

Sri Lanka College of Endocrinologists

Clinical Practice Guideline:

Diabetes

2025



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Guideline committee:

Dimuthu Muthukuda
 Neomal De Silva
 Nipun Lakshitha De Silva
 Muditha Weerakkody
 Manilka Sumanatilleke
 Chandrika J Subasinghe
 Shani Mathara Diddhenipothage
 Dulani Kottahachchi
 Tharanga Samarasekara

Contributors:

Dayakshi Abeyaratne	Semini Gunawardane	Himansa Ranawaka
Dilini Abeyratne	Uditha Hettiarachchi	Dhanushka Rathnayaka
Sachith Abhayaratna	Anuradha Jayasuriya	Gowri M. Ratnayake
Shamila Adikari	Chathuri Jayawardena	Gajawathana Sakthilingam
Muneer Ahmad	Kushalee Jayawickreme	Tharanga Samarasekara
Gayana Amiyangoda	Dhulashiha Jegavanthan	Hashini Senanayake
Charles Antonypillai	Shaminda Kahandawa	Sumudu Seneviratne
Maulee Arambewela	Vidumini Kaluarachchi	Sivajini Shanmugalingam
Ishara Athurugiriya	Sudheera Kalupahana	Sharmika Sivageethan
Kapila Banduthilaka	Nayananjani Karunasena	Noel Somasundaram
Uditha Bulugahapitiya	Dharshini Karuppiyah	Chandrika J Subasinghe
Anagi Chamika	Prasad Katulanda	Manilka Sumanatilleke
Samanthi Cooray	Dulani Kottachi	Rameshkumar Thevarajah
Chinthana Dematapitiya	Dineesha Kumaratunga	Vithiya Umaipalan
Neomal De Silva	Harishanthi Mahendran	Athri Wanninayake
Nipun Lakshitha De Silva	Arjuna Marasinghe	Milanka Wattegama
Shani Mathara	Niranjala	Charitha Weerasinghe
Diddhenipothage	Meegodavidanage	Vindya Wellala
Vidana Gamage Dinithi	Dimuthu Muthukuda	Lavanya Wijekumar
Harsha Dissanayake	Pavithera Packiyarajah	Udai Wijethunga
Kavinga Gamage	Sivatharshya	Piyumi Wijewickrama
Tharuka Gamage	Pathmanathan	Nilukshana
Chaminda Garusinghe	Raiyees Rafiuldeen	Yogenthirathan
Sonali Gunathilake	Ishara Ranathunga	Jeewaka Zoysa

Scope and purpose of the guideline

The need for a National guideline in diabetes was recognised due to rising burden of the disease in Sri Lanka and its impact on morbidity, mortality, quality of life and health care expenditure. We emphasise the need to streamline the management of diabetes which requires commitment of multiple stakeholders and integration of several disciplines.

This clinical practice guideline addresses diagnosis and management of diabetes in adults and adolescents in the Sri Lankan context. The writing committee reviewed previously published guidelines, clinical research and related consensus statements and formulated a strategy applicable to the local setting. Guidance on diagnosis, classification, screening for and management of complications, and follow up mainly focusing on type 2 diabetes are included. Separate guidelines covering type 1 diabetes and diabetes in pregnancy will follow. Chapters on special situations and groups address the issues related to management of diabetes in special populations.

We propose a care model for provision of diabetes care in Sri Lanka, aiming to streamline primary, secondary and tertiary care services to optimise the coordinated delivery of preventive and curative services for people living with or at risk of diabetes. This document will provide guidance for clinicians caring for people living with diabetes at all levels of health care services encompassing primary to tertiary care levels.

We have aimed to develop a comprehensive guidance while maintaining simplicity through a reader-friendly format. This document is intended to be an easy-to-use reference for day-to-day practice. The Sri Lanka College of Endocrinologists will be duty-bound in spearheading the National Diabetes Care Plan along with multifaceted stakeholders.

Sri Lanka College of Endocrinologists

Abbreviations

ALT	-	alanine transaminase
ACR	-	albumin creatinine ratio
ACEi	-	angiotensin converting enzyme inhibitor
ARB	-	angiotensin II receptor blocker
ART	-	anti-retroviral therapy
AST	-	aspartate transaminase
BBR	-	basal bolus regimen
BG	-	blood glucose
BP	-	blood pressure
BMI	-	body mass index
CBG	-	capillary blood glucose
CGM	-	continuous glucose monitoring
DENO	-	diabetes educator nursing officer
DPN	-	diabetic peripheral neuropathy
DSMES	-	diabetes self-management education and support
DSPN	-	diabetic distal sensory motor polyneuropathy
DKA	-	diabetic ketoacidosis
DME	-	diabetic macular oedema
DR	-	diabetic retinopathy
DPP-4i	-	dipeptidyl peptidase -4 inhibitor
ECG	-	electrocardiogram
eGFR	-	estimated glomerular filtration rate
FPG	-	fasting plasma glucose
FRII	-	fixed rate insulin infusion
GLP-1 RA	-	glucagon-like peptide-1 receptor agonist
HFpEF	-	heart failure with preserved ejection fraction
HFrEF	-	heart failure with reduced ejection fraction
HDL	-	high density lipoprotein
HHS	-	hyperglycaemic hyperosmolar state
IAH	-	impaired awareness of hypoglycaemia
IM	-	intramuscular
IV	-	intravenous
LVEF	-	left ventricular ejection fraction
LOPS	-	loss of protective sensation
LDL	-	low density lipoprotein
MNT	-	medical nutrition therapy
NPH	-	neutral protamine Hagerdon
NPDR	-	non-proliferative diabetic retinopathy
OGTT	-	oral glucose tolerance test
PDR	-	proliferative diabetic retinopathy
RAAS	-	renin-angiotensin-aldosterone system
SMBG	-	self-monitoring of blood glucose
SGLT2i	-	sodium-glucose cotransporter-2 inhibitor
SIHG	-	steroid induced hyperglycaemia
TSH	-	thyroid-stimulating hormone
TDD	-	total daily dose
T1D	-	type 1 diabetes
T2D	-	type 2 diabetes
OSA	-	obstructive sleep apnoea
PAD	-	peripheral arterial disease
PPPG	-	postprandial plasma glucose
UACR	-	urine albumin/ creatinine ratio
VRII	-	variable rate insulin infusion
VPT	-	vibration perception threshold

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1. INTRODUCTION

Diabetes is a complex metabolic disease characterised by chronic hyperglycaemia. It results from complex interplay between genetic and environmental factors that cause absolute or relative insulin deficiency with or without insulin resistance. Many other pathogenic mechanisms contribute to the disease causation.

Longstanding hyperglycaemia and associated alterations in metabolic, immune and inflammatory pathways result in microvascular damage and acceleration of atherosclerosis, adversely affecting the structure and function of any organ.

1. What is the global prevalence of diabetes?

- The IDF Diabetes Atlas for 2025 estimates that 589 million adults (20-79 years) are living with diabetes worldwide. This number is projected to increase to 853 million by 2050.
- Over 4 in 5 adults (81%) with diabetes live in low- and middle-income countries.

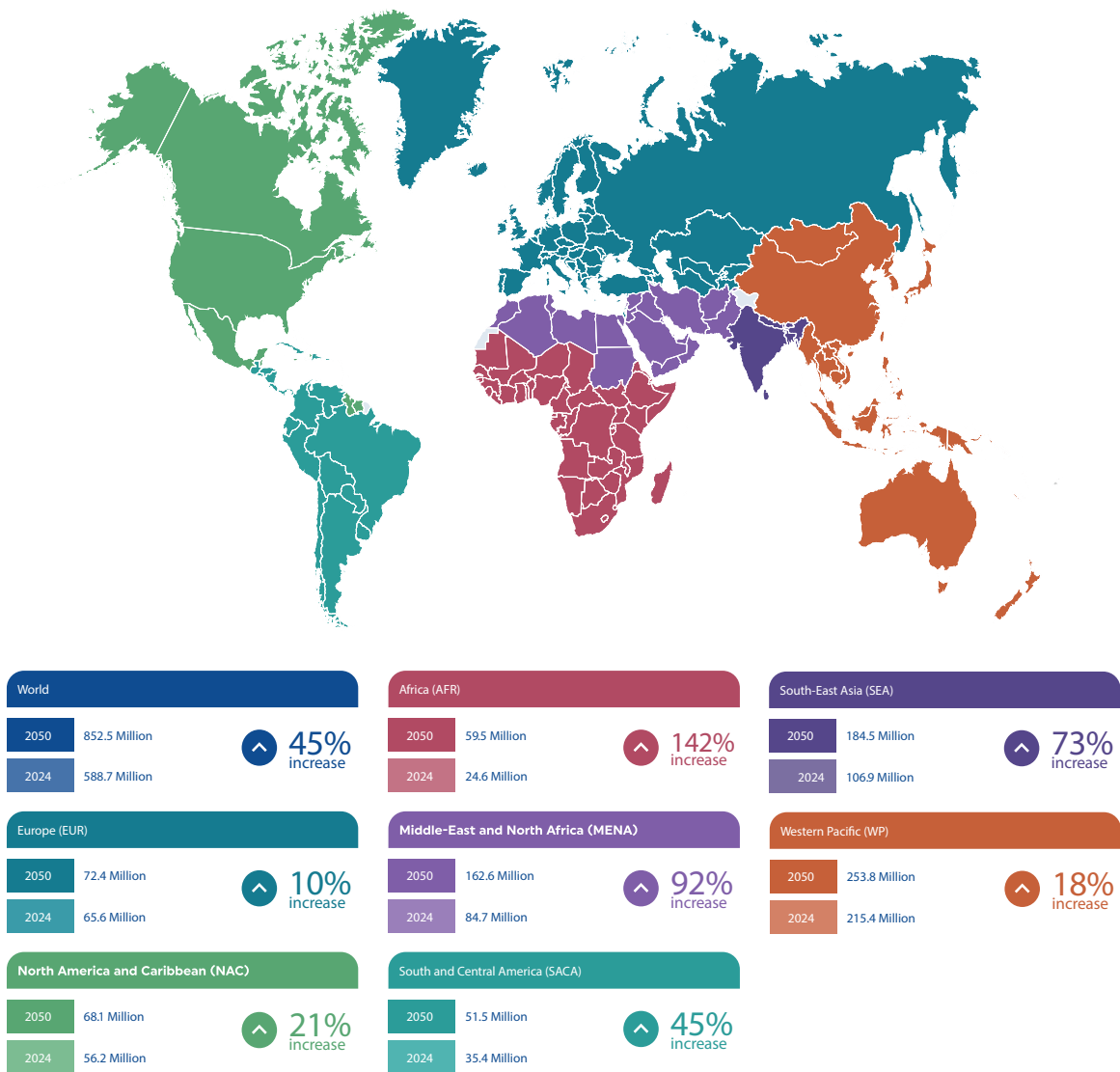


Figure 1.1 Global impact of diabetes and future projections

Source: International Diabetes Federation. IDF Diabetes Atlas 11th edition, 2025.

2. What is the prevalence of diabetes among Sri Lankans?

- 1.6 million Sri Lankan adults (20-79 years) are estimated to have diabetes in 2025. This number is projected to increase to 1.9 million by 2050.
- Prevalence has gradually increased over years (table 1.1).
- It is higher in urban than rural populations.

Table 1.1 Prevalence of diabetes and prediabetes in Sri Lankan adults

Study, year	Setting	Prediabetes (%)			Diabetes (%)		
		Urban	Rural	Total	Urban	Rural	Total
Dassanayake et al., 2007	Ragama (n = 2985)				23.8		
Katulanda et al., 2008	Islandwide (excluding Northern and Eastern provinces) (n = 4532)			11.5	16.4	8.7	10.3
Somasundaram et al., 2014	Colombo (n = 463)	30.3			27.6		
Sri Lanka health and ageing survey, 2018/2019	Islandwide (n = 6665)			30.5			23
STEPS survey by Ministry of Health, 2021	Islandwide (n = 5089)						14.6

3. What is the morbidity resulting from diabetes among Sri Lankans?

- Microvascular complications affect up to half of Sri Lankan adults with diabetes.
- Atherosclerotic cardiovascular disease is the major macrovascular complication among adults with diabetes

Table 1.2 Prevalence of complications among individuals with diabetes in Sri Lanka*

Study and year	Setting	Microvascular (%)			Macrovascular (%)		
		Retino	Nephro	Neuro	IHD	Stroke/TIA	PVD
Amarasinghe et al., 2007	Hospital	20	22.8	5.2	12.4		
Katulanda et al., 2008	Community (n = 528)	27.4		24			
Balasuriya et al., 2012	Diabetes centre	25.7	20.4	28.4	5.6		0.5
Sujanitha et al., 2015	Hospital (n = 3000)	12	39.5	34.1	21.1	3.9	
Arambewala et al., 2016	Hospital	26.1	50.8	62.6	10.6	1.1	4.7
Nuwarapaksha et al., 2021	Hospital (n = 309)	50.8	11.3	58.9	28.8	10.4	27.5
De Silva et al., 2022	Hospital (n = 400)	52.9	27.9	55.4	40.2	5	2.3

*The wide heterogeneity in reported prevalence data are likely due to differences in methods used to diagnose the complications, study settings and study participant characteristics.

IHD-ischaemic heart disease, Nephro-nephropathy, Neuro-neuropathy, PVD-peripheral vascular disease, Retino-retinopathy, TIA-transient ischaemic attack

4. What is the mortality resulting from diabetes among Sri Lankans?

- Diabetes is the third leading cause of death in Sri Lankan adults .
- It is also a major risk factor for stroke and ischaemic heart disease, the two leading causes of death.

Table 1.3 Top 10 causes of death and disability based on (DALYs) in Sri Lanka (2019 and 2021)

Causes of death and disability	Rank - 2019	Rank - 2021
Ischemic heart disease	1	2
Stroke	2	1
Diabetes	3	3
Asthma	4	
Chronic kidney disease	5	
COPD	6	6
COVID 19		4
Neonatal disorders		5

Source: Institute for Health Metrics Evaluation. Used with permission. All rights reserved.

References

1. DeFronzo RA. From the triumvirate to the ominous octet: a new paradigm for the treatment of type 2 diabetes mellitus. *Diabetes*. 2009;58(4):773-795.
2. IDF diabetes Atlas: Global estimates of undiagnosed diabetes in adults for 2025.
3. Katulanda P, Constantine GR, Mahesh JG, et al. Prevalence and projections of diabetes and pre-diabetes in adults in Sri Lanka--Sri Lanka. *Diabetes, Cardiovascular Study (SLDCS)*. *Diabet Med*. 2008;25(9):1062-9.
4. Somasundaram, N. P., Ranathunga, I., Gunawardana, K., & Ediriweera, D. S. (2019). High prevalence of diabetes mellitus in Sri Lankan urban population – data from Colombo urban study. *Sri Lanka Journal of Diabetes Endocrinology and Metabolism*, 9(2), 8-15. <https://doi.org/10.4038/sjdem.v9i2.7393>
5. Vos T, Lim SS, Abbafati C, et al. Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. *The Lancet*. 2020;396(10258):1204-1222.
6. Katulanda P, Ranasinghe P, Jayawardena R. Prevalence of retinopathy among adults with self-reported diabetes mellitus: the Sri Lanka diabetes and Cardiovascular Study. *BMC Ophthalmol*. 2014;14:100.
7. Amarasinghe DACL, Fonseka P, Fernando DJS, Dalpatadu KCS. Risk factors for long-term complications in patients with type 2 diabetes, in Sri Lanka. *J Coll Community Phys Sri Lanka*. 2004;9(1):8.
8. Balasuriya BMAC, Sumanatilleke MR, Jayasekera TI, Wijesuriya MA, Somasundaram NP. Prevalence of micro and macrovascular complications of diabetes detected at single visit screening. *Sri Lanka J Diabetes Endocrinol Metab*. 2012;2(1):17-21.
9. Sujanitha V, Sivansuthan S, Selvakaran P, Parameshwaran R. Overweight, obesity and chronic complications of diabetes mellitus in patients attending Diabetic Centre, Teaching Hospital, Jaffna, Sri Lanka. *Ceylon Med J*. 2015;60(3):94.
10. Arambewela MH, Somasundaram NP, Ranjan Jayasekara HBP, et al. Prevalence of chronic complications, their risk factors, and the cardiovascular risk factors among patients with type 2 diabetes attending the diabetic clinic at a tertiary care hospital in sri lanka. *Journal of Diabetes Research*. 2018;2018:1-10.
11. STEPS survey Sri Lanka 2021, Ministry of Health, Sri Lanka
12. Rannan-Eliya RP, Dissanayake VH, Perera P, Perera B, Herath HM, Wijemunige N, Dalpatadu S, Samarage S, Gamage A, Jayatissa R, Fernando EY. Cohort profile: the Sri Lanka Health and Ageing Study (SLHAS). *International Journal of Epidemiology*. 2024 Apr 1;53(2):dyae044.
13. Nuwarapaksha N, Nandasena R, Nanayakkara N. IDF21-0413 Prevalence of Long-term Complications among Type 2 Diabetes Mellitus Patients at National Hospital–Kandy, Sri Lanka. *Diabetes Research and Clinical Practice*. 2022 Apr 1;186.
14. De Silva P, Kodithuwakku G, Imbulgoda N, Samarawikkrama MD, Palagasingha D, Nanayakkara S, Weeraratna TP. Indications for hospital admission, prevalence of vascular and non-vascular complications and comorbidities among patients with diabetes mellitus admitted to a medical unit in a tertiary care hospital, Sri Lanka. *Asian Journal of Internal Medicine*. 2022 Aug 16;1(2).

2. CLASSIFICATION OF DIABETES

Accurate identification of type of diabetes guides clinical decisions, provides basis for epidemiological studies and allows research related to pathogenesis.

1. What are the types of diabetes?

- Type 1 diabetes** (due to autoimmune β -cell destruction, usually leading to absolute insulin deficiency, including latent autoimmune diabetes in adults).
 - Type 2 diabetes** (due to a non-autoimmune progressive loss of adequate β -cell insulin secretion, frequently on the background of insulin resistance). Occasionally, people with type 2 diabetes may present with diabetic ketoacidosis, who may present with ketosis- prone type 2 diabeteses.)
 - Specific types of diabetes** (due to other causes, e.g., monogenic diabetes syndromes, diseases of the exocrine pancreas, and drug- or chemical-induced diabeteses.)
 - Gestational diabetes mellitus** (diabetes diagnosed in the second or third trimester of pregnancy, that was not clearly overt diabetes prior to gestation or other types of diabetes occurring throughout pregnancy, such as type 1 diabetes).

Source: *Diagnosis and Classification of Diabetes: Standards of Care in Diabetes—2025*. https://diabetesjournals.org/care/article/48/Supplement_1/S27/157566/2-Diagnosis-and-Classification-of-Diabetes

Note:
The term **Type 5 diabetes** was introduced during the congress of the International Diabetes Federation in April 2025. Patients with Type 5 diabetes exhibit lean BMI (less than 18.5/m²) and are diagnosed as young adults, never experienced ketosis, originate from low socioeconomic backgrounds, suffer persistent micronutrient deficiency, possess a structurally normal pancreas, lack a family history of diabetes mellitus, and maintain glyceamic control with oral antidiabetic drugs.

2. What are the features helpful in differentiating type 1 (T1D) and type 2 diabetes (T2D)?

Table 2.1 Clinical and biochemical characteristics useful in differentiating type 1 and type 2 diabetes.

Type 1	Type 2
Younger age of onset (usually <35 years)	Older age of onset (usually post - pubertal)
Autoimmunity (personal or family history of autoimmune disease or polyglandular autoimmune syndromes)	Strong family history of T2D
Lower body mass index (BMI <25kg/m ²)	Overweight or obesity
	Acanthosis nigricans
Increased risk of diabetic ketoacidosis	
Low C -peptide	Elevated or normal C - peptide
Presence of autoantibodies - Antibodies to glutamic acid decarboxylase (GAD -65), islet cells, insulin, tyrosine phosphatases islet antigen 2(IA -2) and IA -2b, and Zinc transporter 8(ZnT8).	Undetectable autoantibodies

- If beta cell antibodies are not available, use C-peptide levels.
Accuracy is greatest 3 years from the diagnosis.
- Gold standard is stimulated C-peptide levels after mixed meal tolerance test or glucagon stimulation test.
(T1D likely if <0.6 nmol/L).
- Unstimulated fasting C-peptide levels can be used, if arranging stimulated levels are practically difficult (T1D likely if <0.2 nmol/L)

3. How to classify monogenic diabetes?

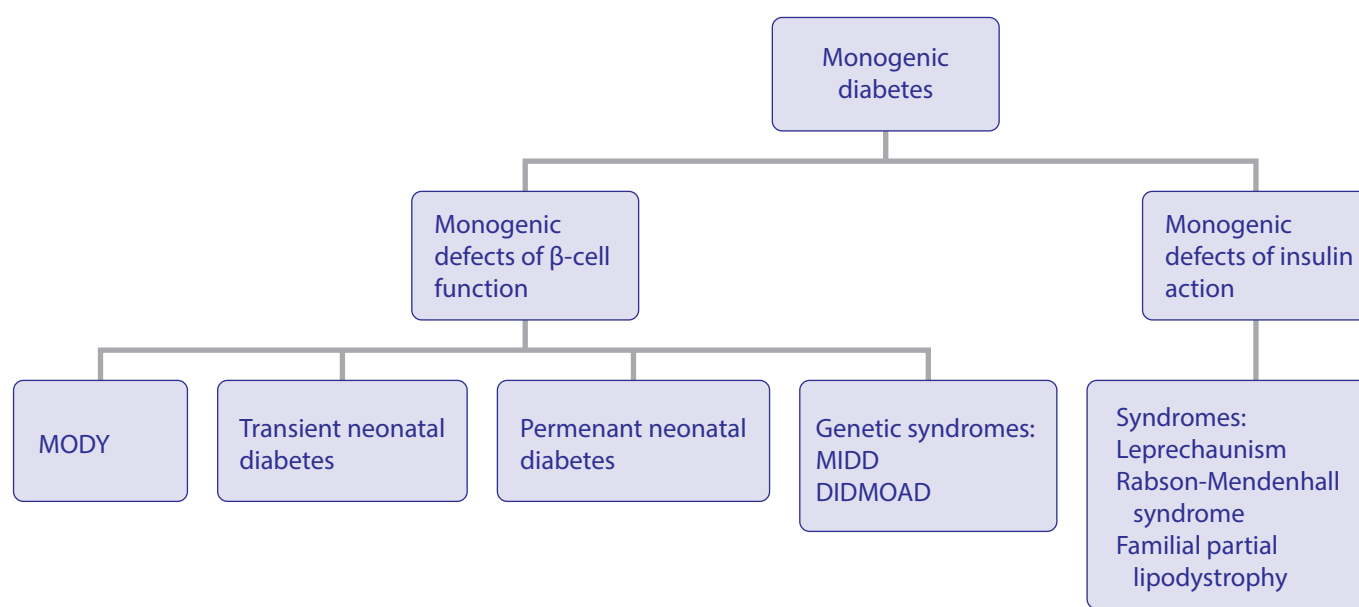


Figure 2.1 Classification of monogenic diabetes

MODY- maturity onset diabetes of the young, MIDD- maternally inherited diabetes and deafness, DIDMOAD- diabetes insipidus, diabetes mellitus, optic atrophy, deafness, GCK- glucokinase, FPG- fasting plasma glucose, HNF- hepatocyte nuclear factor

4. When should you consider MODY?

- MODY occurs at an early age (classically before the age of 25 years, although diagnosis may occur at older ages).
- Diabetes without typical features of type 1 or type 2 diabetes.
- Family history of diabetes in successive generations (suggestive of an autosomal dominant pattern of inheritance).

