably lead to adverse outcomes. Healthy eating, regular physical activity and lifestyle choices can greatly influence long-term metabolic health. Many people with obesity can still be metabolically healthy as their bodies have retained the ability to regulate blood sugar efficiently. The combination of healthy eating, regular physical activity and lifestyle can be a great determinant of long-term metabolic health. People with obesity may be metabolically healthy due to the fact that their pancreas still possesses the ability to manage blood sugar efficiently.

Prernaa Prabhakar

Myth: "Eating frequently (6 times/day) is always best for diabetes."

Fact: Eating more frequently may help some individuals with diabetes by preventing large fluctuations in blood glucose levels, especially for those who are prone to hypoglycemia or who use insulin or certain glucose-lowering medications. Smaller, more frequent meals can reduce post-meal glucose spikes, support consistent energy levels and help with appetite control in some cases.

Studies show that increasing the number of smaller, regular meals throughout the day (while keeping the total calories the same) reduces the amplitude of blood glucose swings and lowers peaks after eating in people with impaired glucose tolerance or diabetes. This helps keep blood sugar more stable overall. In healthy adults, consistent meal timing was linked with more favorable insulin responses and better metabolic patterns versus irregular eating schedules, suggesting that regular meals may support improved insulin sensitivity and lower cardiovascular risk factors.

Population data suggest that higher frequency of eating occasions is associated with lower fasting glucose levels and reduced risk of metabolic syndrome compared with very low meal frequency, although some results vary depending on the diet and lifestyle pattern.

Myth: "Alcohol Consumption leads to MASLD".

Fact: This is a myth. While alcohol can certainly damage the liver, most cases of fatty liver are known to be non-alcoholic, a condition now commonly referred to as MASLD (Metabolic Dysfunction-Associated Steatotic Liver Disease) earlier called NAFLD (Non-Alcoholic Fatty Liver Disease). MASLD develops when excess fat builds up in liver cells, usually due to worsening of metabolic risk factors, which may be independent of alcohol intake. The factors may be sedentary lifestyle, obesity of abdominal fat, diet rich in refined carbohydrate, sugar and unhealthy fats, insulin resistance or Type 2 diabetes. MASLD is highly prevalent in India, affecting around 30-40% of the general population with higher rates in urban areas and among individuals with metabolic conditions like diabetes.

Alcohol intake may also be a cause of fatty liver and leads to alcohol-related liver disease. This has a different mechanism and progression of disease compared to MASLD. Therefore, assigning the blame to alcohol alone overlooks the other possible underlying causes.

Jayshri Jain

DIABETES EDUCATOR CERTIFICATE COURSE

Dr Chandalia's DENMARC in association with Help Defeat Diabetes Trust (HDDT) presents to you a Certificate course for Diabetes Educators (CDE)!

Help Defeat Diabetes Trust (HDDT) is a registered, non-profit public trust, having amongst its many objectives, the main objective of promoting education and awareness about diabetes among people from different fields.

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- **For more details,** visit our website or email us at helpdefeatdiabetesinfo@gmail.com.

MEMBERSHIP FORM

Association of Diabetes Educators (ADE)

(For eligibility criteria: Check Website www.diabeteseducatorsindia.com)
(Kindly print, duly fill, scan and upload)

Name ……..……Age:……Gender:……

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E-mail id: ……

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Work Experience: ……...……

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Certificates attached: ……

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How do you wish to participate in the ADE activities?

- o Update my knowledge and skills
- o As a faculty in ADE's Educational Activities
- o Organizational Activities as Office Bearer

Please pay the membership fees (Rs.2000/-) through NEFT / RTGS/online to the following bank account. The details are as follows:

Account name: Association of Diabetes Educators

Account type: Savings Account

Name of the bank: Bank of India

Account number: 006610110001734

IFSC Code: BKID0000066

……..……

Signature

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