5. What are the most common types of MODY?

Table 2.2 Most common types of MODY

Gene	Inheritance	Clinical features
<i>HNF1A</i>	AD	Large rise in 2-h PG level on OGTT (>90 mg/dL); low hs - CRP; sensitive to sulfonylureas.
<i>HNF4A</i>	AD	May have large birth weight (macrosomia) and transient neonatal hypoglycemia; sensitive to sulfonylureas
<i>HNF1B</i>	AD	Developmental renal disease (typically cystic); genitourinary abnormalities; atrophy of the pancreas; hyperuricemia; gout.
<i>GCK</i>	AD	Stable, non-progressive elevated fasting blood glucose; typically, does not require treatment; microvascular complications are rare.

AD- Autosomal Dominant

6. What are the drugs known to cause diabetes?

- Glucocorticoids
- Thiazide diuretics
- Some HIV medications
- Second generation antipsychotic medications
- Statins
- Alpha adrenergic agonists
- Beta adrenergic agonists
- Immune check point inhibitors
- Interferon-alpha
- Pentamidine

7. What are the endocrinopathies known to cause diabetes?

- Acromegaly
- Cushing syndrome
- Thyrotoxicosis
- Glucagonoma
- Pheochromocytoma
- Somatostatinoma

References

1. Classification of diabetes mellitus. Geneva: World Health Organization; 2019. License: CC BY-NC-SA 3.0 IGO.
2. American Diabetes Association, Classification and diagnosis of diabetes: Standards of Medical Care in Diabetes—2025. *Diabetes Care*, 2025. 48(Supplement 1): p. S27-S49.
3. Levitt Katz L. E. (2015). C-Peptide and 24-Hour Urinary C-Peptide as Markers to Help Classify Types of Childhood Diabetes. *Hormone research in paediatrics*, 84(1), 62–64. <https://doi.org/10.1159/000430094>

3. SCREENING AND DIAGNOSIS OF DIABETES

Diabetes has a long asymptomatic period. It can be easily detected through early screening and is associated with improved patient outcome. Therefore, every opportunity should be utilized to screen for diabetes.

1. Who should be screened for diabetes?

- Adults with overweight or obesity (BMI ≥ 23 kg/m²) who have one or more of the following risk factors.
 - First-degree relative with diabetes
 - History of cardiovascular disease
 - Hypertension ($>130/80$ mmHg or on therapy for hypertension)
 - HDL cholesterol level < 35 mg/dL (< 0.9 mmol/L) and/ or triglyceride level >250 mg/dL (>2.8 mmol/L)
 - Individuals with polycystic ovary syndrome
 - Physical inactivity
 - Other clinical conditions associated with insulin resistance (e.g., severe obesity, acanthosis nigricans, and metabolic dysfunction–associated steatotic liver disease)
- People with prediabetes (A1C $>5.7\%$ [>39 mmol/ mol], IGT, or IFG) should be tested yearly.
- People who were diagnosed with GDM should have testing at least every 1–3 years.
- For all other people, testing should begin at age 35 years.
- Individuals in other high-risk groups (e.g., people with HIV, exposure to high-risk medicines, evidence of periodontal disease, and history of pancreatitis) should also be closely monitored.

2. How to screen for diabetes?

- Employ the following methods to screen
 - Opportunistic screening - whenever an individual seeks healthcare.
 - Passive screening - facilitate those who seek screening.
 - Active screening - systematic population screening.

Use any of the following depending on the feasibility, availability, cost, and preference.

- Fasting plasma glucose
- Oral glucose tolerance test
- HbA1c

3. In what settings should screening be performed?

Perform screening in any healthcare setting,

- Curative/ preventive
- Government/ private
- Emergency/ non-emergency
- Medical/ surgical/ gynaecological/ dental etc.
- In-patient/ out-patient
- Health campaigns
- Any other applicable setting

4. How to interpret the above test results?

Table 3.1 Diagnostic criteria for diabetes and prediabetes

Diagnostic test	Prediabetes	Diabetes
Fasting plasma glucose*	100 mg/dL (5.6 mmol/L) to 125 mg/dL (6.9 mmol/L) (IFG)	≥ 126 mg/dL (≥ 7.0 mmol/L)
HbA1c**	5.7 – 6.4% (39–47 mmol/mol)	≥ 6.5% (≥ 48 mmol/mol)
2-hour plasma glucose during OGTT ***	140 mg/dL (7.8 mmol/L) to 199 mg/dL (11.0 mmol/L) (IGT)	≥ 200 mg/dL (≥ 11.1 mmol/L)
Random plasma glucose		≥ 200 mg/dL (≥ 11.1 mmol/L)

DCCT - Diabetes Control and Complications Trial; FPG - fasting plasma glucose; IFG-impaired fasting glucose; IGT- impaired glucose tolerance, OGTT - oral glucose tolerance test; WHO - World Health Organization

* No calorie intake for at least 8 hours

**The test should be performed in a laboratory using a method that is NGSP certified and standardized to the DCCT assay.

***Use 75 g anhydrous glucose (glucose monohydrate BP 82.5g) dissolved in 250 ml water. People should follow a mixed eating pattern with at least 150 g of carbohydrates during the 3 days prior to OGTT.

5. How to confirm the diagnosis of diabetes?

- Classic symptoms of hyperglycaemia or hyperglycaemic crisis
 - Any single diagnostic test confirms the diagnosis.
 - Additionally, random plasma glucose ≥ 200 mg/dL (11.1 mmol/L), confirms the diagnosis of diabetes.
- Asymptomatic
 - Requires two abnormal test results from the same sample or in two separate test samples.
 - If using two separate test samples, the second test, may either be a repeat of the initial test or a different test.
 - Repeat the test that has a result above the diagnostic cutoff point if a patient has discordant results from two different tests.

Table 3.2 Recommended strategies according to results of screening tests for diabetes

Result	Retesting interval	Interventions	Notes
Normal	3 years; Earlier if additional risk factors appear	Behaviour modification therapy	Consider another screening test in selected individuals with higher risk
Prediabetes	1 year	Arrange lifestyle modification ± pharmacotherapy	Refer to prediabetes section
Diabetes	Immediately to confirm	Manage as newly diagnosed diabetes	Refer to sections on diabetes management

6. What are the considerations related to the use and interpretation of laboratory measurements of glucose and HbA1c?

Table 3.3 Considerations related to the use and interpretation of laboratory measurements of glucose and HbA1c

	Glucose	HbA1c
Cost	Inexpensive and available in most laboratories	More expensive and not widely available
Fasting	Fasting or timed samples required	Non-fasting test; no preparation is needed
Pre analytic stability	Poor	Good
Reflection of glycaemic variability	High	Low
Assay standardization	Not standardized	Need of the NGSP certified assay
Time frame of hyperglycaemia	Acute measure	Chronic measure of glucose exposure over the past 2 – 3 months
Acute factors that can affect levels	Food intake, stress, recent illness and activity	Unaffected by recent food intake, stress, illness and activity

NGSP- National Glycohaemoglobin Standardisation Program

Source: *Diagnosis and Classification of Diabetes: Standards of Care in Diabetes—2025*. https://diabetesjournals.org/care/article/48/Supplement_1/S27/157566/2-Diagnosis-and-Classification-of-Diabetes

7. What are the conditions that may impact haemoglobin glycation independent of glycemia?

Table 3.4 Effect of non-glycaemia related factors on HbA1c

Mechanisms	Increased HbA1c	Decreased HbA1c
Erythropoiesis	Decreased erythropoiesis due to iron, vitamin B12 or folate deficiency	Treatment with erythropoietin, iron, vitamin B12 or folate Recent acute blood loss Chronic liver disease
Glycation	Alcoholism Chronic renal failure	Vitamin C Vitamin E
Erythrocyte destruction	Increased erythrocyte life span: splenectomy	Decreased erythrocyte life span: - recent blood transfusion - haemolytic anaemia - haemoglobinopathies - splenomegaly Drugs: anti-retroviral, ribavirin and dapsone
Assay interference	Hyperbilirubinaemia Carbamylated haemoglobin Alcoholism Large doses of aspirin Chronic opiate use	Hypertriglyceridaemia

References

1. American Diabetes Association, Classification and diagnosis of diabetes: Standards of Medical Care in Diabetes—2025. *Diabetes Care*, 2025. 48(Supplement 1): p. S27-S49.,
2. World Health Organization and International Diabetes Federation. Definition and diagnosis of diabetes mellitus and intermediate hyperglycemia: report of a WHO/IDF consultation. World Health Organization: Geneva, 2006.
3. Gallagher EJ, Bloomgarden ZT, Le Roith D. Review of hemoglobin A1c in the management of diabetes. *Journal of Diabetes*. 2009;1:9–17.

4. PREDIABETES: DIAGNOSIS AND MANAGEMENT

1. What is prediabetes?

Prediabetes is the term used for individuals whose glucose or HbA1c levels do not meet the criteria for diabetes, yet have abnormal carbohydrate metabolism that results in elevated glucose levels (dysglycemia) intermediate between normoglycemia and diabetes.

2. What are the diagnostic criteria of prediabetes?

Table 4.1 Diagnostic criteria of prediabetes

Test	Result	Interpretation
Fasting plasma glucose	100 mg/dL (5.6 mmol/L) to 125 mg/dL (6.9 mmol/L)	Impaired fasting glucose (IFG)
2 - hour 75 g oral glucose tolerance test	140 mg/dL (7.8 mmol/L) to 199 mg/dL (11.0 mmol/L)	Impaired glucose tolerance (IGT)
HbA1c	5.7 – 6.4% (39 – 47 mmol/mol)	Prediabetes

3. Why it is important to treat prediabetes?

- Prediabetes is associated with obesity (especially abdominal or visceral obesity), dyslipidemia with high triglycerides and/ or low HDL cholesterol, and hypertension.
- Prediabetes increases the risk of microvascular and macrovascular complications.
- Those with an HbA1c between 5.5% and 6.0% had a substantially increased risk of diabetes (5 year incidence from 9% to 25%).

4. How should a patient with prediabetes be managed?

- Metabolic risk factor modification
 - Lifestyle – Medical nutrition therapy (reduced calorie diet), physical activity (moderate intensity physical activity 150 minutes per week), cessation of smoking, avoidance of excess alcohol intake, stress management.
 - Cardiovascular risk reduction – Control blood pressure and dyslipidaemia
 - 7-10% weight loss if overweight or obese (of initial body weight)
 - Sleep health - Prioritize adequate and quality sleep as part of lifestyle modification

b) Pharmacotherapy

Consider metformin for people with prediabetes if they are:

- Overweight or obese
- Young (age < 60 years)
- Having a past history of gestational diabetes

Measures for weight reduction

In people with overweight / obesity, 7-10% weight loss and maintenance are required to improve metabolic health.

Following interventions are useful for weight reduction:

- Medical nutrition therapy
- Physical activity (refer to section 9)
- Pharmacotherapy
- Metabolic surgery (prediabetes itself is not an indication)

References

1. Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Report of the expert committee on the diagnosis and classification of diabetes mellitus. *Diabetes Care* 2003;26(Suppl.1):S5–S20
2. Meijnikman AS, De Block CEM, Dirinck E, et al. Real-world performance of an OGTT in screening for diabetes and prediabetes in a high-risk adult Caucasian population. *Int J Obes* 2017;41:611–616
3. Knowler WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 2002;346:393–403
4. Herman WH, Edelstein SL, Ratner RE, et al. The 10-year cost-effectiveness of lifestyle intervention or metformin for diabetes prevention: an intent-to-treat analysis of the DPP/DPPOS. *Diabetes Care* 2012;35:723–730
5. Eddy DM, Schlessinger L, Kahn R. Clinical outcomes and cost-effectiveness of strategies for managing people at high risk for diabetes. *Ann Intern Med* 2005;143:251–264
6. Zhuo X, Zhang P, Selvin E, et al. Alternative HbA1c-based definitions of prediabetes: impact on prevalence and risk of progression to diabetes. *Diabetes Care* 2021;44:254–260
7. Herman WH, Ye W, Griffin SJ, et al. Early detection and treatment of type 2 diabetes reduce cardiovascular morbidity and mortality: a simulation of the results of the Anglo-Danish-Dutch Study of Intensive Treatment in People with Screen Detected Diabetes in Primary Care (ADDITION-Europe). *Diabetes Care* 2015;38:1449–1455

5. PATIENT-CENTRED DIABETES CARE

1. What are the principles of patient-centered diabetes care?

- Shared decision making - individual preferences and values, prognosis and comorbidities, social determinants and financial considerations.
- Interprofessional team-based care - consisting of primary care and specialty clinicians, diabetes educators, dietitians, pharmacists and behavioural health professionals.
- DSMES (Diabetes Self-Management Education and Support)
- Social determinants of health – including access to health care, financial constraints, cultural and language barriers

A. Diabetes self-management education and support

2. What is DSMES?

Ongoing process of facilitating the knowledge, skills and ability necessary for diabetes selfcare activities that assist in implementing and maintaining the behaviours needed to manage diabetes.

3. What are the benefits of DSMES?

It supports,

- informed decision-making
- selfcare behaviour
- problem-solving
- active collaboration with the health care team

It achieves improved,

- clinical outcomes
- health status
- wellbeing

in a cost-effective manner

4. Who should deliver DSMES?

- Diabetes educator
- Diabetes Educator Nursing Officer (DENO)
- Support groups
- Medical professionals - medical officers/ postgraduate trainees/ specialists

5. When should DSMES be delivered/ reviewed?

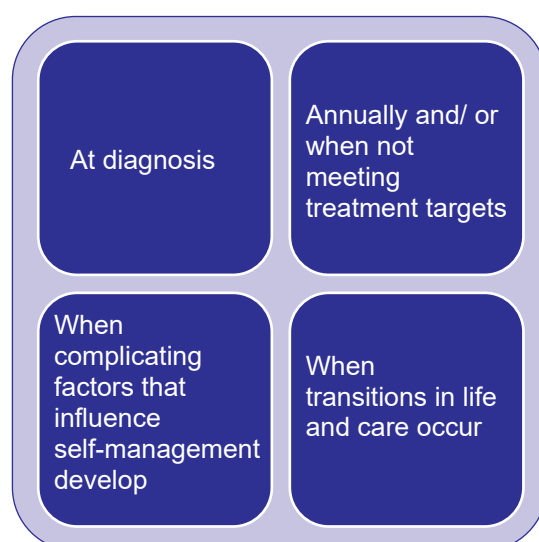


Figure 5.1 Four critical times to deliver/ review DSMES

6. What should be addressed in DSMES?

- General information
 - Chronicity of the disease
 - Importance of proper follow up
 - Goals of treatment
 - Comorbidities
- Prevention and detection of complications
 - Foot care
 - Hypoglycaemia
- Pharmacological therapy
- Non-pharmacological therapy
 - Medical nutrition therapy (MNT) (refer to section 9)
 - Physical activity
- Other behavioural changes
 - Smoking cessation
 - Avoidance of excess alcohol intake
- Reproductive health
 - Family planning and preconceptional counselling

7. Evidence for the Benefits DSMES?

- Improved diabetes knowledge and self-care behaviours, lower A1C, lower self-reported weight, improved quality of life, reduced all cause mortality risk, positive coping behaviours, and lower health care costs.
- Increased use of primary care and preventive services and less frequent use of acute care and inpatient hospital services.
- Better outcomes were reported for DSMES interventions that were more than ten hours over the course of 6–12 months , included ongoing support , were culturally and age appropriate, were tailored to individual needs and preferences, addressed psychosocial issues, and incorporated behavioural strategies .
- Individual and group approaches are effective, with a slight benefit for those who engage in both.

8. What are the technology-enabled diabetes self-management solutions?

- Continuous Glucose Monitors (CGM)
- Automated Insulin Delivery (AID)

(Improve HbA1c most effectively when there is two-way communication between the person with diabetes and the health care team, individualized feedback, use of person-generated health data, and education)

9. How to incorporate these concepts to provide patient-centred care?

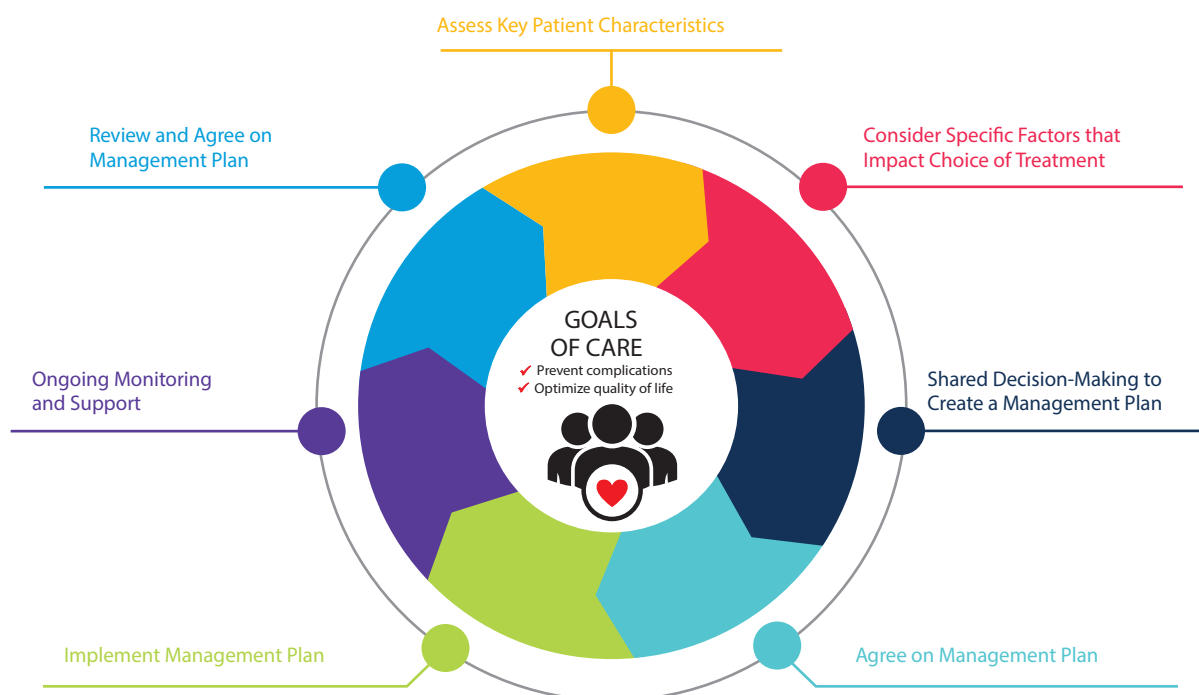


Figure 5.2 Decision cycle for patient-centred glycaemic management in diabetes

Source: 'Management of Hyperglycemia in Type 2 Diabetes, 2018. A Consensus Report by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD)' by Malanie J Davies, David A D'Alessio, Judith Fradkin et al. *Diabetes Care*. 2018.;41(12):2669–2701 © 2018 American Diabetes Association and European Association for the Study of Diabetes

B. Promoting psychological well-being

Recognising mental health issues and providing support to improve psychological well-being is important to obtain active participation of the patient in management decisions.

10. What are the psychological concerns associated with diabetes and their impact in outcome?

Table 5.1 Psychosocial concerns and their association with diabetes-related outcomes in adults with type 1 diabetes

	Increased HbA1c	Increased blood pressure	Increased cholesterol	Increased macrovascular complications	Increased microvascular complications	Decreased selfcare behaviors	Comorbid psychosocial concerns	Decreased quality of life	Increased mortality
Diabetes distress	+++	?	+	+++	+++	+++	+++	+++	?
Depression and depressive symptoms	+++	?	+++	+++	+++	+++	+++	+++	+++
Anxiety	+++	?	?	?	?	+++	+++	+++	?
Disordered eating behaviors (insulin omission)	+++	?	?	?	+++	+++	+++	+++	+++
Serious mental illness (schizophrenia, personality disorders)	+++	?	+	+++	+++	?	+++	?	+++
Cognitive impairment	+++	+++	+++	+++	+++	?	+++	?	+++

+++Strong evidence, +limited evidence, ?no data available

Source: *Facilitating Positive Health Behaviors and Well-being to Improve Health Outcomes: Standards of Care in Diabetes—2025*

11. What is diabetes distress?

- The emotional response to,
 - living with diabetes
 - burden of self-management
 - long-term complications

12. What is diabetes burnout?

- A state of physical and emotional exhaustion due to continuous diabetes distress
- Manifests as non-compliance, demotivation and disengagement in disease management (selfcare, diet and lifestyle).

13. How to identify and manage diabetes distress in an individual with diabetes?

When to suspect?

- Suboptimal glycaemic control (normal HbA1c does not exclude diabetes distress)
- Defaulting clinic visits
- Reduced engagement in selfcare tasks (SMBG, medication adherence)
- Ineffective coping strategies (e.g., emotional eating)
- Multiple negative life stressors - financial/ employment related
- Impaired relationship with healthcare professionals/ family/ friends
- Being passive or aggressive during consultation

How to screen?

- Direct verbal inquiry for features
- Using standard tools,
 - PAID scale
 - Diabetes distress scale

How to manage?

- Ask and assess the feelings/ concerns using open ended questions
- Advise on the consequences of diabetes distress, acknowledge their efforts, reduce negative emotions
- Assist and assign appropriate support- diabetes educator (DENO), psychologist, support groups
- Revise the current management
- Monitor closely and follow up

Figure 5.3 Assessment and management of diabetes distress

PAID scale- Problem Areas In Diabetes scale, SMBG- Self-Monitoring of Blood Glucose

14. How to assess psychosocial well-being in people with diabetes?

Screen during initial and annual assessment and in following circumstances;

- Hospitalisation
- With new onset of complications
- During significant transitions in care such as from paediatric to adult care
- When problems with achieving HbA1c goals, quality of life, or self-management are identified
- General assessment
 - Attitudes about diabetes
 - Expectations
 - Quality of life
 - Available resources - financial, social, and emotional
- Screen for common mental health problems

Table 5.2 Common mental health problems among people with diabetes

	Depression	Anxiety	Cognitive impairment
When to screen?	Adolescents/ young adults Older adults Females Poor financial and social support History of stressful life events Poor glycaemic control, recurrent hypoglycaemia Longer duration of diabetes Long-term complications	Evidence of fear, irrational thoughts, repetitive behaviours or avoidance behaviour Poor adherence to monitoring or insulin due to needle phobia Chronic hyperglycaemia due to fear of hypoglycaemic episode	Older adults People with glycaemic variability Multiple vascular risk factors
How to screen?	Informal verbal inquiries		
	Modified-Patient-Health-Questionnaire PHQ-9	Beck Anxiety Inventory (BAI) Hypoglycaemia Fear Survey-II (HFS-II)	Mini Mental Scale Examination (MMSE) Montreal Cognitive Assessment (MOCA)
What to do if positive?	Psychotherapy Reinforce DSMES	Reinforce DSMES Behaviour therapy	Address reversible contributors to cognitive dysfunction (depression, medicines, thyroid disease and delirium) Minimise the risk of hypoglycaemia Simplify treatment regime
	Refer to a mental health specialist		

References

1. American Diabetes Association, Facilitating Behavior Change and Well-being to Improve Health Outcomes: Standards of Medical Care in Diabetes—2025. *Diabetes Care*, 2025. 44(Supplement 1):
2. Fisher L, Polonsky WH, Hessler D. Addressing diabetes distress in clinical care: a practical guide. *Diabet Med*. 2019 Jul;36(7):803-812. doi: 10.1111/dme.13967. Epub 2019 May 7. PMID: 30985025.
3. Fisher, L., Gonzalez, J. S., & Polonsky, W. H. (2014). The confusing tale of depression and distress in patients with diabetes: a call for greater clarity and precision. *Diabetic medicine : a journal of the British Diabetic Association*, 31(7), 764–772. <https://doi.org/10.1111/dme.12428>
4. Davies, M.J., et al., Management of Hyperglycemia in Type 2 Diabetes, 2018. A Consensus Report by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). *Diabetes Care*, 2018. 41(12): p. 2669-2701.

6. PREVENTION OF DIABETES MELLITUS

1. What are the lifestyle and behaviour changes recommended for diabetes prevention?

- Intensive lifestyle interventions are recommended for high risk individuals.
- Goals: $\geq 7\%$ weight loss, ≥ 150 min/ week moderate intensity physical activity.
- Evidence-based eating patterns - Mediterranean diet, low carbohydrate diet, intermittent fasting have shown benefits.

2. What are the recommendations for nutrition & physical activity?

- Reduce fat and calorie intake
- Aim for a calorie deficit of 500 - 1000 kilocalories/ day.
- Customize macronutrients based on preferences/ goals.
- Aim for at least 700 kcal/ week expenditure from physical activity (for ease it is described as at least 150 min of moderate-intensity physical activity per week, similar in intensity to brisk walking)
- Combine aerobic exercise and resistance training.
- Break up sedentary time.

3. What is the association between sleep and diabetes?

- Both < 6 hours and > 8 hours of sleep increase type 2 diabetes risk.
- Poor sleep quality & evening chronotype (go to bed late and get up late) linked to higher type 2 diabetes risk
- Sleep is promoted as a key lifestyle factor.

4. What are the pharmacological prevention methods of type 2 diabetes?

- Metformin is effective for high risk groups especially those aged 25–59 years with BMI ≥ 23 kg/m², higher fasting plasma glucose (e.g., ≥ 110 mg/dL), and higher HbA1c (e.g., $\geq 6.0\%$), and in individuals with prior gestational diabetes mellitus.
- Monitor for vitamin B12 deficiency with long term use.
- Other agents with benefits: liraglutide, semaglutide, pioglitazone
- Semaglutide and tirzepatide have been shown to reduce the incidence of type 2 diabetes in those with prediabetes.

5. What are the components of type 1 diabetes prevention?

- Lifestyle factors (e.g., activity, sugar intake) may affect progression of type 1 diabetes.
- Teplizumab: delays stage 3 type 1 diabetes in at risk children (≥ 8 years)
- Requires specialist care and selection based on markers.

References

1. Knowler WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 2002;346:393–403
2. Gong Q, Zhang P, Wang J, et al. Morbidity and mortality after lifestyle intervention for people with impaired glucose tolerance: 30-year results of the Da Qing Diabetes Prevention Outcome Study. *Lancet Diabetes Endocrinol* 2019;7:452–461
3. Knowler WC, Fowler SE, Hamman RF, et al. 10-year follow-up of diabetes incidence and weight loss in the Diabetes Prevention Program Outcomes Study. *Lancet* 2009;374:1677–1686
4. Diabetes Prevention Program (DPP) Research Group. The Diabetes Prevention Program (DPP): description of lifestyle intervention. *Diabetes Care* 2002;25:2165–2171
5. Ramachandran A, Snehalatha C, Mary S, et al. The Indian Diabetes Prevention Programme shows that lifestyle modification and metformin prevent type 2 diabetes in Asian Indian subjects with impaired glucose tolerance (IDPP-1). *Diabetologia* 2006;49:289–297
6. Salas-Salvadó J, Bulló M, Babio N, et al. Reduction in the incidence of type 2 diabetes with the Mediterranean diet: results of the PREDIMED-Reus nutrition intervention randomized trial. *Diabetes Care* 2011;34:14–19
7. Zare M, Delpino F, Speer B, et al. Using community health workers to address barriers to participation and retention in Diabetes Prevention Program implementation. *Prim Care Diabetes* 2023;17:125–131
8. Sattar N, Gill JMR, Alazawi W. Improving prevention of type 2 diabetes in primary care: a systematic review of interventions using weight loss as the goal. *Lancet Diabetes Endocrinol* 2021;9:563–574
9. Lau NKH. Nutrition therapy for adults with prediabetes or diabetes. *Diabetol Metab Syndr* 2023;15:110
10. Gerstein HC, Bosch J, Dagenais GR, et al. Basal insulin and cardiovascular and other outcomes in dysglycemia. *N Engl J Med* 2012;367:319–328

11. Li G, Zhang P, Wang J, et al. Cardiovascular mortality, all-cause mortality, and diabetes incidence after lifestyle intervention for people with impaired glucose tolerance in the Da Qing Diabetes Prevention Study: a 23-year follow-up study. *Lancet Diabetes Endocrinol* 2014;2:474-480
12. Aroda VR, Edelstein SL, Goldberg RB, et al. Long-term metformin use and vitamin B12 deficiency in the Diabetes Prevention Program Outcomes Study. *J Clin Endocrinol Metab* 2016; 101:1754–1761

7. PATIENT ASSESSMENT AND FOLLOW UP

A. Initial and annual assessment

1. What are the factors to assess during the initial and annual assessment of a person with diabetes?

A complete medical evaluation should be performed at initial visit with the following aims.

- Confirm the diagnosis and classify diabetes
- Assess glycemic status and previous treatment
- Evaluate diabetes complications and other potential comorbidities
- Develop a plan for continuing care considering the social and structural barriers

Table 7.1 Factors to assess during initial and annual assessment

Domains	Factors
History related to diabetes*	• Confirm the diagnosis and classify type of diabetes • Characteristics at onset (eg., age, symptoms) • Previous treatment regimens and response • Current medication regimen and medication taking behaviour • Medication intolerance and side effects • Complementary and alternative medicine use • Previous hospitalizations for diabetes related reasons
Glycaemic control	• Self-monitoring of blood glucose • Recent laboratory tests related to glycaemic control • Recent hyperglycaemic or hypoglycaemic episodes
Diabetes related complications	• Macrovascular disease ▪ Cerebrovascular disease ▪ Cardiovascular disease ▪ Peripheral vascular disease • Microvascular disease ▪ Diabetes-related eye disease ▪ Diabetic kidney disease ▪ Diabetic neuropathy • Hypoglycaemia ▪ Awareness ▪ Severity, frequency and timing • Foot ▪ Skin changes, neuropathic/ vascular ulcers, joint deformities, loss of protective sensation, footwear
Comorbidities	• Hypertension • Dyslipidaemia • Non-alcoholic fatty liver disease • Periodontal disease • Hypothyroidism • Tuberculosis (persistent cough, unexplained weight loss etc.) • Calculate WHO 10-year CVD risk
Family history*	• Type 2 diabetes, atherosclerotic cardiovascular disease, chronic kidney disease, autoimmune disorders, etc.
Lifestyle factors	• Nutrition/ eating pattern • Physical activity • Weight history • Sleep behaviour • Tobacco, alcohol and other substance use

Diabetes self-management skills	• Education related to diabetes and complications • Medical nutrition therapy • Self-monitoring of glucose • Social and economic constraints
Psychological condition	• Depression/ anxiety and eating disorders • Cognitive impairment when indicated • Family and social support
Pregnancy planning	• For women with childbearing potential, contraceptive need and preconception care
Vaccinations	• Vaccination history • Assessment of the requirement
Physical examination	• Height, weight, BMI, waist circumference, neck circumference • Blood pressure, (orthostatic drop of blood pressure when indicated) • Fundoscopic eye examination (refer to the specialist) • Thyroid palpation • Skin examination (acanthosis, injection sites, skin infections, diabetic dermopathy, lipodystrophy) • Comprehensive foot examination ▪ Visual inspection (skin integrity, callus, ulcers, foot deformity, toenails) ▪ Pedal pulses, ABPI if diminished ▪ Sensory examination: ♦ Large fibre: Vibration, 10 g monofilament test ♦ Small fibre: temperature or pin prick sensation
Investigations	• HbA1c • Fasting plasma glucose, postprandial plasma glucose • Lipid profile • AST, ALT • Spot urine albumin: creatinine ratio (UACR) • Serum creatinine and eGFR • Serum electrolytes • Electrocardiogram • Full blood count Optional if clinically indicated • Urinalysis • TSH • Vit B12 level if on long term metformin or neuropathy • Vitamin D level

*May not be needed in the annual assessment

ABPI-ankle brachial pressure index, BMI-body mass index, CVD-cardiovascular disease, eGFR-estimated glomerular filtration rate, TSH-thyroid-stimulating hormone

- Prioritise the components based on available resources.
- Ensure that every patient receives comprehensive annual assessment.
- Refer to annexure 7.1 and 7.2 for checklists for initial and annual assessment.

B. Interval follow up

2. What is the frequency of follow up visits?

Individualise the interval of follow up visits depending on the clinical context.

- 1-3 months for most patients
- Earlier after an acute illness, insulin initiation or if suboptimal glycaemic control

3. What are the factors to assess during follow up visits?

Table 7.2 Factors to assess during follow up visits

Domains	Factors
History related to diabetes	- Interval medical history (significant changes since last visit) - Preconception care - Hypoglycaemia/ hyperglycaemia - Medication taking behaviour and intolerance/ side effects
Lifestyle factors	- Diabetes self-management behaviour - Nutrition and physical activity - Psychosocial health
Physical examination	BMI, BP, skin, changing foot conditions
Laboratory evaluation	- Glycaemic control - Therapeutic goals/ metabolic targets
Referrals	Need for referrals, immunisations, or other routine health maintenance screening

C. Assessment of comorbidities

(Refer to cardiovascular and diabetic kidney disease assessment)

4. How to assess and manage metabolic liver disease?

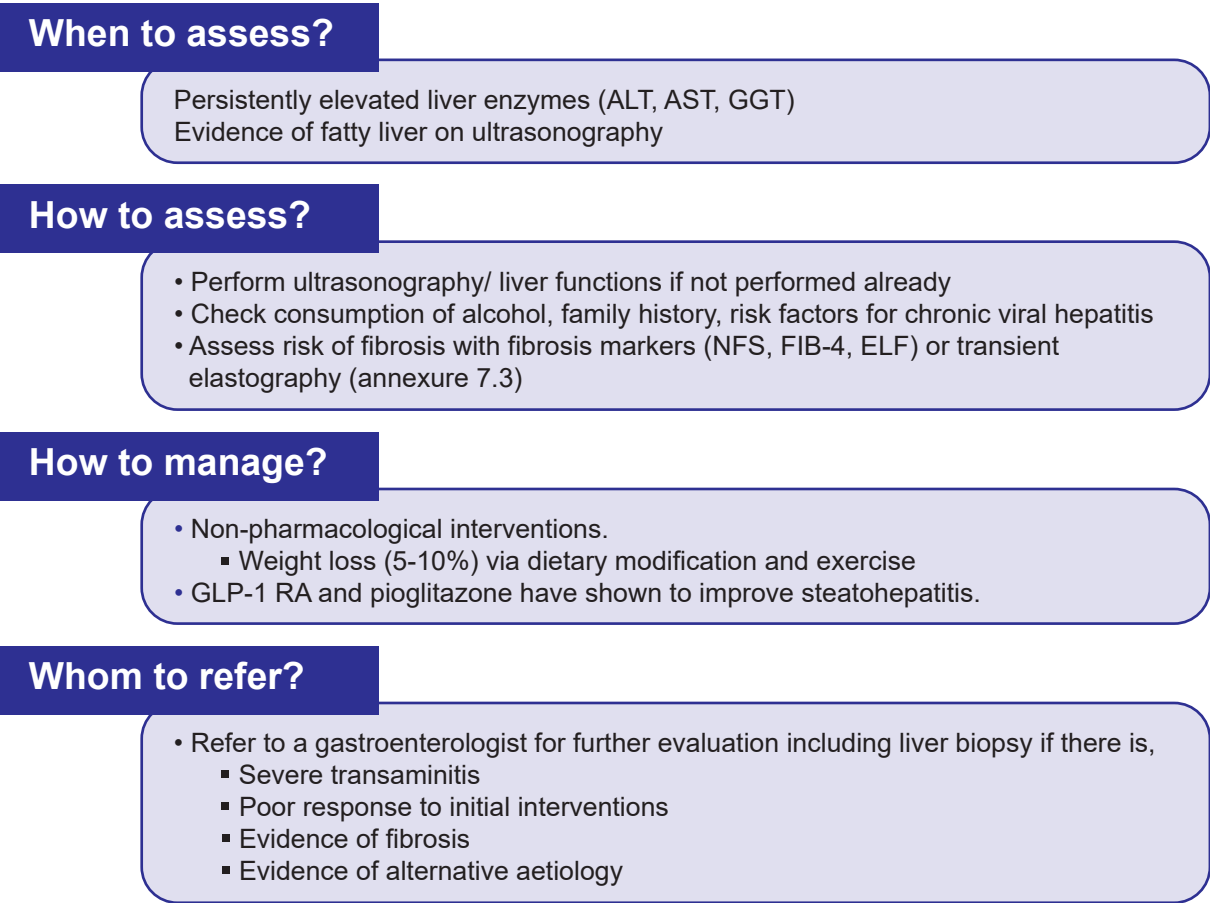


Figure 7.1 Assessment and management of metabolic liver disease in people with diabetes

5. How to assess and manage obstructive sleep apnoea (OSA)?

How to screen?

- All individuals with type 2 diabetes should be screened for symptoms suggestive of OSA
 - Does the person snore?
 - Does the person complain of day time sleepiness?
 - Does the person have obesity, retrognathia of the lower jaw or hypertension?

How to evaluate?

- Comprehensive sleep history
 - Witnessed snoring, apnoea, choking at night
 - Excessive sleepiness not explained by other factors including assessment of severity of sleepiness with the Epworth Sleepiness Scale
 - Non-refreshing sleep, total sleep amount, sleep fragmentation, nocturia, morning headaches, decreased concentration, memory loss, decreased libido, irritability
- Examination
 - BMI, neck circumference, modified Mallampati score, retrognathia, macroglossia, tonsillar hypertrophy, nasal abnormalities
- Evaluate for complications
 - Hypertension, stroke, myocardial infarction, cor pulmonale, decreased day time alertness, motor vehicle accidents

How to assess severity?

- Polysomnography
 - In the sleep laboratory
 - Home testing with portable monitors

How to manage?

- Refer to sleep specialist
 - General education - weight loss, sleep position, alcohol avoidance, risk factor modification and medication effects.
 - Positive airway pressure therapy - mainstay of treatment for mild, moderate and severe OSA

Figure 7.2 Assessment and management of obstructive sleep apnoea in people with diabetes

6. How to assess for autoimmune disorders?

- Screen individuals with type 1 diabetes for following autoimmune diseases;
 - Thyroid disease
 - at diagnosis and
 - periodically thereafter
 - Screen for other conditions if clinically indicated,
 - Coeliac disease
 - Addison disease
 - Pernicious anaemia

7. How to assess and manage sexual dysfunction in men with diabetes?

- Sexual dysfunction in men with diabetes includes,
 - Erectile dysfunction
 - Ejaculatory disorders (premature ejaculation, retrograde ejaculation)
 - Hypoactive sexual desire disorder
- Sexual dysfunction is commonly multifactorial in individuals with following conditions
 - Autonomic neuropathy
 - Arterial insufficiency
 - Endothelial dysfunction
 - Mental health issues
 - Hypogonadism
 - Treated with multiple medications

How to assess?

History: sexual history, morning erections, metabolic control, drug history, evidence of hypogonadism, mental health issues, cardiovascular risk
Examination: assessment for complications of diabetes (vascular disease, neuropathy), genital examination, features of hypogonadism
Investigations: tests for metabolic control and complications, total testosterone etc.

How to optimise?

- Patient ± partner education
- Optimise metabolic control (blood glucose, blood pressure and lipids)
- Establish healthy lifestyle and healthy body weight
- Adjust medications (thiazide diuretics, tricyclic antidepressants and selective serotonin and norepinephrine reuptake inhibitors as these are associated with erectile dysfunction)
- Treat any specific aetiology
 - Hypogonadism
 - Depression, performance anxiety

How to treat?

- Firstline pharmacotherapy - phosphodiesterase inhibitors (contraindicated with nitrates)
 - Sildenafil - 25 - 100 mg on demand
 - Tadalafil - 5 - 20 mg on demand/ 2.5 - 5 mg daily

How to treat non-responders?

- Intra-urethral/ intracavernosal alprostadil
- Vacuum erection devices
- Penile prosthesis

Figure 7.3 Assessment and management of sexual dysfunction in men with diabetes

8. What are the recommended vaccines?

- Annual Influenza vaccine:
 - All people above 6 months of age
- Pneumococcal vaccine:
 - <65 years - one dose of 23 - valent pneumococcal polysaccharide vaccine (PPSV23) and one final dose at 65 years or older and it should be given at least 5 years after the most recent dose of PPSV23.
 - ≥ 65 years - one dose of pneumococcal vaccine (PPSV23), 5 years or more after any prior dose of PPSV23. No additional PPSV23 doses are required following the dose administered at 65 years of age or older.

References

1. American Diabetes Association, Comprehensive Medical Evaluation and Assessment of Comorbidities: Standards of Medical Care in Diabetes- 2025. Diabetes Care, 2025.
2. Targher, G., A. Lonardo, and C.D. Byrne, Nonalcoholic fatty liver disease and chronic vascular complications of diabetes mellitus. Nature reviews endocrinology, 2018. 14(2): p. 99-114.
3. Reutrakul, S. and B. Mokhlesi, Obstructive sleep apnea and diabetes: a state of the art review. Chest, 2017. 152(5): p. 1070-1086.
4. Kizilay, F., H.E. Gali, and E.C. Serefoglu, Diabetes and Sexuality. Sex Med Rev, 2017. 5(1): p. 45-51.

8. GLYCAEMIC TARGETS

1. What tests can be used to monitor glycaemic control in people with diabetes?

- Self-monitoring of blood glucose (SMBG): self-testing using glucose measuring devices at home
- HbA1c
- Fasting plasma glucose (FPG) and postprandial plasma glucose (PPPG) after main meals if above not available or not affordable.
- Continuous glucose monitoring (CGM)

2. Who should be offered SMBG?

- Almost all people with diabetes will benefit from SMBG.
- It is especially indicated in following circumstances,
 - Individuals treated with insulin
 - Evidence of hypoglycaemic episodes
 - Using medications that may increase the risk of hypoglycaemia while driving or operating machinery
 - Pregnancy or planning for a pregnancy

3. How does HbA1c compare to FPG/ PPPG for glycaemic monitoring in clinical practice?

Table 8.1 Advantages and disadvantages of HbA1c compared to plasma glucose in monitoring

	HbA1c	FPG/PPPG
Advantages	High precision and accuracy Reflects control over a long duration Better correlation with patient outcomes (morbidity and mortality)	Low cost Wide availability
Disadvantages	Interferences related to non-glycaemia related causes (refer section 3) Does not reflect glycaemic fluctuations (hypoglycaemia and hyperglycaemia) Errors due to assay used Higher cost Limited availability	Higher variability Usually does not reflect overall fluctuations (hypoglycaemia and hyperglycaemia) Usually underestimates glycaemic burden

Utilise HbA1c in all people with diabetes to monitor glycaemic control

4. What assay should be used to measure HbA1c?

National Glycohaemoglobin Standardisation Program (NGSP) standardised, Diabetes Complication and Control Trial (DCCT) or International Federation of Clinical Chemistry (IFCC) traceable method

5. How frequently should HbA1c be checked?

- 6 monthly if meeting treatment goals and having a stable glycaemic control. At least annually in a resource limited setting
- Every 3 months and as needed in whose therapy recently changed and/or are not meeting glycaemic goals

6. What is the correlation between HbA1c and estimated average glucose?

Table 8.2 Correlation between HbA1c and average blood glucose

HbA1c (%)	HbA1c (mmol/mol)	mg/dL
5	31	97 (76–120)
6	42	126 (100–152)
7	53	154 (123–185)
8	64	183 (147–217)
9	75	212 (170–249)
10	86	240 (193–282)
11	97	269 (217–314)
12	108	298 (240–347)

7. What are the alternative parameters to monitor glycaemic control when HbA1c is not reliable?

- Quality controlled plasma glucose profiles
- Fructosamine estimation
- Total glycated haemoglobin

8. What are the serum glycated protein assays used as alternatives to HbA1c?

- **Fructosamine:** reflects total glycated serum proteins (mostly albumin).
- **Glycated albumin:** reflect the proportion of total albumin that is glycated.
 - Due to the turnover rate of serum protein, fructosamine and glycated albumin reflect glycemia over the past 2 – 4 weeks, a shorter-term time frame than that of A1C.
 - Individuals who have conditions where the interpretation of A1C may be problematic or when A1C cannot be measured (e.g. haemoglobinopathies) these assays are a useful alternative.

9. What are the recommended glycaemic goals?

- | | | |
|--|---|----------------|
| 1) HbA1c | - | <7% |
| 2) Pre-prandial capillary plasma glucose | - | 80 – 130 mg/dL |
| 3) Peak post prandial capillary plasma glucose | - | <180 mg/dL |

Use individualised targets according to the patient characteristics.

10. How should glycaemic targets be individualised?

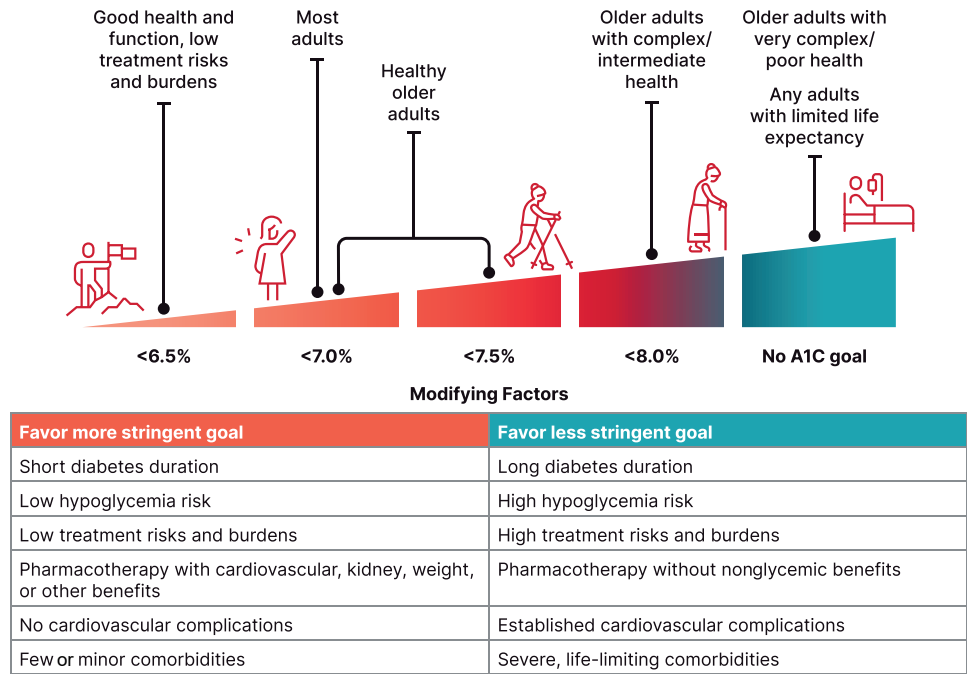


Figure 8.1 Individualized A1C goals for nonpregnant adults

Source: American Diabetes Association Professional Practice Committee; 6. Glycemic Goals and Hypoglycemia: Standards of Care in Diabetes—2025. Diabetes Care 1 January 2025; 48 (Supplement_1): S128–S145

11. What are the important CGM metrics, and what should be the goals?

Table 8.3 CGM metrics for clinical care in nonpregnant individuals with type 1 or type 2 diabetes

CGM use

Metric	Interpretation	Recommended Goal
CGM Wear Time	Number of days CGM is worn	≥14 days (for reliable pattern assessment)
CGM Active Time	Percentage of time CGM is recording data	≥70% over the 14-day period

Glycaemic metrics

Metric	Interpretation	Recommended Goal
Mean Glucose	Average glucose level over the monitoring period	Individualised; used to calculate GMI
GMI (Glucose Management Indicator)	Estimate of HbA1c based on mean glucose	Used with lab HbA1c; not a replacement
Glucose CV (%)	Degree of glucose variability	≤36%; <33% may reduce hypoglycaemia risk
TAR >250 mg/dL (Level 2 hyperglycaemia)	Time with severe hyperglycaemia	<5% (most adults); <10% (older adults)
TAR 181–250 mg/dL (Level 1 hyperglycaemia)	Time with mild hyperglycaemia	<25% (most adults); <50% (older adults)
TIR 70–180 mg/dL	Time in target glucose range	>70% (most adults); >50% (older adults)
TBR 54–69 mg/dL (Level 1 hypoglycaemia)	Time with mild hypoglycaemia	<4% (most adults); <1% (older adults)
TBR <54 mg/dL (Level 2 hypoglycaemia)	Time with clinically significant hypoglycaemia	<1% for all

CGM-continuous glucose monitoring, TAR-time above range, TBR-time below range,

References

1. American Diabetes Association Professional Practice Committee; 6. Glycemic Goals and Hypoglycemia: Standards of Care in Diabetes—2025. *Diabetes Care* 1 January 2025; 48 (Supplement_1): S128–S145
2. National Institute for Clinical Excellence. Type 2 diabetes in adults: management. 2015
3. Garber AJ, Handelsman Y, Grunberger G, et al. Consensus statement by the American association of clinical endocrinologists and American college of endocrinology on the comprehensive type 2 diabetes management algorithm – 2020 executive summary. *Endocrine Practice*. 2020;26(1):107-139.
4. Inzucchi SE, Bergenstal RM, Buse JB, et al. Management of hyperglycemia in type 2 diabetes, 2015: a patient-centered approach: update to a position statement of the American Diabetes Association and the European Association for the Study of Diabetes. *Diabetes Care* 2015;38: 140–149
5. Goldenberg RM, Aroda VR, Billings LK, Donatsky AM, Frederiksen M, Klonoff DC, Kalyanam B, Bergenstal RM. Correlation Between Time in Range and HbA1c in People with Type 2 Diabetes on Basal Insulin: Post Hoc Analysis of the SWITCH PRO Study. *Diabetes Ther*. 2023 May; 14(5):915-924. doi: 10.1007/s13300-023-01389-2. Epub 2023 Mar 11. PMID: 36905485; PMCID: PMC10126196

9. NON-PHARMACOLOGICAL MANAGEMENT

A. Medical Nutrition Therapy

1. What are the goals of medical nutrition therapy in diabetes?

- Encourage healthy and pleasurable eating
To achieve and maintain;
 - healthy body weight
 - glycaemic targets
 - metabolic goals
- Provide a meal plan
Based on;
 - cultural and personal preferences
 - health literacy
 - affordability, availability and accessibility

Allow pleasure of eating while motivating to change food habits and behaviour.

2. What are the general recommendations for dietary planning for adults with diabetes?

- MNT is associated with HbA1C absolute decreases of 1.0–1.9% for people with type 1 diabetes and 0.3–2.0% for people with type 2 diabetes
- There is inadequate evidence for a particular eating pattern in the Sri Lankan context.
- Plate method is the widely used method.

3. What is the plate method?

- The plate method encourages a balanced meal with;
 - Grains and starchy vegetables (1/4 of the plate)
 - Protein rich foods such as fish, white meat and pulses (1/4 of the plate)
 - Non-starchy vegetables, green leaves (1/2 of the plate)
- Consumption of such a mixed meal effectively reduces the glycaemic index of the starchy food and reduces post meal glucose excursions.

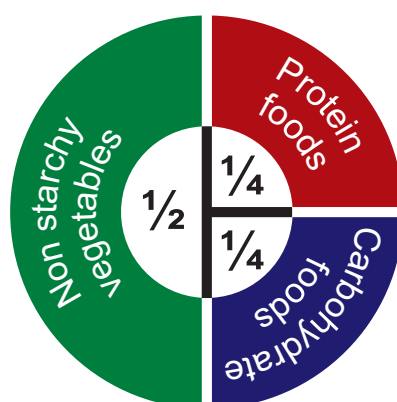


Figure 9.1 Suggested plate model to demonstrate a healthy meal for people with diabetes

Reprinted from 'Carb Counter- for people living with diabetes' by Maulee Arambewela, Chandrika Subasinghe and Sonali Gunatilake (Ed.), 2021, Sri Lanka College of Endocrinologists

4. What types of carbohydrates should be chosen?

An appropriate portion size in a mixed-meal is more important than type of carbohydrate in achieving metabolic targets.

- An appropriate portion size is important to reduce the glycaemic load. (e.g., 1 cup of rice or 2 slices of bread)
- Encourage minimally processed whole grains which are high in fibre over refined carbohydrates such as baked products.
- Educate on hidden carbohydrates,
 - Starchy vegetables (potato, sweet potato, manioc, bread fruit, jack fruit etc.)
 - Pulses (dhal, green gram, chickpeas, cowpea etc.)
 - Fruits
 - Dairy (milk, yoghurt, ice cream etc.)
- Minimise the intake of sugary food (candy, biscuits, cakes etc.) and sugar sweetened beverages

Sugar substitutes

- These include high-intensity sweeteners, artificial sweeteners, non-nutritive sweeteners, and low-calorie sweeteners.
- These may adversely affect hunger and satiety.
- Evidence on long term control of weight, glycaemic and cardio-metabolic risk is limited.
- These are best avoided.

5. What are the additional recommendations for those on insulin?

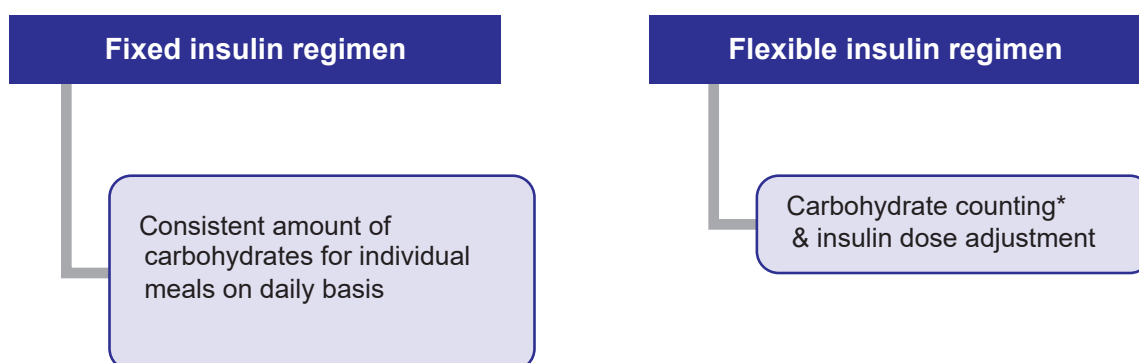


Figure 9.2 Dietary approach based on the type of insulin therapy

**Arambewela M, Subasinghe S, Gunatilake S. Carb Counter for People Living with Diabetes. Triple Publishers, 2021 and Atapattu N, De Silva S. Food guide for children with diabetes. Triple publishers; 2015, Carb counter Sri Lanka App*

6. What is carb exchange and how is it going to help individuals with diabetes?

- A carb exchange is the amount of food containing approximately 15 g of carbohydrates.
- A carb exchange list provides information on the serving size of food that is equal to 1 exchange.
- Different carbohydrate foods can be exchanged for one another so that a similar amount of carbohydrates is consumed for individual meals on a daily basis.
- This will help minimise blood glucose fluctuations in patients on fixed drug regimens (oral hypoglycaemic agents/ insulin).

7. What types of protein sources are encouraged?

- High quality protein containing foods.
 - e.g., fish, chicken (skinless), lean meat, eggs and soya
 - minimise red meat intake e.g., beef, pork and mutton
- Avoid processed meat e.g.,
 - sausages, salami and bacon
- Pulses are a good source of protein; however consider the moderate amount of carbohydrates they contain
 - dhal, green gram, chick peas
- Recommend a protein intake of 0.8 g/kg/day and for older adults at least 1g/kg/day.

8. Which types of fat containing foods are recommended?

- Total fat intake can be reduced by lowering the consumption of coconut milk and oil.
- Encourage healthy fats such as oily fish, avocados, nuts, seeds, olive oil, gingelly oil and canola oil containing mono and polyunsaturated fatty acids.
- Discourage foods containing saturated fatty acids such as cakes, pastries, margarine, red meat and deep fried foods containing trans-fats.
- Discourage deep frying. Stir-frying with a small amount of a healthy oil is acceptable. However,
 - Minimise frying, tempering and oiling.
 - Promote alternative cooking methods such as boiling and baking.
 - Advise trimming off visible fat before cooking.

Note:

Supplementation with 4 g/day of pure eicosapentaenoic acid significantly lowered the risk of adverse cardiovascular events. (REDUCE-IT)

9. What general tips are suggested for adults with diabetes?

- Distribute total calories consumed on a day into 3 main meals and 2 - 3 snacks.
- Fruits are best consumed as snacks in between main meals rather than immediately after meals.
- Encourage 1 - 2 portions of low fat dairy products per day
 - without added sugar
 - before breakfast or in the evening
- Advise to limit salt intake (salt intake of <5 g/ day or sodium <2300 mg/ day).
 - reduce the amount of added salt
 - use other flavours such as herbs, spices, garlic and citrus
 - avoid salt-rich food such as restaurant food, processed and packed food.
- Alcohol is best avoided to prevent glycaemic fluctuations and weight gain.

10. What is the evidence for specific eating patterns/ meal plans?

Table 9.1 Specific eating patterns used by people with diabetes, their advantages and disadvantages

	Low and very low carbohydrate diet	Low total fat diet	Mediterranean diet	DASH diet	Intermittent fasting
What is it?	Very low-carbohydrate diet: 21–70 g/day of carbohydrate Moderately low-carbohydrate diet: 30–40% of kcal as carbohydrate	Reduced total fat intake to <25% of daily energy intake	More vegetables, whole grains, fruit, legumes, nuts, fish, and MUFA/PUFA Less red meat and saturated fat and some alcohol (red wine)	Vegetables and fruits, lean meat and dairy products Reduction of sodium 1500 mg/day Minimally processed and fresh food	Time restricted: fasting ranging from 12 to 20 hours Alternate day fasting: fasting days alternating with days where foods and beverages are consumed
Advantages	Reduce HbA1C and weight Improvement in lipids except HDL	Weight loss	May improve weight, HDL cholesterol and triglycerides	Improves blood pressure	Weight loss
Disadvantages	Lack of long term data	No impact on glycaemia or CVD risk	No independent effect on HbA1c	No impact on HbA1c, weight and lipids	No impact on HbA1c Inadequate published literature

DASH-Dietary Approaches to Stopping Hypertension, MUFA-monounsaturated fatty acids, PUFA-poly-unsaturated fatty acids, HDL-high density lipoprotein, CVD-cardiovascular disease

11. What are the types of intermittent fasting?

- Alternate-day fasting (energy restriction of 500 – 600 calories on alternate days)
- The 5:2 diet (energy restriction of 500 – 600 calories on two consecutive or non-consecutive days with usual intake the other five days)
- Time-restricted eating (daily calorie restriction based on a daily 16 hours fasting and an 8 hours eating window).

12. What are the effects of intermittent energy restricted eating?

- Mild to moderate weight loss (3 – 8% loss from baseline) over short durations (8–12 weeks)
- Intermittent energy restriction produces small but significant reductions in waist circumference and fat-free mass but were otherwise not superior to continuous energy restriction diets
- Generally, time-restricted eating or shortening the eating window can be adapted to any eating pattern and has been shown to be safe for adults with type 1 or type 2 diabetes.

B. Physical activity

13. What are the benefits of exercise in adults with diabetes?

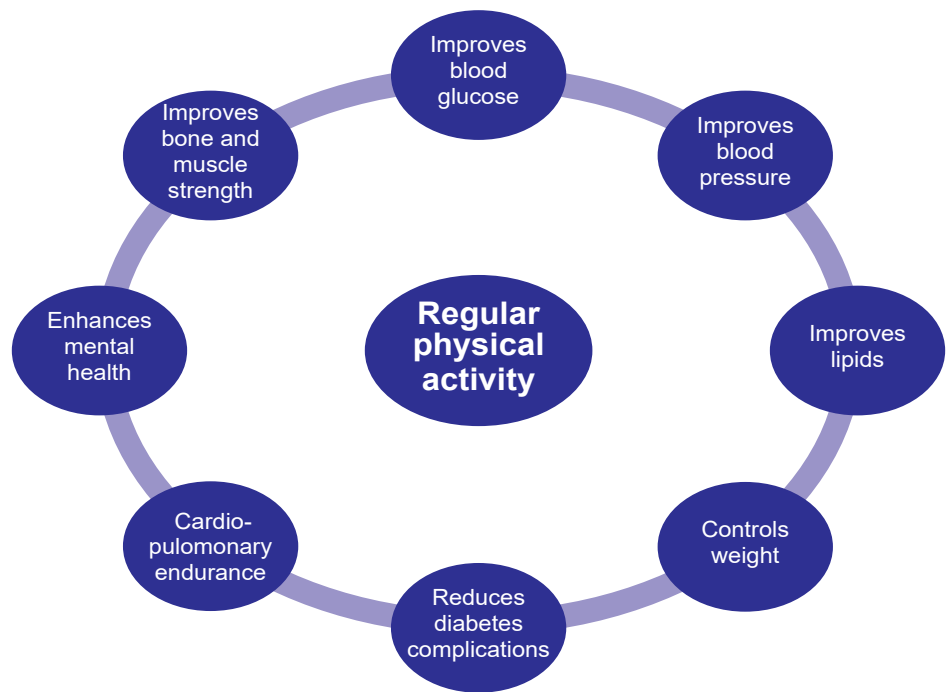


Figure 9.3 Benefits of exercise in adults with diabetes

14. What types of exercise and physical activity are recommended?

Table 9.2 Recommended types of exercise and examples for people with diabetes

	Moderate intensity aerobic activity	Muscle strengthening	Flexibility and balance exercises (particularly important in older adults)
What?	• Brisk walking • Jogging • Swimming (slowly) • Cycling on flat ground	• Using own body weight : squats, push-ups, calf raises • Using free weights (such as dumbbells) and machine assisted weights	• Yoga • Tai chi
How much?	• At least 30 min/day (continuous or 10-minute short bouts) • At least 150 min/week (for weight loss, double this amount is required) • No more than 2 consecutive rest days	• 2–3 sessions/week on non-consecutive days • 2-3 sets each of 8-12 repetitions • In a single schedule target to do 5-10 different exercises on different muscle groups	

Note :
Reduce the amount of time spent being sedentary and break up bouts of sedentary activity (30 min) by briefly standing, walking etc. Encourage step counting and promote 6000-10000 steps per day.

15. What are the types and quantities of physical exercise recommended for those with diabetes?

- 150 min or more of moderate- to vigorous-intensity aerobic activity per week, spread over at least 3 days/ week, with no more than 2 consecutive days without activity. Shorter durations (minimum 75 min/ week) of vigorous-intensity or interval training may be sufficient for more physically fit individuals.
- 2–3 sessions/ week of resistance exercise on non-consecutive days.
- Flexibility training and balance training 2–3 times/ week for older adults with diabetes. Yoga and tai chi may be included based on individual preferences to increase flexibility, muscular strength, and balance.
- For people who do not meet activity guidelines, encourage an increase in physical activities (e.g., walking, yoga, housework, gardening, swimming, and dancing) above baseline, and prolonged sitting should be interrupted at least every 30 min for blood glucose benefits.

16. What is High-Intensity Interval Training (HIIT)?

Short bursts of aerobic training performed between 65% and 90% VO₂ peak (a measure of maximal aerobic capacity) or between 75% and 95% peak heart rate for 10 seconds to 4 minutes with 12 seconds to 5 minutes of active or passive recovery.

17. What are the glycaemic and cardiovascular effects of HIIT?

- HIIT reduces A1C and BMI and improves fitness levels in individuals with T2D.
- In T1D, HIIT reduces A1C and insulin requirements and improves cardiometabolic risk profiles.
- Because HIIT can lead to transient increases in post-exercise hyperglycemia, individuals with T1D may need to use bolus correction and individuals with T2D are encouraged to monitor blood glucose when starting HIIT.

18. Which types of people with diabetes require pre-exercise evaluation?

- Pre-exercise medical evaluation is not needed for asymptomatic individuals receiving standard diabetes care who want to engage in low to moderate intensity exercises
- Refer individuals with,
 - uncontrolled hypertension and glucose fluctuations
 - cardiac disease
 - untreated proliferative retinopathy
 - autonomic neuropathy
 - peripheral neuropathy
 - history of foot ulcers or Charcot footfor specialised care before embarking on an exercise program.

19. How to prevent exercise induced hypoglycaemia in individuals on sulphonylurea or insulin?

- Educate on warning symptoms of hypoglycaemia.
- Advise to check CBG before exercise, during and after prolonged exercise.
 - CBG of 100 - 240 mg/dl before exercises is safe.
- Advise to carry 15 - 20 g of a rapid acting carbohydrate source (eg., one small bottle of fruit juice or 3 tsp of glucose to be consumed if hypoglycaemia develops.)
- During prolonged exercises (more than 60 minutes), patients should consume 15 - 20 g of carbohydrate source intermittently.
 - medium/ large sized banana
 - a sandwich
 - 2 - 3 biscuits
- If CBG is not available, advise to take a snack before exercise.

20. What are the recommendations regarding exercise in the setting of diabetic retinopathy?

- If proliferative or severe non-proliferative diabetic retinopathy is present, vigorous-intensity aerobic or resistance exercise may be contraindicated because of the risk of triggering vitreous haemorrhage or retinal detachment.
- Consultation with an ophthalmologist prior to engaging in an intense exercise plan may be appropriate (in those with diagnosed retinopathy).

21. What are the recommendations regarding exercise in the setting of peripheral neuropathy?

- 150 min/ week of moderate exercise improved outcomes in people with prediabetic neuropathy.
- All individuals with DPN should wear proper footwear and examine their feet daily to detect lesions early.
- Decreased pain sensation and a higher pain threshold in the extremities can result in an increased risk of skin breakdown, infection, and Charcot joint destruction.
- Therefore, a thorough assessment should be done to ensure that neuropathy does not alter kinesthetic or proprioceptive sensation during physical activity.
- Anyone with a foot injury or open sore should be restricted to non-weight-bearing activities.
- Moderate-intensity walking may not lead to an increased risk of foot ulcers or re-ulceration in those with DPN who use proper footwear.

22. How can autonomic neuropathy increase the risk of exercise-induced injury or adverse events?

- Decreased cardiac responsiveness to exercise
- Postural hypotension
- Impaired thermoregulation
- Impaired night vision due to impaired papillary reaction
- Greater susceptibility to hypoglycemia
- Cardiovascular autonomic neuropathy is also an independent risk factor for cardiovascular death and silent myocardial ischemia

23. What are the recommendations regarding exercise in the setting of autonomic neuropathy?

Individuals with autonomic neuropathy should undergo cardiac investigation before beginning physical activity more intense than they are accustomed to.

C. Obesity and overweight**24. What are the targets of weight reduction?**

- In prediabetes, the weight loss goal is at least 3 – 7% from baseline body weight, and higher for reducing risk of progression to type 2 diabetes.
- In conjunction with healthy lifestyle behaviours, medication-assisted weight loss can be considered for people at risk for T2D when needed to achieve and sustain 7 – 10% weight loss.
- For individuals with overweight/ or obesity alongside T2D, at least 5% weight loss is needed to achieve beneficial outcomes in glycemic management, lipids, and blood pressure
- More intensive weight loss goals (i.e., 15%) may be appropriate to maximize benefit depending on need, feasibility, and safety

25. What are the recommendations for individuals with overweight/ obesity and diabetes?

- Diet, physical exercises and behavioural therapy to achieve and maintain weight loss of at least $\geq 5\%$ over 3 - 6 months.
- Focus on diet and exercises to achieve a 500 - 750 kcal/day deficit for an individual;
 - reduce total calories from daily diet by about 500 kcal
 - increase daily activities (daily step count of 10,000 = 300 - 400 kcal expenditure)
- Get help of a trained nutrition specialist and exercise specialist, whenever possible.

Greater benefit of glycaemic control and cardiovascular risk reduction achieved with greater weight loss and maintenance.

26. What is the role of metabolic surgery in obese individuals with diabetes?

- It has multiple benefits including;
 - cost-effectiveness
 - improvement of morbidity and mortality
 - remission of diabetes and other metabolic complications
 - improvement of quality of life
- Refer to a multidisciplinary team
- Provide long term support and monitoring after surgery including micronutrient status and nutritional supplementation.

27. Who should be referred for metabolic surgery?

- Those with obesity class II (BMI $>30 \text{ kg}^2/\text{m}^2$) with type 2 diabetes

References

1. Facilitating Behavior Change and Well-being to Improve Health Outcomes: Standards of Medical Care in Diabetes; Diabetes Care, 44(Supplement 1), S53-S72.
2. Vitolins MZ, Anderson AM, Delahanty L, et al.; Look AHEAD Research Group. Action for Health in Diabetes (Look AHEAD) trial: baseline evaluation of selected nutrients and food group intake. J Am Diet Assoc 2009;109:1367–1375
3. Wheeler ML, Dunbar SA, Jaacks LM, et al. Macronutrients, food groups, and eating patterns in the management of diabetes: a systematic review of the literature, 2010. Diabetes Care 2012;35:434–445
4. Rubino, F., Nathan, D. M., Eckel, R. H., Schauer, P. R., Alberti, K. G. M. M., Zimmet, P. Z., Del Prato, S., Ji, L., Sadikot, S. M., Herman, W. H., Amiel, S. A., Kaplan, L. M., Taroncher-Oldenburg, G., & Cummings, D. E. (2016). Metabolic Surgery in the Treatment Algorithm for Type 2 Diabetes: A Joint Statement by International Diabetes Organizations. Diabetes Care, 39(6), 861 – 877.
5. Physical Activity/Exercise and Diabetes: A Position Statement of the American Diabetes Association Diabetes Care 2016;39:2065–2079
6. Obesity Management for the Treatment of Type 2 Diabetes: Standards of Medical Care in Diabetes—2025; Diabetes Care, 44 (Supplement 1), S100-S110.
7. Evert, A. B., Dennison, M., Gardner, C. D., Garvey, W. T., Lau, K. H. K., MacLeod, J., Mitri, J., Pereira, R. F., Rawlings, K., Robinson, S., Saslow, L., Uelman, S., Urbanski, P. B., & Yancy, W. S. (2019). Nutrition Therapy for Adults With Diabetes or Prediabetes: A Consensus Report. Diabetes Care, 42(5), 731 – 754.
8. Deepak L. Bhatt, M.D., M.P.H. <https://orcid.org/0000-0002-1278-6245>, P. Gabriel Steg, M.D., Michael Miller, M.D., Eliot A. Brinton, M.D., Terry A. Jacobson, M.D., Steven B. Ketchum, Ph.D., Ralph T. Doyle, Jr., B.A., +5 , for the REDUCE-IT Investigators*

10. PHARMACOLOGICAL MANAGEMENT

1. When should pharmacological treatment be initiated?

- Commence pharmacological therapy along with lifestyle modification as soon as the diagnosis is made.

Reasons for early pharmacotherapy

- Most individuals progress over time
- Most individuals fail on MNT alone
- Metabolic memory;
 - Early aggressive control improves long-term outcome
 - Poor control in early stages leads to higher long-term complications

2. What are the factors to consider when selecting pharmacological agents?

- Use a patient-centred approach to guide the choice of pharmacological agents.
- Consider the following:

- Effectiveness of drugs in terms of metabolic response
- Safety and tolerability
- Hypoglycaemia risk
- Impact on weight
- Risk of side effects

- Established atherosclerotic cardiovascular disease (ASCVD)
- Indicators of high-risk / chronic kidney disease (CKD) / heart failure (HF)

- Risks from drug interactions
- Patient preferences and needs
- Economic status of patient
- Recommended first line/ second line therapy

3. What are the preferred initial pharmacological agents?

- Metformin is preferred as the initial agent and should be continued as long as it is tolerated and not contraindicated, especially in patients without special considerations.
 - Offer metformin initially, and increase dose gradually to minimise gastrointestinal side effects.
 - Efficacy in lowering blood glucose - HbA1C reduction 1-2%
 - Low risk of hypoglycaemia
 - Potential benefits in weight - weight neutral
 - Reduces cardiovascular mortality
- Special considerations regarding metformin
 - GI intolerance – can be mitigated by graded dose titration or extended-release formulations
 - Increased risk of lactic acidosis when eGFR drops below 45ml/min/1.73m²
 - Vitamin B12 deficiency – with long term use, suggest periodic testing of Vitamin B 12 level.
- SGLT2 inhibitors can be considered early or even as the initial agent for patients with;
 - Cardiovascular comorbidities (established atherosclerotic cardiovascular disease or indicators of high risk [age ≥55 years with coronary, carotid, or lower extremity artery stenosis >50% or left ventricular hypertrophy])
 - Chronic kidney disease (eGFR 30 - 60 ml/min or UACR >30 mg/g, particularly UACR >300 mg/g)
 - Heart failure (LVEF<45%)

4. What are the initial alternatives if metformin is contraindicated?

Use any of the following;

- Dipeptidyl peptidase-4 inhibitor (DPP-4i)
- Sulfonylurea
- Pioglitazone
- Sodium-Glucose Cotransporter-2 inhibitor (SGLT2i)
- Glucose-like peptide-1 Receptor Agonist (GLP-1 RA)
- Insulin

5. When to consider initial combination therapy?

- Consider in patients presenting with HbA1c levels 1.5 - 2.0% above the target.
- Incorporation of high glycemic efficacy therapies.
- Adding therapies for cardiovascular and kidney disease risk reduction.
- Each new class of oral agents when added to metformin generally lowers A1C approximately by 0.7 – 1.0%.
- Addition of GLP-1 RA or dual GLP-1 RA and GIP-1 agonists to metformin results in >2% lowering of A1C.
- DO NOT recommend using GLP-1 RA together with a DPP-4 inhibitor - no added glucose-lowering benefit.
- In adults with type 2 diabetes and biopsy-proven MASH or those at high risk for liver fibrosis (based on noninvasive tests), pioglitazone, a GLP-1 RA, or a dual GIP and GLP-1 RA is preferred for glycemic management due to potential beneficial.

6. When to consider initial combination therapy with insulin?

Consider early introduction of insulin, in the following scenarios;

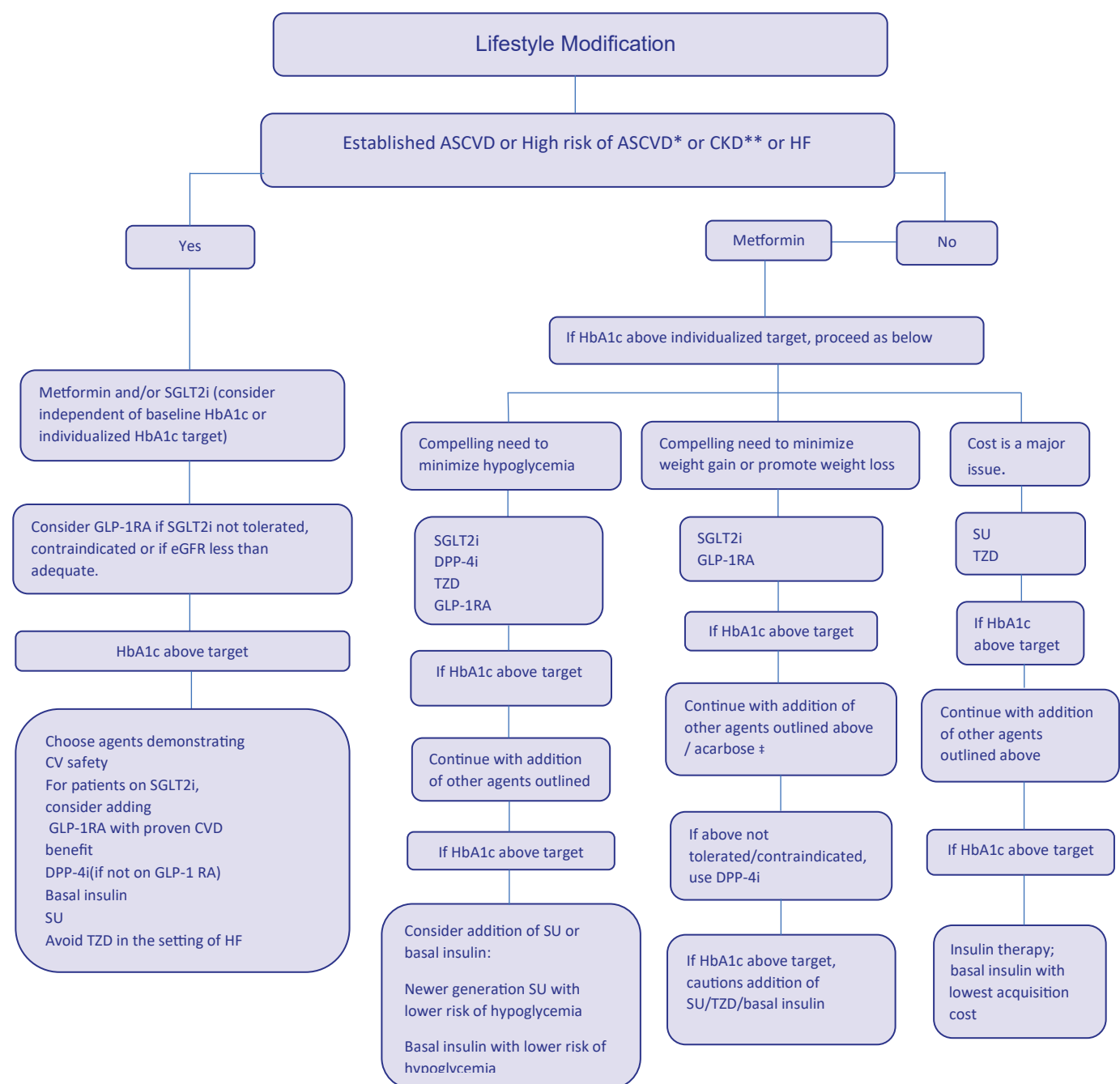
- Features of catabolism
 - Unintentional weight loss ($\geq 5\%$ of body weight over 6-12 months)
 - Ketonaemia
 - Hypertriglyceridaemia
- Severe symptoms of hyperglycaemia (significant polyuria/ polydipsia)
- HbA1c levels $>10\%$

7. How to monitor and intensify treatment?

- Multiple factors including progression of diabetes and patient factors affect glycaemic control
- Regular monitoring and addition of combination therapy will become necessary
- Re-evaluate the medication regimen and medication-taking behaviour at regular intervals (every 2 - 3 months).
- Adjust medications as needed to incorporate specific factors that impact choice of treatment.
- Do not delay intensification of treatment for patients not meeting treatment goals.

Break the therapeutic inertia

- Therapeutic inertia: lack of timely adjustment of therapy when patient's treatment goals are not met
- Could occur due to,
 - Patient factors
 - Clinician factors
 - Healthcare system factors
- Particularly common at stage of insulin initiation.
- Due to inertia, the patient lives with avoidable glycaemic burden for a longer duration leading to increased risk of complications.
- Act proactively to minimise glycaemic burden by breaking the inertia.



ASCVD: atherosclerotic cardiovascular disease
CKD: chronic kidney disease
HF: heart failure
HFrEF- heart failure with reduced ejection fraction
LVEF- left ventricular ejection fraction
LVH: left ventricular hypertrophy
GLP-1RA: glucagon like peptide -1 receptor agonist
SGLT2i: sodium-glucose transport protein 2 inhibitors
DPP4 i: dipeptidyl-peptidase 4 inhibitors
TZD: thiozolidinedione
SU: sulphonylurea
UACR- urine albumin: creatinine ratio

*age≥55 years with coronary, carotid, or lower extremity artery stenosis > 50%, or LVH
**CKD: Specifically, eGFR 30-60 mL/min/1.73m² or UACR >30 mg/g, particularly UACR>300 mg/g
‡ Acarbose may be useful in patients with significant post-prandial hyperglycaemia

Figure 10.1 Approach to selection of non-insulin pharmacotherapy in diabetes

In adults with type 2 diabetes and biopsy-proven MASH or those at high risk for liver fibrosis (based on noninvasive tests), pioglitazone, a GLP-1 RA, or a dual GIP and GLP-1 RA is preferred for glycemic management due to potential benefits.

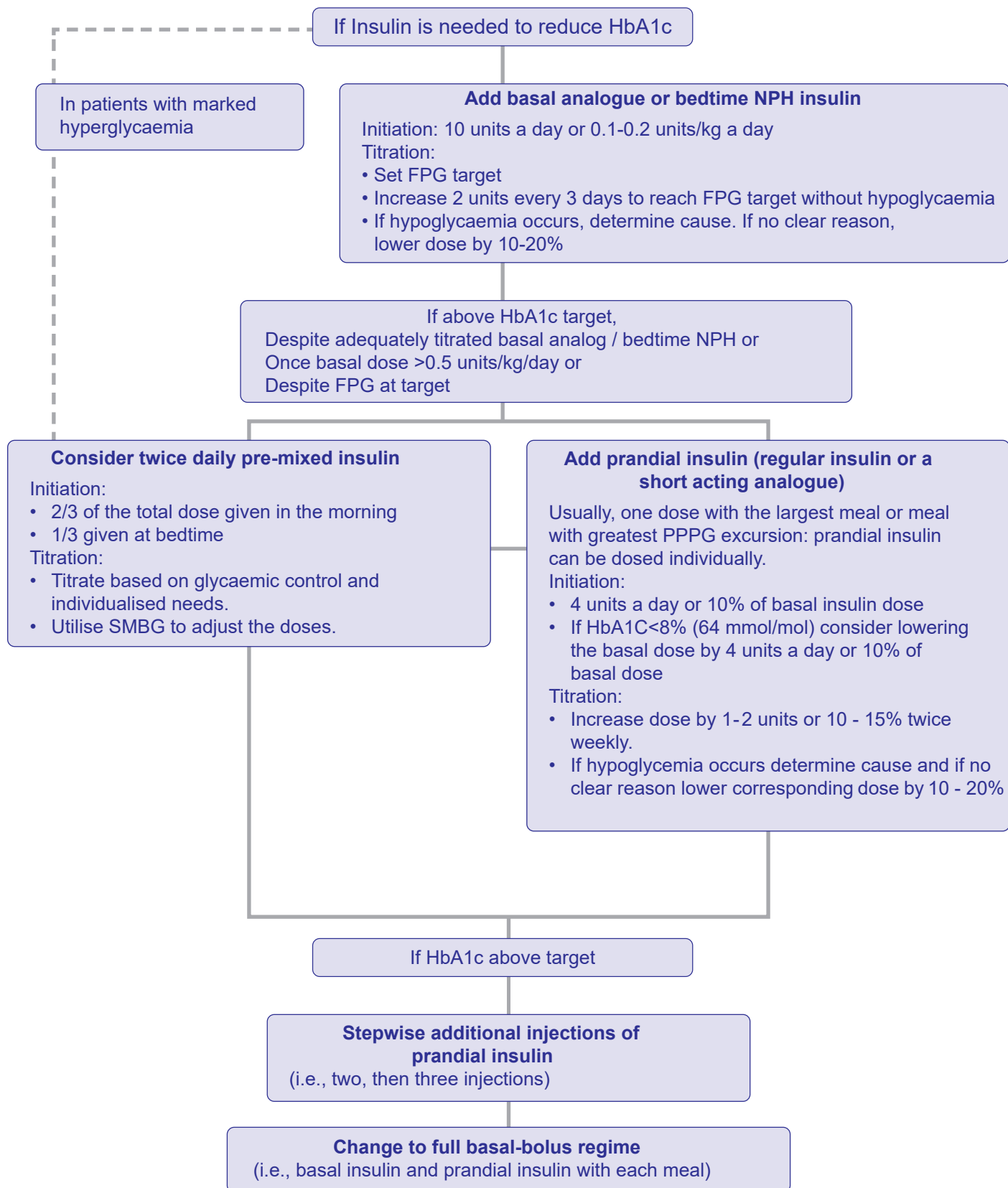


Figure 10.2 Approach to initiation and intensification of insulin in diabetes

Table 10.1 Details of non-insulin agents used in pharmacological therapy in T2D

Agent	Tablet strength	Maximum dose	Advantages	Adverse effects	Contraindications	Additional considerations
Metformin	500 mg 850 mg 1000 mg	2000 mg	Weight neutral No hypoglycaemia risk	GI side effects: Abdominal discomfort, reduced appetite, diarrhoea (usually transient), nausea, altered taste, vomiting	Renal insufficiency (eGFR <30 mL/min/ 1.73m ²) Acute acidosis (heart failure/ organ failure)	Caution in those at risk of a sudden deterioration in kidney function and those at risk of eGFR falling below 45 mL/min/1.73m ²
Sulphonylurea: Gliclazide (immediate release) Gliclazide (modified release) Glibenclamide Tolbutamide Glimepiride Glipizide	40 mg 80 mg 30 mg 60 mg 2.5, 5, 10 mg 500 mg 1, 2, 4 mg 5 mg	320 mg 120 mg 20 mg 2000 mg 6 mg 20 mg	Rapid onset of action	Weight gain, hypoglycaemic risk (especially with glibenclamide)	Diabetic ketoacidosis	Caution in the presence of renal impairment and elderly (particularly with second generation)
Thiazolidinedione: Pioglitazone	15, 30, 45 mg	45 mg	No hypoglycaemia risk Improved lipid profile Potential decrease in myocardial infarction	Weight gain, fractures, increased risk of infection, numbness, visual impairment	Heart failure Current, or a history of bladder cancer Un-investigated macroscopic haematuria	Caution in elderly (increased risk of heart failure, fractures, and bladder cancer) Increased risk of fractures, particularly in women
DPP-4 inhibitors: Linagliptin Sitagliptin Vildagliptin	5 mg 25, 50, 100 mg 50, 100, mg	5 mg 100 mg 100 mg	Weight neutral No hypoglycaemia risk	Uncommon Headache, dizziness, constipation, angio-oedema	Diabetic Ketoacidosis	Vildagliptin: caution if history of pancreatitis, avoid in severe heart failure. (Refer to table 12.6 for dosing in kidney disease)
SGLT-2 inhibitors: Dapagliflozin Canagliflozin Empagliflozin	5, 10 mg 100, 300 mg 10, 25 mg	10 mg 300 mg 25 mg	Weight loss No hypoglycaemia risk Reduction in systolic blood pressure Reduced cardiovascular mortality in patients with established ASCVD Improved renal outcomes in patients with nephropathy	Dapagliflozin: Back pain, balanoposthitis, euglycaemic ketoacidosis (discontinue immediately), dyslipidaemia, increased risk of infections. Canagliflozin: Fractures, lower limb amputations, acute kidney injury, DKA Empagliflozin: Vulvovaginal candidiasis Balanoposthitis Urinary tract infections Rarely Fournier's gangrene	Diabetic ketoacidosis	Caution in elderly (risk of volume depletion), elevated haematocrit, hypotension, risk of volume depletion, eGFR < 30 mL/ min/1.73 m ²
GLP-1 receptor agonist: Liraglutide subcutaneous solution multi-dose pen	18 mg/ 3 ml (doses of 0.6, 1.2, 1.8 mg)	1.8 mg	Weight loss Reduction in major adverse cardiovascular events No hypoglycaemia risk	Frequent GI side effects (gastrointestinal discomfort, altered taste, vomiting), headache, increased risk of infection, insomnia, skin reactions, toothache	Diabetic ketoacidosis Diabetic gastroparesis Inflammatory bowel disease	Caution in severe congestive heart failure, thyroid disease
Alpha-glucosidase inhibitors: Acarbose	25, 50, 100 mg	300 mg	Weight neutral	Frequent GI side effects (nausea, vomiting, diarrhoea, abdominal discomfort)	Inflammatory bowel disease, hernia (condition may deteriorate)	Avoid if eGFR < 30 mL/ min/ 1.73 m ²

Table 10.2 Insulin preparations available in Sri Lanka (2025)

Type of insulin	Generic Name	Available preparations
Ultra-short acting	Aspart	Novorapid® flexpen® 100 IU/ml (3 ml disposable pen)
	Glulisine	Apidra Solostar 100 IU/ml (3 ml disposable pen)
	Lispro	Humalog 100 IU/ml (3 ml penfill)
		Humalog KwikPen 100 IU/ml (3 ml disposable pen)
Short acting	Human soluble/ Human regular	Actrapid® HM Penfill® 100 IU/ml (3 ml penfill)
		Actrapid® HM Injection 100 IU/ml (10 ml vial)
		Humulin R 100 IU/ml (3 ml penfill)
		Wosulin R 100 IU/ml (3 ml penfill)
		Wosulin R 100 IU/ml (10 ml vial)
Intermediate acting	NPH (Isophane)	Humulin N Injection 100 IU/ml (3 ml penfill)
		Insulatard® HM Penfill® 100 IU/ml (3 ml penfill)
		Insulatard® HM Injection 100 IU/ml (10 ml vial)
		Wosulin N 100 IU/ml (3 ml penfill)
		Wosulin N 100 IU/ml (10 ml vial)
Long acting	Detemir	Levemir® flexpen® 100 IU/ml (3ml disposable pen)
	Glargine	Abasaglar 100 IU/ml Injection (3 ml penfill)
		Basalog 100 IU/ml (3 ml penfill)
		Glaritus 100 IU/ml (3 ml penfill)
		Lantus Solostar 100 IU/ml (3 ml disposable pen)
		Lantus 100 IU/ml (3 ml penfill)
Pre mixed	Isophane 70% + Human soluble 30%	Humulin 70/30 100 IU/ml (3 ml penfill)
		Insugen 30/70 100 IU/ml (10 ml penfill)
		Mixtard® 30 HM Penfill® 100 IU/ml (3 ml penfill)
		Mixtard® 30 HM Injection 100 IU/ml (10 ml vial)
		Wosulin 30/70 100 IU/ml (3 ml penfill)
		Wosulin 30/70 100 IU/ml (10 ml vial)
	Isophane 75% + Human soluble 25%	Insuman 25/75 (3 ml penfill)
	Protamine-crystallised aspart 70%+aspart 30%	Novomix® 30 flexpen® 100 IU/ml (3 ml disposable pen)
		Novomix® 30 Penfill® 100 IU/ml (3 ml penfill)
	Lispro protamine 75% + lispro 25%	Humalog Mix 25 100 IU/ml (3 ml penfill)
	Lispro protamine 50% + lispro 50%	Humalog Mix 50 100 IU/ml (3 ml penfill)
		Humalog Mix 50 KwikPen (3 ml disposable pen)

References

1. American Diabetes Association. Pharmacological approach to glycaemic control: Standards of Medical Care in Diabetes—2025. *Diabetes Care*, 2025.
2. Cosentino F, Grant PJ, Aboyans V et al., 2019 ESC Guidelines on diabetes, pre-diabetes, and cardiovascular diseases developed in collaboration with the EASD: the Task Force for diabetes, pre-diabetes, and cardiovascular diseases of the European Society of Cardiology (ESC) and the European Association for the Study of Diabetes (EASD). *European heart journal*, 2020. 41(2): p. 255-323.
3. Garber AJ, Handelsman Y, Grunberger G, et al. Consensus statement by the American association of clinical endocrinologists and American college of endocrinology on the comprehensive type 2 diabetes management algorithm – 2020 executive summary. *Endocrine Practice*. 2020;26(1):107-139.
4. Buse, J.B, Wexler DJ, Tsapas A, et al., 2019 update to: management of hyperglycemia in type 2 diabetes, 2018. A consensus report by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). *Diabetes care*, 2020. 43(2): p. 487-493.

11. CARDIOVASCULAR DISEASE AND RISK MANAGEMENT

A. Cardiovascular disease (CVD) related complications of diabetes

1. What are the CVD complications that occur in diabetes?

- Atherosclerotic cardiovascular disease (ASCVD)
 - History of acute coronary syndrome, myocardial infarction (MI), stable or unstable angina or coronary or other arterial revascularization, stroke, transient ischaemic attack (TIA), peripheral artery disease (PAD) including aortic aneurysm
- Heart failure
 - Heart failure with preserved ejection fraction (HFpEF)
 - Heart failure with mildly reduced ejection fraction (HFmEF)
 - Heart failure with reduced ejection fraction (EFrEF)

2. What are the risk factors for CVD in diabetes?

- Diabetes itself is an independent ASCVD risk factor
- Cardiometabolic risk factors – hypertension, hyperlipidemia and obesity.
- Smoking
- Family history of premature coronary disease
- Diabetic kidney disease (even mildly elevated albuminuria)

Starting from the state of insulin resistance to prediabetes and metabolic syndrome, the risk of CVD is increased

3. What is the impact of CVD in diabetes?

Compared to people without diabetes, people with diabetes have a higher risk of CVD related death.

B. Screening and preventive measures

Benefits of screening for CVD risk factors

- Helps to guide therapy
- Controlling individual cardiovascular risk factors helps to prevent or slow ASCVD
- Benefits are greater when multiple cardiovascular risk factors are addressed simultaneously

4. How to assess the CVD risk?

- Screen for CVD risk factors at the time of diagnosis, then at least annually
- Use WHO Southeast Asia cardiovascular disease risk chart (annexure 11.1) for risk estimation
- Use same measures even in people with prediabetes

5. How to detect CVD?

- Symptoms and signs
 - Chest pain, chest discomfort, unexplained dyspnoea, abnormal cardiac examination
 - History suggestive of stroke/ transient ischaemic attack, carotid bruits
 - Intermittent claudication, rest pain, tissue loss in lower limbs, absent or reduced lower limb pulsesRefer for evaluation if any of the above is present
- Resting ECG

6. How to prevent CVD in people with diabetes and prediabetes?

- A - HbA1c (blood glucose control)
- B - Blood pressure control <130/80 mmHg
- C - Cholesterol target
- D - Drugs to protect heart (antihypertensives, statins, antiplatelet agents, SGLT2i and GLP-1 RA)
- E - Exercise and Eating - regular physical activity, healthy eating and achieving and maintaining a healthy body weight
- S - Smoking cessation and Stress management

C. Blood pressure management

7. What is the importance of blood pressure control?

Early and effective treatment of high blood pressure,

- Prevents cardiovascular disease (CVD)
- Minimises the rate of progression of diabetic nephropathy and retinopathy

8. What is the target BP in people with diabetes?

- BP targets should be individualised depending on cardiovascular risk, potential adverse effects of antihypertensive medications, and patient preferences.

Table 11.1 Target blood pressure in individuals with diabetes

Group	Definition	Target
Higher cardiovascular risk	Existing ASCVD or 10-year ASCVD risk $\geq$ 15%	< 130/80 mmHg
Lower cardiovascular risk	10 - year ASCVD risk < 15%	< 140/90 mmHg
Older adults	> 65 years	<140/90 mmHg

9. What are the treatment strategies for hypertension?

- Lifestyle management
- Pharmacologic Interventions

10. What lifestyle interventions should be used to manage hypertension?

For patients with BP > 120/80 mmHg,

- Weight loss via caloric restriction – in overweight and obese patients
- Dietary Approaches to Stop Hypertension (DASH)-style eating pattern,
 - Plenty of vegetables and fruits (8 – 10 servings per day)
 - Whole grains
 - 1-2 servings of low fat milk
 - Modest amount of lean proteins- fish and poultry
 - Salt intake of < 5 g/day (sodium <2000 mg/day)
 - Increase potassium intake
 - Restrict meals such as red meat, sugar containing sweets or beverages
- Alcohol is best avoided
 - Advise to limit to less than 2 servings per day in men and less than 1 serving per day in women
- Increase physical activity
- Advise on adequate sleep and stress management

11. In addition to life style intervention, when to start antihypertensive medications?

- Confirmed office BP $\geq 130/80$ mmHg - Initiate pharmacological therapy and titrate to achieve recommended BP $\leq 130/80$ mmHg.
- Confirmed office BP $\geq 150/90$ mmHg - Initiate pharmacological therapy promptly and titrate to combination therapy (two drugs or a single pill combination of drugs.)

12. Which antihypertensive medications can be used in people with diabetes and hypertension?

Initial treatment for hypertension should include an agent of any drug class which is known to reduce cardiovascular events: ACEi, ARB, thiazide like diuretics or dihydropyridine calcium channel blockers.

Table 11.2 Suggested antihypertensive agents according to individual characteristics

Patient characteristics	Preferred agents
Established coronary artery disease, Albuminuria	Angiotensin converting enzyme inhibitor (ACEi), Angiotensin receptor blocker (ARB)
Prior MI, active angina, or HFrEF	Beta -blockers
Other patients	ACEi, ARBs, thiazide - like diuretics, long-acting dihydropyridine calcium channel blockers (CCB)

Avoid combinations of,
 • ACEi and ARB
 • ACEi/ ARB and direct renin inhibitors
 Due to high risk of hyperkalaemia, syncope, and acute kidney injury.

13. How to treat hypertension in prediabetes?

- Treat with renin-angiotensin-aldosterone system (RAAS) rather than beta-blockers/ diuretics to reduce the risk of new onset diabetes

14. What are the adverse effects of these antihypertensive medications and how to monitor?**Table 11.3** Adverse effects of antihypertensive agents and suggested monitoring

Antihypertensive	Adverse effects	Laboratory monitoring
Angiotensin converting enzyme inhibitors	Acute kidney injury Hyperkalaemia Dry cough	Serum creatinine and electrolytes - 1-2 weeks after starting or increasing dose and then at least annually
Angiotensin receptor blockers	Acute kidney injury Hyperkalaemia	
Thiazide diuretics	Acute kidney injury Hypokalaemia Hyponatraemia Hypomagnesaemia Hypercalcaemia	Serum electrolytes - before starting, first week of treatment and then at least annually Serum creatinine- before starting and then at least annually
Dihydropyridine calcium channel blockers	Flushing, headache, excessive hypotension, oedema and reflex tachycardia	Nothing specific

15. How to treat resistant hypertension?

- For individuals with hypertension who are not meeting blood pressure goals with three classes of antihypertensive medications (including diuretics) consider adding a mineralocorticoid receptor antagonist (MRA).
- Screen for secondary causes in people with suspected resistant hypertension. Liaise with endocrinology team for evaluation.

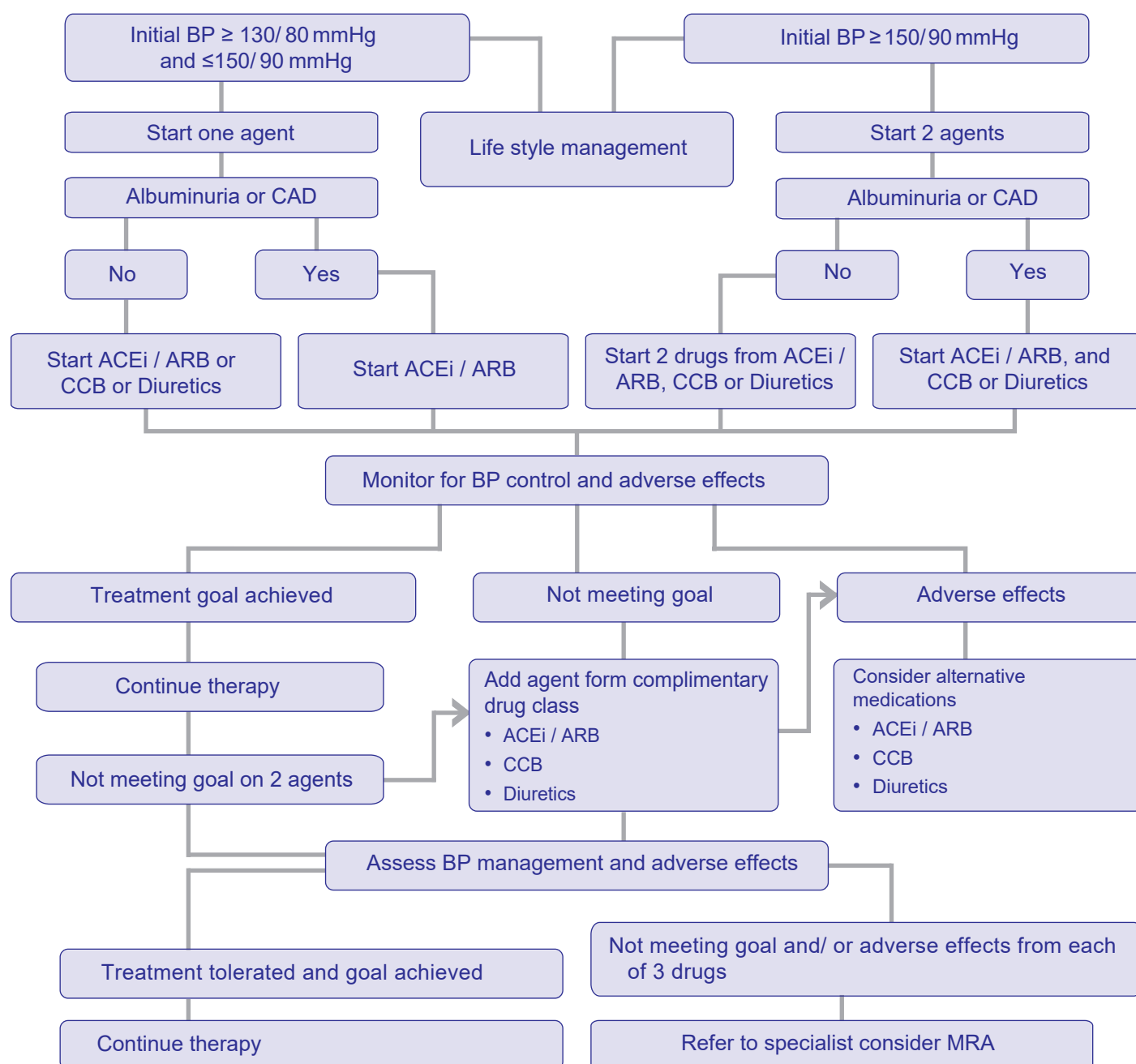


Figure 11.1 Approach to treatment of hypertension in diabetes

D .Lipid management and Statin Use

16. How to check for lipids?

- Fasting lipid profile (after 8 - 12 hours of fasting, water only) is recommended in the following situations:
 - Triglycerides >400 mg/dL (≥ 4.5 mmol/L) in a non-fasting sample
 - Suspected familial hyperlipidaemia or chylomicronemia
 - Prior to initiating fibrate therapy
- Non - fasting lipid profile is an acceptable alternative for routine screening and follow-up in other situations.

17. How to manage dyslipidaemia in people with diabetes?

- Lifestyle interventions
 - Dietary modification
 - Mediterranean or DASH eating pattern
 - Reduction of saturated fat and trans fat
 - Increase of dietary N-3 fatty acids, viscous fiber and plant
 - Stanol and sterol intake
 - Regular exercise
 - Weight reduction
 - Control of alcohol consumption
- Pharmacotherapy - statins

18. How to prescribe a statin to an individual with diabetes?

Table 11.4 Statin treatment for individuals with diabetes

Primary Prevention	40 – 75 years	Without ASCVD	Moderate intensity statin
		With one or more ASCVD risk factors	High intensity statin therapy with a target to reduce LDL cholesterol by > 50% of the baseline and LDL target of <70mg/dL (<1.8 mmol/L).
	>75 years	With ASCVD already on statin	Continue statin
		Without ASCVD	
	20 – 39 years	With additional ASCVD risk factors	Reasonable to start moderate intensity statin
Secondary prevention (established atherosclerotic cardiovascular disease)		All patients	High intensity statins to target an LDL cholesterol reduction of >50% from baseline and an LDL cholesterol goal of <55 mg/dL (<1.4 mmol/L).

- Moderate intensity statin therapy (lowers LDL - C by 30 – 49%)
 - Atorvastatin 10 - 20 mg
 - Rosuvastatin 5 - 10 mg
 - Simvastatin 20 - 40 mg
- High intensity statin therapy (lowers LDL - C by >50%)
 - Atorvastatin 40 - 80 mg
 - Rosuvastatin 20 - 40 mg

For individuals who do not tolerate the intended statin intensity, the maximum tolerated statin dose should be used with additional lipid lowering medications if necessary.

19. What are the LDL - C targets?

Table 11.5 LDL - C targets for people with diabetes

Cardiovascular risk	Patient characteristics	LDL-C target
Very high risk	Established CVD or Other target organ damage (proteinuria, eGFR <30 mL/min/1.73 m ² , left ventricular hypertrophy, retinopathy) or Three or more major risk factors (age, hypertension, dyslipidaemia, smoking, obesity) or Early onset T1D of long duration (>20 years)	<55 mg/dL and >50% reduction from baseline
High risk	Patients with diabetes duration >10 years without target organ damage plus any other additional risk factor (age, hypertension, dyslipidaemia, smoking, obesity)	<70 mg/dL and >50% reduction from baseline
Moderate risk	Young patients (T1D aged <35 years or T2D aged <50 years) with diabetes duration <10 years, without other risk factors	<100 mg/dL

20. What should be done if the targets are not met?

- Discuss adherence to lifestyle interventions
- Discuss adherence to medications
- Consider increasing the dose of statin if not started on the specified dose mentioned above
- Addition of new lipid lowering drugs eg., ezetimibe 10 mg daily

21. How to treat high triglyceride levels in people with diabetes?

- Exclude secondary causes of high TG

Table 11.6 Management of triglycerides in people with diabetes

Category	Triglyceride level (mg/dL)	Management focus
Normal	<150	No specific therapy needed
Borderline	150 - 174	Reduce CVD risk by reduction of LDL - C: 6 months trial of lifestyle modification followed by addition of a statin
High	175 - 499	Reduce CVD risk by reduction of non - HDL - C: Restrict dietary fat to 30% of total calories Intensify statin therapy Consider starting a fibrate (fenofibrate)*
Very high	>500	Evaluate for secondary causes of hypertriglyceridemia and consider medical therapy to reduce the risk of pancreatitis.

*In men with triglycerides >200 mg/dl HDL <35 mg/dL

22. How to monitor individuals for adverse effects during treatment with lipid-lowering therapy?

Liver enzyme elevation

- ALT before treatment and in three months after initiation.

Table 11.7 Approach to abnormal liver enzymes in people on/ awaiting statin therapy

Liver enzyme level	Decision of starting statin if found during baseline	Proposed action if found during follow up
ALT < 3 × ULN	Investigate for an aetiology Not contraindicated in compensated cirrhosis and in Non-alcoholic fatty liver disease (NAFLD) /Nonalcoholic Steatohepatitis (NASH). Contraindicated in decompensated cirrhosis or acute liver failure	Continue therapy Recheck liver enzymes in 4 - 6 weeks
ALT > 3 × ULN	Avoid starting statin Evaluate for a cause	Stop or reduce the dose of lipid lowering therapy Recheck liver enzymes within 4 - 6 weeks Consider cautious re-introduction after ALT returns to normal If ALT remains elevated, check for other reasons

ULN- upper limit of the normal

Statin-associated muscle symptoms

- Check creatine kinase (CK) if the individual develops myalgia while on treatment.
- Consider concomitant vitamin D deficiency.

Table 11.8 Approach to abnormal muscle enzymes in people on statin therapy

Level of CK	Muscle symptoms	Plan of management
>4 × ULN	Not applicable	Stop statin Monitor CK for 6 weeks/ until normalisation Re-challenge with a lower dose or alternate day or once/ twice weekly dosing regimen
<4 × ULN	Absent	Continue therapy Monitor CK in 2 - 6 weeks
	Present	Stop statin Monitor for normalisation of CK, if symptoms persist look for alternative cause Re-challenge with a lower dose or alternate day or once/ twice weekly dosing regimen

ULN- upper limit of the normal

E. Anti-platelet therapy

23. When should aspirin be prescribed to an individual with diabetes?

- For secondary prevention in patients with atherosclerotic cardiovascular disease
- Aspirin may be considered as primary prevention strategy in those with diabetes who are at increased cardiovascular risk, after weighing the benefit to risk ratio.

24. What is the dose recommended for secondary prevention?

Aspirin 75mg daily

F. Glucose lowering agents for cardiovascular risk reduction

Numerous large randomised trials have shown statistically significant reductions in cardiovascular events with the following SGLT2i and GLP-1 RA in people with already established cardiovascular disease.

• SGLT2i

Empagliflozin

Canagliflozin

Dapagliflozin

• GLP-1 RA

Liraglutide

Dulaglutide

Semaglutide

References

1. American Diabetes Association. Cardiovascular disease and risk management: Standards of Medical Care in Diabetes—2025. *Diabetes Care*, 2025, 44(Supplement 1): p. S125-S150.
2. Mach F, Baigent C, Catapano AL, et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk.
3. *European Heart Journal*. 2020 Jan 1;41(1):111–188. doi:10.1093/eurheartj/ehz455
4. The 2018 ACC/AHA Cholesterol Guideline specifies these categories and dosage thresholds, defining moderate intensity as 30–49% LDL C reduction, and high intensity as $\geq 50\%$

12. MICROVASCULAR COMPLICATIONS

1. What are the risk factors for microvascular complications?

- Degree of control and duration of glycaemia & blood pressure
- Dyslipidaemia
- Presence of other complications
- Smoking
- Increasing age and pregnancy
- Genetic predisposition

2. When to screen for microvascular complications?

- At the diagnosis and annually
- Monitor more frequently, if abnormalities are detected at the screening

3. How to prevent microvascular complications?

- Intensive glycaemic control with near normal glycaemia aiming at HbA1c <7%
- Blood pressure control with a target blood pressure of <140/90 mmHg
- Stringent lipid management
- Smoking cessation

A. Diabetic kidney disease (DKD)

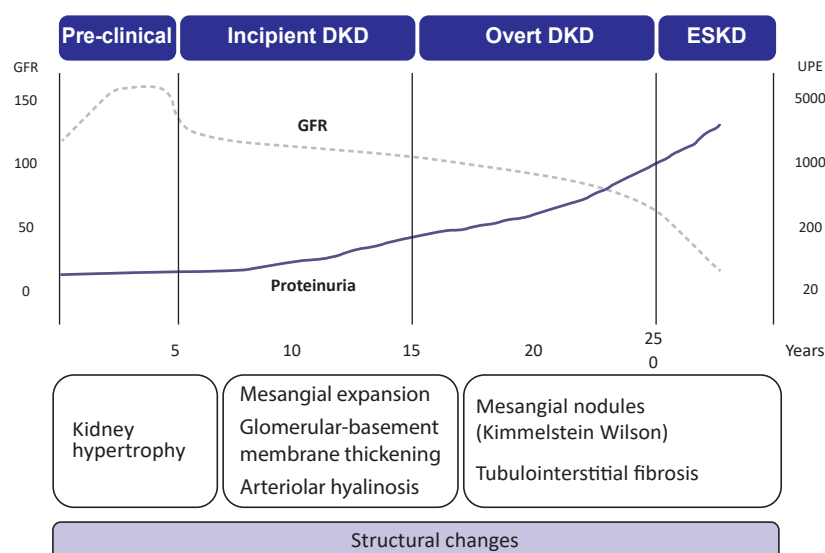
4. What is DKD?

DKD is defined as albuminuria and/or progressive reduction in estimated glomerular filtration rate (eGFR) in the setting of long duration of diabetes in the absence of signs or symptoms of other primary causes of kidney damage.

5. How common is DKD?

- DKD is the primary etiology of end stage kidney disease (ESKD) in individuals with diabetes, accounting for 30 – 50% of ESKD cases globally and approximately 40% of individuals with diabetes develop DKD.
- In Sri Lanka 10% of the population, approximately 2.1 million are affected by CKD and among them 35.9% is due to DKD.
- The prevalence of nephropathy at diagnosis of diabetes and at 16 - 20 years was 18.8% and 23.8% respectively.

6. What is the natural history of DKD?



GFR: glomerular filtration rate (mL/min/1.73m²), UPE: urine protein excretion (mg/day)

Figure 12.1. Natural progression of diabetic kidney disease

- The rate of decline of eGFR increases with degree of albuminuria. Those with moderately increased albuminuria shows an eGFR decline of 2.3 mL/min/1.73 m²/year, whereas those with severely increased albuminuria show an eGFR decline of 3.7 mL/min/1.73 m²/year to 5.4 mL/min/1.73 m²/year.

KDIGO: Prognosis of CKD by GFR and albuminuria categories				Persistent albuminuria categories		
				Description and range		
				A1	A2	A3
				Normal to mildly increased <30 mg/g <3 mg/mmol	Moderately increased 30–300 mg/g 3–30 mg/mmol	Severely increased >300 mg/g >30 mg/mmol
GFR categories (mL/min/1.73 m ²) Description and range	G1	Normal or high	≥90			
	G2	Mildly decreased	60–89			
	G3a	Mildly to moderately decreased	45–59			
	G3b	Moderately to severely decreased	30–44			
	G4	Severely decreased	15–29			
	G5	Kidney failure	<15			

Green: low risk (if no other markers of kidney disease, no CKD); Yellow: moderately increased risk; Orange: high risk; Red: very high risk. GFR, glomerular filtration rate.

Figure 12.2 Current Chronic Kidney Disease nomenclature used by KDIGO

Source : KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease

7. How to screen for DKD?

- Assess kidney function (i.e., spot urine albumin-to-creatinine ratio [UACR]), creatinine and estimated glomerular filtration rate [eGFR] in people with type 1 diabetes with duration of ≥5 years and in all people with type 2 diabetes regardless of duration.
- At least annually, urinary albumin (e.g., spot UACR) and eGFR should be assessed.
 - If UACR >30 mg/g,
 - Consider urinalysis
 - Repeat UACR in 3 months
- Serum creatinine and estimated glomerular filtration rate (eGFR; using CKD EPI formula preferably)
- Consider ultrasound scan if:
 - Rapid decline in eGFR
 - Severe proteinuria equivalent to >300 mg/g of albumin excretion rate or >500 mg/g proteinuria
 - Recurrent urinary tract infections
 - Suspected obstructive uropathy/ nephrolithiasis/ hereditary kidney disease
 - Active urinary sediment in urinalysis
 - Refractory hypertension

Albuminuria Screening

- Perform when person is free from acute illness and in stable glucose control, as acute illness and hyperglycaemia can transiently increase albuminuria.
- Albuminuria may increase in the upright posture and with exercise, thus measurements are best made in an early morning urine sample, however a spot urine sample is acceptable if there is no alternative.
- If first sample is abnormal, further sample should be obtained, within 1–3 months. At least two out of three should be abnormal before diagnosing albuminuria.

8. When to exclude secondary etiology?

Referral to a nephrologist for further diagnosis, including the possibility of kidney biopsy, should be considered.

- If proteinuria is present before 5 years of T1D duration
- In the presence of active urinary sediment (haematuria, cellular casts, etc.)
- Rapidly increasing albuminuria or total proteinuria
- The presence of nephrotic syndrome
- Rapid decline in eGFR.
- Absence of retinopathy (in type 1 diabetes)
- If there are signs or symptoms of other systemic diseases.
- Refractory hypertension.
- Chronically well-managed blood glucose.
- Suspected obstructive uropathy/ nephrolithiasis/ hereditary kidney disease

9. How to monitor DKD?

In people with established chronic kidney disease (CKD), monitor urinary albumin (e.g., spot UACR) and eGFR 1–4 times per year depending on the stage of kidney disease.

CKD is classified based on: • Cause (C) • GFR (G) • Albuminuria (A)				Albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30–299 mg/g 3–29 mg/mmol	≥300 mg/g ≥30 mg/mmol
GFR categories (ml/min/1.73 m ²) Description and range	G1	Normal or high	≥90	Screen 1	Treat 1	Treat 3
	G2	Mildly decreased	60–89	Screen 1	Treat 1	Treat 3
	G3a	Mildly to moderately decreased	45–59	Treat 1	Treat 2	Treat 3
	G3b	Moderately to severely decreased	30–44	Treat 2	Treat 3	Treat 3
	G4	Severely decreased	15–29	Treat* 3	Treat* 3	Treat 4+
	G5	Kidney failure	<15	Treat 4+	Treat 4+	Treat 4+
				Low risk (if no other markers of kidney disease, no CKD)	High risk	Very high risk
				Moderately increased risk		

Figure 12.3 Frequency of monitoring glomerular filtration rate (GFR) and albuminuria in people with chronic kidney disease (CKD). Albuminuria and GFR grid reflects the risk of progression by intensity of coloring (green, yellow, orange, red, and deep red). The numbers in the boxes are a guide to the frequency of monitoring (number of times per year).

Table 12.1 Complications of DKD and relevant physical and laboratory evaluations.

Complication	Physical and laboratory evaluation
Blood pressure	Blood pressure
Volume overload	History, physical examination, weight
Electrolyte abnormalities	Serum electrolytes
Metabolic acidosis	Serum electrolytes
Anaemia	Hemoglobin, serum iron, transferrin saturation and ferritin
Metabolic bone disease	Serum calcium, phosphate, Vitamin D

10. Holistic approach for improving outcomes of diabetic kidney disease

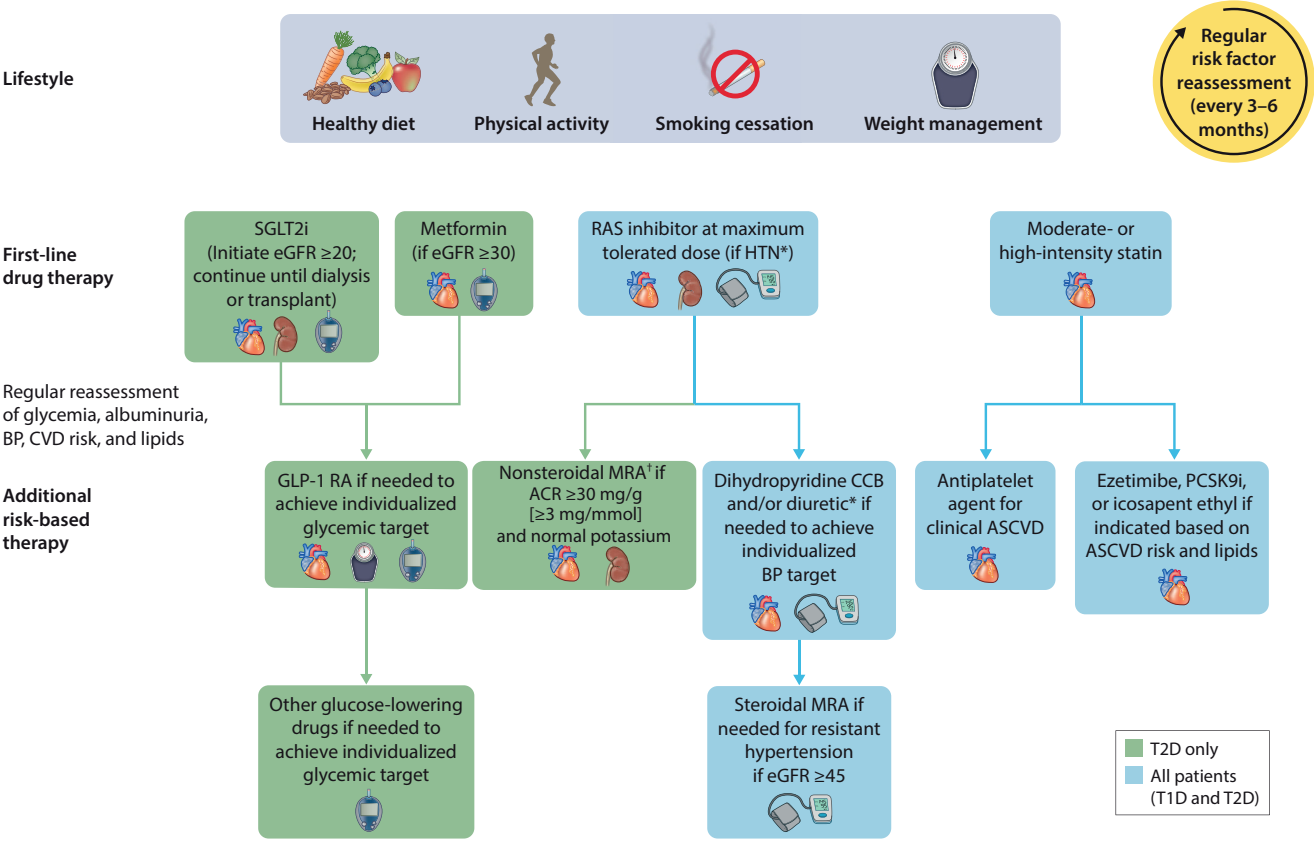


Figure 12.4 Holistic approach to chronic kidney disease (CKD) treatment and risk modification.
*Angiotensin-converting enzyme inhibitor or angiotensin II receptor blocker should be first-line therapy for blood pressure (BP) control when albuminuria is present, otherwise dihydropyridine calcium channel blocker (CCB) or diuretic can also be considered.

Source: KDIGO 2022 Clinical Practice Guideline for Diabetes Management in Chronic Kidney Disease. *Kidney Int.* 2022;102:S1–S127.2

11. How to manage hypertension in DKD?

Table 12.2 Management of hypertension in people with DKD and albuminuria.

Agents	First line	Angiotensin converting enzyme inhibitors (ACEi) or Angiotensin receptor blockers (ARB)
	Second line	Dihydropyridine calcium channel blockers Cilnidipine Aldosterone receptor antagonists
Target	<130/80 mmHg	
Monitoring	Serum creatinine, eGFR and electrolytes before and 2-4 weeks after starting ACEi or ARBs	If there is >30% rise in serum creatinine or >25% decrease in eGFR; evaluate for renal artery stenosis Manage hyperkalaemia by dietary modifications first Reduce or stop ACEi/ ARBs as the last resort

Table 12.3 Management of hypertension in people with DKD without albuminuria

Agents	First line	ACEi or ARB Dihydropyridine CCB Thiazide diuretics
Blood pressure targets	<140/90 mmHg	
Monitoring	Refer to table 12.2	

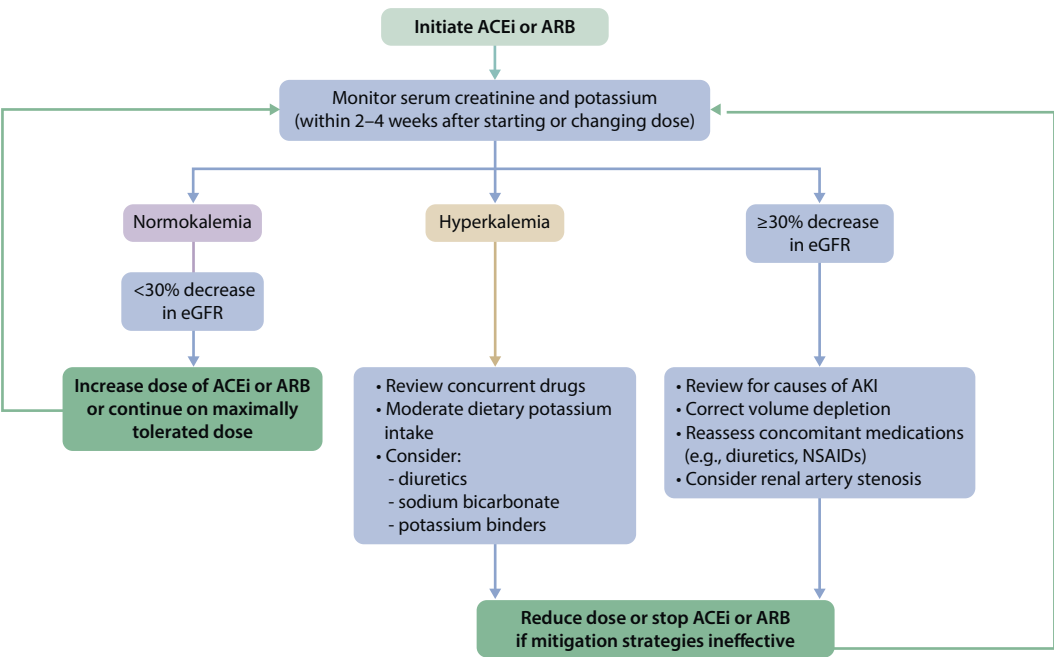


Figure 12.5 Algorithm for monitoring of potassium and estimated glomerular filtration rate (eGFR) after the initiation of renin-angiotensin system inhibitors

Source: KDIGO 2022 Clinical Practice Guideline for Diabetes Management in Chronic Kidney Disease. *Kidney Int.* 2022;102: S1–S127.2

Table 12.4 Drugs used in the management of DKD

Class	Drug	Starting dose	Maximum daily dose	Kidney impairment
ACE inhibitors	Captopril	12.5 mg to 25mg 2 to 3 times daily	Usually 50mg 3 times daily (may go up to 450mg/day)	Half-life is increased in patients with kidney impairment eGFR 10 - 50ml/min: administer 75% of normal dose every 12 - 18 hours eGFR <10ml/min: administer 50% of normal dose every 24 hours Hemodialysis: administer after dialysis. About 40% of the drug is removed by HD.
	Enalapril	5mg once daily	40mg	eGFR ≤30ml/min: Reduce initial dose to 2.5mg once daily 2.5mg PO after HD on dialysis days: dosage on non-HD days should be adjust according to clinical response.
	Perindopril	2mg once daily	8 mg	Use not recommended when eGFR ≤30ml/min. Perindopril and its metabolites are removed by HD.
	Ramipril	2.5mg once daily	20mg	Administer 25% of normal dose when (eGFR < 40ml/min.) minimally removed by HD.
	Lisinopril	10mg once daily	40mg	eGFR 10 - 30 ml/min: reduce initial dose by 50% eGFR <10ml/min :reduce initial dose to 2.5mg once daily
Angiotensin Receptor blockers	Losartan	50mg once daily	100mg	No dosage adjustments.
	Telmisartan	40mg once daily	80mg	No dosage adjustments
	Irbesartan	150mg once daily	300 mg	No dosage adjustments.
	Candesartan	16mg once daily	32mg	eGFR <30 min/ml consider reduction
ns- MRA	Finrenone	10mg daily	20mg daily	Serum K monitoring

12. What are the specific measures for glycaemic control in DKD?

- Intensive glycaemic control
 - However, individualise according to needs, associated comorbidities and stage of DKD
- SGLT2i preferred as the first line agent
- GLP-1 RA considered as an alternative

Table 12.5 Management of hyperglycaemia in DKD

Therapies	1st Line: SGLT2 inhibitors and/ or Metformin 2nd Line: GLP-1 receptor agonists or other glucose-lowering agents (based on eGFR, availability, and individual needs)
Target	Individualized HbA1c target < 6.5% to <8.0% in patients with diabetes and CKD not treated with dialysis
Monitoring	Measure long term glycaemic control by HbA1c at least twice per year- More frequent monitoring if, • Glycemic target not met • Therapy changes • Comorbidities present Accurate HbA1c measurement decline with advanced CKD (G4 – G5), use FBG/ PPBS

Table 12.6 Modifications of medications in diabetes according to eGFR

Medication	Recommendations based on eGFR
Metformin	Consider discontinuation if eGFR < 30 mL/min or on dialysis Reduce dose if eGFR <45 mL/min May initiate between eGFR 30-44 mL/min and may titrate upwards to half of the maximum dose
SGLT2i	Practice caution if eGFR <30 mL/min Not recommended in patients on dialysis
GLP-1 RA	Stop exenatide if eGFR < 30 mL/min Stop other GLP - 1 RA if eGFR <15 mL/min
DPP-4i	Linagliptin: no dose adjustment Sitagliptin • eGFR 30 – 50 mL/min: maximum dose 50 mg/day • eGFR < 30 mL/min: maximum dose 25 mg/day Vildagliptin • eGFR < 60 mL/min: maximum dose 50 mg/day Saxagliptin • eGFR 15 - 30 mL/min: maximum dose 2.5 mg/day • eGFR < 15 mL/min: stop
Sulphonylurea	Practise caution due to risk of hypoglycaemia
Alpha - glucosidase inhibitors	Stop if eGFR < 30 mL/min
Thiazolidinedione	Practise caution due to risk of pulmonary oedema

13. What are the specific measures in nutrition in DKD?

- Protein intake
 - Stages 3 – 5 ND CKD, 0.8 g/kg/day.
 - Intake < 0.8 g/kg/day is not recommended.
 - Low-protein eating patterns should only be followed alongside guidance from a health care professional experienced in managing nutrition for people with CKD.
 - Could consume between 1.0 and 1.2 g protein/kg /d if on HD or PD. (>1.3 g/kg/day have been associated with more rapid kidney function loss.)
- Sodium intake
 - Sodium recommended intake is < 2 g of per day (or <90 mmol of sodium per day, or < 5 g of sodium chloride per day)
 - Dietary sodium intake should be individualized based on comorbid conditions, medication use, blood pressure, and laboratory data.
- Potassium Intake
 - Individualization of dietary potassium may be necessary to control serum potassium concentrations.
- Exercise
 - Advised to undertake moderate-intensity physical activity for a cumulative duration of at least 150 minutes per week, or to a level compatible with their cardiovascular and physical tolerance.

14. When to screen for complications of DKD?

- The prevalence of DKD complications correlates with eGFR.
- When eGFR is <60 mL/min/1.73 m², screening for complications of DKD is indicated.
- Anaemia
 - Once the eGFR is <60ml/min/1.73m² start screening for anaemia in DKD.
 - Do full blood count at annual screening and if suboptimal more frequent monitoring with Iron studies including transferrin saturation and serum iron levels more frequently.
 - Exclude secondary causes for anemia. (e.g., ongoing blood loss/ hematological disorders etc.)
 - If suboptimal start on iron therapy with a referral to a nephrologist for initiation of erythropoietin.
- Mineral bone disease:
 - Do calcium and phosphate levels annually when eGFR is <60ml/min/1.73m².
 - Assess vitamin D status.
 - If sub optimal more frequent monitoring after exclusion of secondary causes,
 - PTH levels only if indicated. (e.g., suspicion of co-existing primary hyperparathyroidism, suspicion of adynamic bone disease etc.)
 - Bone density scan (DEXA scan) is not routinely indicated.
 - Start calcium and 1 alpha cholecalciferol for the management of hyperphosphatemia and secondary hyperparathyroidism respectively.
 - Refer to a nephrologist once suspicion of metabolic bone disease is present

15. When to refer to a nephrologist?

- Uncertainty about the etiology of kidney disease
- Difficult management issues (significant increases in albuminuria despite good blood pressure management, electrolyte disturbances, anaemia, secondary hyperparathyroidism, metabolic bone disease or resistant hypertension).
- Advanced kidney disease (eGFR < 30 mL/min/1.73 m²) requiring discussion of renal replacement therapy for ESKD.

Consultation with a nephrologist when stage 4 CKD develops (eGFR < 30 mL/min/1.73 m²) has been found to reduce cost, improve quality of care, and delay dialysis

B. Diabetes-related eye disease**16. How is diabetes related eye disease classified?**

Common types of diabetes related eye diseases include,

- Diabetic retinopathy (DR)
- Diabetic macular oedema (DME)
- Cataract
- Glaucoma

17. What is diabetic retinopathy?

Diabetic retinopathy (DR) is a common, highly specific progressive potentially sight-threatening neurovascular complication of diabetes

Chronic hyperglycemia which activates multiple pathways leading to abnormal vascular permeability, occlusion with ischemia and subsequent neovascularization drives the development and progression of DR.

18. What are the factors that increase the risk of, or are associated with, retinopathy ?

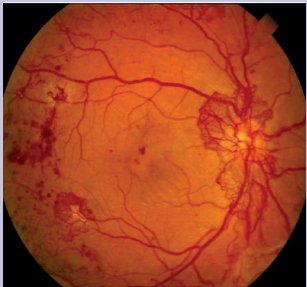
- Duration of diabetes
- Chronic hyperglycaemia
- Nephropathy
- Hypertension
- Dyslipidaemia

19. How common is DR?

Diabetic retinopathy (DR) is the commonest cause of preventable visual loss in the working group and the leading contributor to moderate to severe visual impairment.

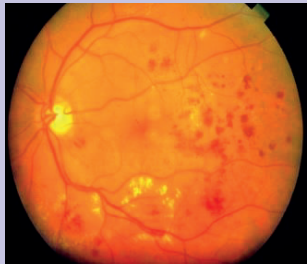
One third of people with diabetes suffer from DR.

Table 12.7 Classification of diabetic retinopathy and macular oedema

Diabetic retinopathy		
No apparent retinopathy		No abnormalities
Mild non-proliferative diabetic retinopathy (NPDR)		Microaneurysms only
Moderate non-proliferative diabetic retinopathy (NPDR)		More than just microaneurysms, but not meeting the criteria for severe NPDR
Severe non-proliferative diabetic retinopathy (NPDR)		Any one of the following findings with NO evidence of proliferative retinopathy • > 20 intraretinal haemorrhages in all 4 retinal quadrants • Venous beading in ≥ 2 quadrants • Intraretinal microvascular abnormalities in ≥ 1 quadrant
Proliferative diabetic retinopathy (PDR)		Severe NPDR and one or more of the following • New vessel formation • Vitreous / pre-retinal haemorrhage • Tractional retinal detachment

To be cont.

Table 12.7 cont.

Macular oedema		
Stage of macular oedema	Findings on dilated ophthalmoscopy	
No apparent DME		No retinal thickening or hard exudates in posterior pole
DME that does not involve the centre of the macula		Retinal thickening or hard exudates in posterior pole but not involving the centre of the macula
DME that involves the centre of the macula		Retinal thickening or hard exudates involving the centre of the macula

20. Components of DR screening?

- Visual acuity
- Colour vision
- Dilated fundoscopy / retinal photography
- Slit lamp examination
- Intra-ocular pressure

In resource limited setting, refer to ophthalmology services for comprehensive assessment.

21. When to screen for DR?

- Adults with type 1 diabetes within 5 years of diagnosis
- Adults with type 2 diabetes at the diagnosis
- If there is no evidence of DR 1-2 yearly screening

22. How to manage risk factors in diabetes retinopathy?

- Glycemic control
 - Greater benefits with a better glycaemic control (based on general clinical background)
 - One percent decrease in HbA1c reduces the incidence by 35% and reduces the progression by 15 - 25%.
 - GLP - 1 RA associated with reducing intraocular pressure and reduced risk of glaucoma
- Blood pressure control
 - For most, BP < 140/90 mmHg; for individuals at high risk of CVD, < 130/80 mmHg
 - Strict blood pressure control < 120/80 mmHg does not impart additional benefit.
 - Recommend ACEI or ARB.
- Lipid management
 - Addition of fenofibrate slows early diabetic retinopathy at baseline
 - The presence of retinopathy is not a contraindication to aspirin therapy for cardioprotection, as aspirin does not increase the risk of retinal haemorrhage

23. How to manage DR?

Table 12.8 Management of diabetic retinopathy and macular oedema

Stage of diabetic retinopathy	Management advice
No apparent retinopathy	Optimise modifiable risk factors (glucose, BP, lipids) Annual screening
Mild non-proliferative diabetic retinopathy (NPDR)	As above
Moderate non-proliferative diabetic retinopathy (NPDR)	Referral to an ophthalmologist Screen in 6 - 12 months Monitor for maculopathy
Severe non-proliferative diabetic retinopathy (NPDR)	Early referral to an ophthalmologist
Proliferative diabetic retinopathy (PDR)	Urgent referral to an ophthalmologist
Stage of macular oedema	Management advice
No apparent DME	Optimise modifiable risk factors (glucose, BP, lipids) Annual screening
DME that does not involve the centre of the macula	Referral to an ophthalmologist to be seen within 2-3 months
DME that involves the centre of the macula	Urgent referral to an ophthalmologist

- In pre-existing diabetes in pregnancy, if retinopathy is not present prior to pregnancy, rescreening is needed at 28 weeks. If retinopathy is present at the onset of pregnancy, screening is needed at 16-20 weeks, and 28 weeks by an ophthalmologist

24. What is the specific management of DR?

- Photocoagulation surgery

Panretinal photocoagulation is indicated to reduce the risk of vision loss in individuals with

- High-risk PDR
- Severe nonproliferative diabetic retinopathy

Macular focal/ grid laser photocoagulation technique is effective in treating clinically significant macular oedema in diabetes

- Anti VEGF therapy
- Effective alternative for PDR to reduce vision loss
- First-line treatment for most eyes with diabetic macular oedema that involves the foveal center and impairs visual acuity
- Five anti VEGF agents available – Bevacizumab, Ranibizumab, Aflibercept, Brolucizumab and Faricimab
- Vision rehabilitation
- Can help people with vision loss achieve maximum function, independence, and quality of life.

C. Diabetic Neuropathy

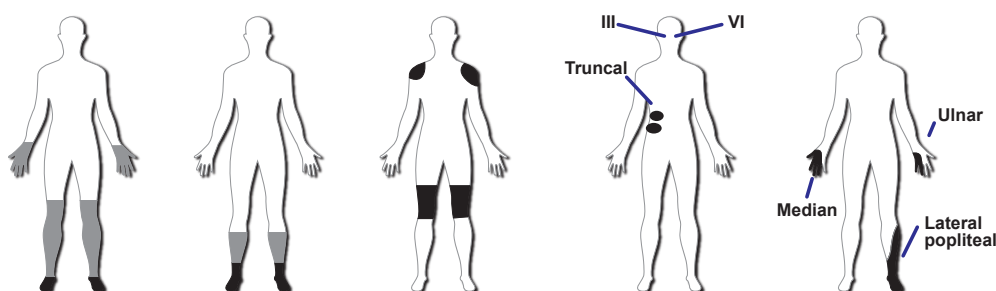
25. What is diabetic neuropathy?

Diabetic neuropathy is defined as a heterogeneous group of disorders with signs and symptoms of peripheral nerve dysfunction in people with diabetes in whom other cases of peripheral nerve dysfunction have been excluded.

26. How common is diabetic neuropathy?

- Diabetic neuropathy is the leading cause of non-traumatic limb amputation in Sri Lanka and worldwide.
- Prevalence of diabetic neuropathy among people with diabetes is around 50%
- Up to 50% of diabetic peripheral neuropathies may be asymptomatic.

27. How to classify diabetic neuropathy?



	Large fibre neuropathy	Small fibre neuropathy	Proximal motor neuropathy	Acute mononeuropathies	Entrapment neuropathies
Sensory loss	0 to +++ (touch / vibration)	0 to + (pain / temperature)	0 to +	0 to +	+ to +++
Pain	+ to +++	+ to +++	+ to +++	+ to +++	+ to ++
Motor deficit	0 to +++	No	+ to +++	+ to +++	+ to +++
Tendon reflexes	Normal – absent	Normal / diminished	Diminished	Normal	Normal

Figure 12.6 Classification of diabetic peripheral neuropathy

Source: 'Diabetic Microvascular Disease: An Endocrine Society Scientific Statement' by Barrett EJ, Liu Z, Khamaisi M et al, *The Journal of Clinical Endocrinology & Metabolism*, 2017

28. How to screen for diabetic neuropathy?

- History

Small fiber – pain

Large fiber – imbalance, numbness, loss of protective sensation

- Examination

Small-fiber function: pinprick and temperature sensation.

Large-fiber function: lower-extremity reflexes

vibration perception (128-Hz tuning fork or biothesiometer)

protective sensation (10-g monofilament)

29. What are the clinical features of diabetic distal sensory motor polyneuropathy (DSPN)?

- Symptoms:
 - Negative symptoms: numbness
 - Positive: symptoms (eg., "asleep numbness," prickling or stabbing, burning or aching pain) predominantly in the toes, feet, or legs
- Signs:
 - Symmetric decrease of distal sensation
 - Decreased or absent ankle reflexes

30. When to consider referral for electrophysiological testing or to a neurologist?

- When diagnosis is unclear or different aetiology is suspected
 - Motor greater than sensory neuropathy
 - Rapid onset
 - Asymmetrical presentation
 - Upper limb predominant involvement
 - Features of another underlying cause

31. How to manage diabetic distal sensory motor polyneuropathy

- Glycaemic control
 - Near normal glycaemia early achievement effectively delays or prevent development of diabetic peripheral neuropathy (DPN) and CAN in T1D
- Modify risk factors
- Weight management
 - Obesity is consistently associated with neuropathy
 - Weight loss by dietary and exercise interventions lead to improvement in neuropathy symptoms
- Dyslipidaemia management
 - Dyslipidaemia is a key factor in the development of neuropathy in diabetes
 - Lipid-lowering pharmacotherapy does not appear to be effective in treating or preventing DPN development.
- Blood pressure management
 - Hypertension is an independent risk factor for DPN development
 - Intensive blood pressure management also decreased the CAN risk by 25%
- Assess fall risk and take measures to prevent falls
- Assess quality of life (e.g., poor sleep)
- Pain management

Table 12.9 Pharmacological agents in the management of painful diabetic neuropathy

Drug class	Agent	Dose	Side effect
Gabapentinoids	Gabapentin	Starting dose: 100-300 mg, 1-3 times/day Effective dose: 900-3600 mg/day	Somnolence, Dizziness, Ataxia, Fatigue
	Pregabalin	Starting dose: 25 - 75 mg, 1-3 times/day Effective dose: 300-600 mg/day	Somnolence, Dizziness, Peripheral oedema, Headache Ataxia, Weight gain
Serotonin - norepinephrine reuptake inhibitors (SSRI)	Duloxetine	Starting dose: 20 - 30 mg/day Effective dose: 60 - 120 mg/day	Somnolence, Dizziness, GI side effects, Fatigue, Insomnia, Decreased libido
	Venlafaxine	Starting dose: 37.5 mg/day Effective dose: 75 - 225 mg/day	Same as above

Tricyclic antidepressants (TCA)	Amitriptyline	Starting dose: 10 - 25 mg/day Effective dose: 25 - 100 mg/day	Xerostomia, Somnolence, Headache, Dizziness, Insomnia, Orthostatic hypotension
Sodium channel blockers	Lamotrigine	Starting 25 mg/day and increase 2 weekly up to 400mg/day	Aseptic meningitis, Blood dyscrasias, Allergic reactions, HLH, GI effects
	Carbamazepine	200 - 400 mg/day in 2 - 4 doses Maximum dose 1.8g/day	Blood dyscrasias, Heart failure, Sinus tachycardia, Hepatotoxicity, Allergic reaction
	Oxcarbazepine	300 - 600mg/day Maximum 1.8g/day	Cytopenias, Allergic reactions, Hyponatremia, Neuropsychiatric effects, Nausea, Vomiting
Capsaicin	Capsaicin 8% patch	topical use 3 - 4 times a day	Burning, Itching, Dryness, Redness or swelling at the site of application

- Combination of gabapentinoids with SSRI or TCA can be used in combination if monotherapy fails.
- Although opioids reduce neuropathic pain there is no long term outcome evidence on use of opioids for chronic pains. Considering long term harm of opioid use, currently it is advised against their use in painful diabetic peripheral neuropathy.
- Vitamin B complex can be used as an adjunct for mild to moderate diabetic neuropathy.

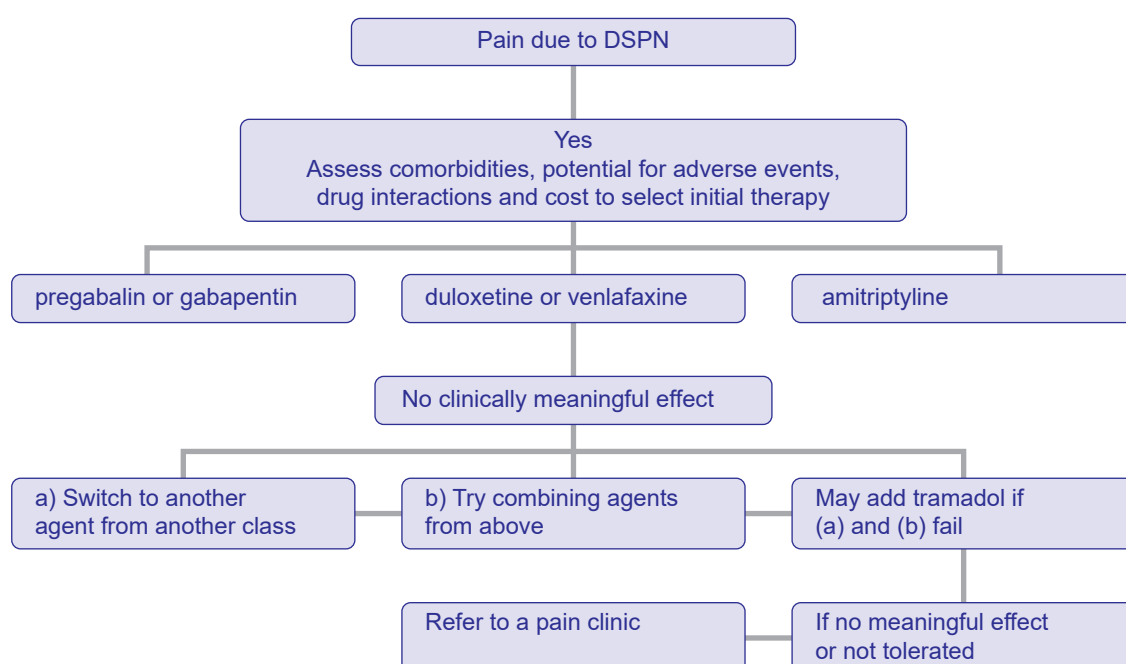


Figure 12.7 Approach to pain relief in painful diabetic neuropathy

Do not prescribe for negative symptoms. Tail off and stop when symptoms are no longer present.

32. Whom to screen for diabetic autonomic neuropathy?

- Patients with other microvascular complications
- Patients with hypoglycaemia unawareness

33. What are the clinical features of diabetic autonomic neuropathy?

Table 12.10 Clinical features of diabetic autonomic neuropathy

Cardiovascular	Gastrointestinal	Urogenital	Sudomotor
Resting tachycardia	Gastroparesis • Nausea • Bloating • Loss of appetite • Early satiety • Postprandial vomiting	Bladder dysfunction • Frequency • Urgency • Nocturia • Hesitancy • Weak stream/dribbling • Urinary incontinence/retention	Anhidrosis • Dry skin
Orthostatic tachycardia/bradycardia			Gustatory sweating
Orthostatic hypotension • Light headed ness • Weakness • Faintness • Visual impairment • Syncope	Oesophageal dysfunction • Heart burn • Dysphagia for solids	Male sexual dysfunction • Erectile dysfunction • Decreased libido • Abnormal ejaculation	
Abnormal blood pressure regulation • Non-dipping • Reverse-dipping	Diabetic diarrhoea • Watery diarrhoea • Faecal incontinence • May alternate with constipation	Female sexual dysfunction • Decreased sexual desire • Decreased sexual arousal • Inadequate lubrication • Increased pain during intercourse	
Exercise intolerance			

34. What is cardiovascular autonomic neuropathy?

- It can be completely asymptomatic and detected only by decreased heart rate variability with deep breathing.
- Advanced disease associated with resting tachycardia (>100 bpm) and orthostatic hypotension

35. How to investigate cardiac autonomic neuropathy?

- Most convenient method in clinical practice is cardiovascular autonomic reflex testing (CART).
- Record electrocardiogram during,
 - Deep breathing
 - Standing from supine position
 - Valsalva manoeuvre
- Measure blood pressure in supine and standing positions

36. How to manage cardiovascular neuropathy?

Orthostatic hypotension

- Nonpharmacologic measures
 - Ensure adequate salt intake,
 - avoid medications that aggravate hypotension,
 - use compressive garments over the legs and abdomen

- Pharmacologic measures
 - Midodrine (alpha adrenergic agonist) and Droxidopa (alpha- and beta-adrenergic agonist)
 - There was low evidence of the efficacy of fludrocortisone use

37. How to diagnose gastroparesis?

- Exclude other causes
 - Medications - Opioid use, GLP-1 RAs
 - Organic gastric outlet obstruction by esophagogastroduodenoscopy or a barium study of the stomach
- Measure gastric emptying
 - Scintigraphy of digestible solids at 15-min intervals for 4 h after food intake
 - ¹³C-octanoic acid breath test

38. How to manage gastroparesis?

- Dietary modifications:
 - Small particle diet
 - Low fat, low fiber diet
- Withdraw drugs affect intestinal motility
 - Opioids, anticholinergics, TCAs, GLP-1RAs, and pramlintide
- Pharmacological measures
 - Metoclopramide
 - Domperidone
 - Erythromycin
- Advanced disease
 - Nasogastric drainage
 - Intra-jejunal feeding
 - Gastric electrical stimulation using a surgically implantable device

39. What are genitourinary disturbances in diabetes?

- Male sexual dysfunction
 - Erectile dysfunction
 - Retrograde ejaculation
- Female sexual dysfunction
 - Decreased sexual desire
 - Increased pain during intercourse
 - Decreased sexual arousal
 - Inadequate lubrication
- Lower urinary tract symptoms
- Urinary incontinence
- Bladder dysfunction (nocturia, frequent urination, urination urgency, and weak urinary stream).

References

1. David J. Leehey, I. M. (2025). Diabetic kidney disease. *BMJ Best practice*
2. Sun, H., Saeedi, P., Karuranga, S., Pinkepank, M., Ogurtsova, K., Duncan, B. B., Stein, C., Basit, A., Chan, J. C. N., Mbanya, J. C., Pavkov, M. E., Ramachandran, A., Wild, S. H., James, S., Herman, W. H., Zhang, P., Bommer, C., Kuo, S., Boyko, E. J., & Magliano, D. J. (2022). IDF Diabetes Atlas: Global, regional and country-level diabetes prevalence estimates for 2021 and projections for 2045. *Diabetes research and clinical practice*, 183, 109119. <https://doi.org/10.1016/j.diabres.2021.109119>
3. hao, M., Cao, Y. & Ma, L. New insights in the treatment of DKD: recent advances and future prospects. *BMC Nephrol* 26, 72 (2025). <https://doi.org/10.1186/s12882-025-03953-3>
4. Prevalence and characteristics of chronic kidney disease in patients attending the nephrology clinics of the National Hospital of Sri Lanka: a cross-sectional study, W. A. N. D. Weerasooriya, H. Karunathilake.
5. Prevalence of micro and macrovascular complications of diabetes detected at single visit screening, B M A C Balasuriya, M R Sumanatilleke, T I Jayasekera, M A Wijesuriya, N P Somasundaram (2012)
6. Wijewickrama, Eranga S.1.; Weerasinghe, Dinushi2; Sumathipala, Pubudu S.2; Horadagoda, Charith1; Lanarolle, Rushika D.2; Sheriff, Rezvi MH.2. Epidemiology of Chronic Kidney Disease in a Sri Lankan Population: Experience of a Tertiary Care Center. *Saudi Journal of Kidney Diseases and Transplantation* 22(6):p 1289-1293, Nov–Dec 2011.)
7. Caramori ML, Rossing P. Diabetic Kidney Disease. [Updated 2022 Aug 3]. In: Feingold KR, Anawalt B, Blackman MR, et al., editors. *Endotext* [Internet]. South Dartmouth (MA): MDText.com, Inc.;
8. Caramori ML, Rossing P. Diabetic Kidney Disease. [Updated 2022 Aug 3]. In: Feingold KR, Anawalt B, Blackman MR, et al., editors. *Endotext* [Internet]. South Dartmouth (MA): MDText.com, Inc.;
9. Subir Ray, Awadhesh Kumar Singh, Jagat Jyoti Mukherjee, Raja Ramachandran, Upal Sengupta, Anil Kumar Virmani, Arup Ratan Dutta, Surendra Kumar Sharma, Sanjay Lal Srivastava, Masood Batin, Protein restriction in adults with chronic kidney disease, with or without diabetes: Integrated Diabetes and Endocrine Academy (IDEA) consensus statement for Indian patients,
10. Diabetes & Metabolic Syndrome: Clinical Research & Reviews, Volume 17, Issue 5, 2023
11. KDOQI Clinical Practice Guideline for Nutrition in CKD: 2020 Update, Ikizler, T. Alp et al. *American Journal of Kidney Diseases*, Volume 76, Issue 3, S1 - S107
12. KDIGO 2022 Look AHEAD trial-secondary analysis of this trial, investigators examined the impact of intensive lifestyle modification on development of very high risk CKD, Intervention reduced the incidence of the very high-risk category of CKD by 31%, suggesting that there are long-term benefits of lifestyle changes in those with diabetes and at risk for CKD)*
13. Sandra C. Naaman, George L. Bakris; Diabetic Nephropathy: Update on Pillars of Therapy Slowing Progression. *Diabetes Care* 1 September 2023; 46 (9): 1574–1586. <https://doi.org/10.2337/dci23-0030>
14. American Diabetes Association Professional Practice Committee; 12. Retinopathy, Neuropathy, and Foot Care: Standards of Care in Diabetes—2025. *Diabetes Care* 1 January 2025; 48 (Supplement_1): S252–S265. <https://doi.org/10.2337/dc25-S012>
15. MG, Karsan W, Peto T, Saravanan P, Virgili G, Preiss D. Diabetic Retinopathy and Quality of Life: A Systematic Review and Meta-Analysis. *JAMA Ophthalmol*. 2024;142(3):199–207. doi:10.1001/jamaophthalmol.2023.6435
16. Simó R, Hernández C. What else can we do to prevent diabetic retinopathy? *Diabetologia*. 2023 Sep;66(9):1614–1621. doi: 10.1007/s00125-023-05940-5. Epub 2023 Jun 6. PMID: 37277664; PMCID: PMC10390367.
17. Jensen ET, Rigdon J, Rezaei KA, Saaddine J, Lundeen EA, Dabelea D, Dolan LM, D'Agostino R, Klein B, Meuer S, Mefford MT, Reynolds K, Marcovina SM, Mottl A, Mayer-Davis B, Lawrence JM. Prevalence, Progression, and Modifiable Risk Factors for Diabetic Retinopathy in Youth and Young Adults With Youth-Onset Type 1 and Type 2 Diabetes: The SEARCH for Diabetes in Youth Study. *Diabetes Care*. 2023 Jun 1;46(6):1252–1260. doi: 10.2337/dc22-2503. PMID: 37043887; PMCID: PMC10234751.
18. Maturi RK, Glassman AR, Josic K, et al.; DRCR Retina Network. Effect of intravitreal anti-vascular endothelial growth factor vs sham treatment for prevention of vision-threatening complications of diabetic retinopathy: the Protocol W Randomized Clinical Trial. *JAMA Ophthalmol* 2021;139:701–712
19. American Diabetes Association Professional Practice Committee; 12. Retinopathy, Neuropathy, and Foot Care: Standards of Care in Diabetes—2025. *Diabetes Care* 1 January 2025; 48 (Supplement_1): S252–S265. <https://doi.org/10.2337/dc25-S012>
20. *BMJ* (<https://bestpractice.bmj.com/topics/en-gb/531>)
21. Atmaca A, Ketenci A, Sahin I, Sengun IS, Oner RI, Erdem Tilki H, Adas M, Soyleli H and Demir T (2024) Expert opinion on screening, diagnosis and management of diabetic peripheral neuropathy: a multidisciplinary approach. *Front. Endocrinol*. 15:1380929. doi: 10.3389/fendo.2024.1380929
22. Ismail-Beigi F, Craven T, Banerji MA, et al.; ACCORD Trial Group. Effect of intensive treatment of hyperglycaemia on microvascular outcomes in type 2 diabetes: an analysis of the ACCORD randomised trial. *Lancet* 2010;376: 419–430
23. Look AHEAD Research Group. Effects of a long-term lifestyle modification programme on peripheral neuropathy in overweight or obese adults with type 2 diabetes: the Look AHEAD study. *Diabetologia* 2017;60:980–988
24. Gogan, A.; Potre, O.; Avram, V.-F.; Andor, M.; Caruntu, F.; Timar, B. Cardiac Autonomic Neuropathy in Diabetes Mellitus: Pathogenesis, Epidemiology, Diagnosis and Clinical Implications: A Narrative Review. *J. Clin. Med*. 2025, 14, 671. <https://doi.org/10.3390/jcm14030671>
25. Rodica Pop-Busui, Autonomic diabetic neuropathies: A brief overview, *Diabetes Research and Clinical Practice*, Volume 206, Supplement 1, 2023, 110762, ISSN 0168-8227, <https://doi.org/10.1016/j.diabres.2023.110762>
26. Camilleri M, Kuo B, Nguyen L, et al. ACG clinical guideline: gastroparesis. *Am J Gastroenterol* 2022;117:1197–1220:92.

13. FOOT DISEASE IN DIABETES

1. Why should clinicians pay attention to footcare in patients with diabetes?

- Diabetes is the leading cause of non-traumatic lower limb amputation.
- This is preventable by early recognition of preceding risk factors.
- Early detection of risk factors and timely multidisciplinary care can significantly reduce amputation risk

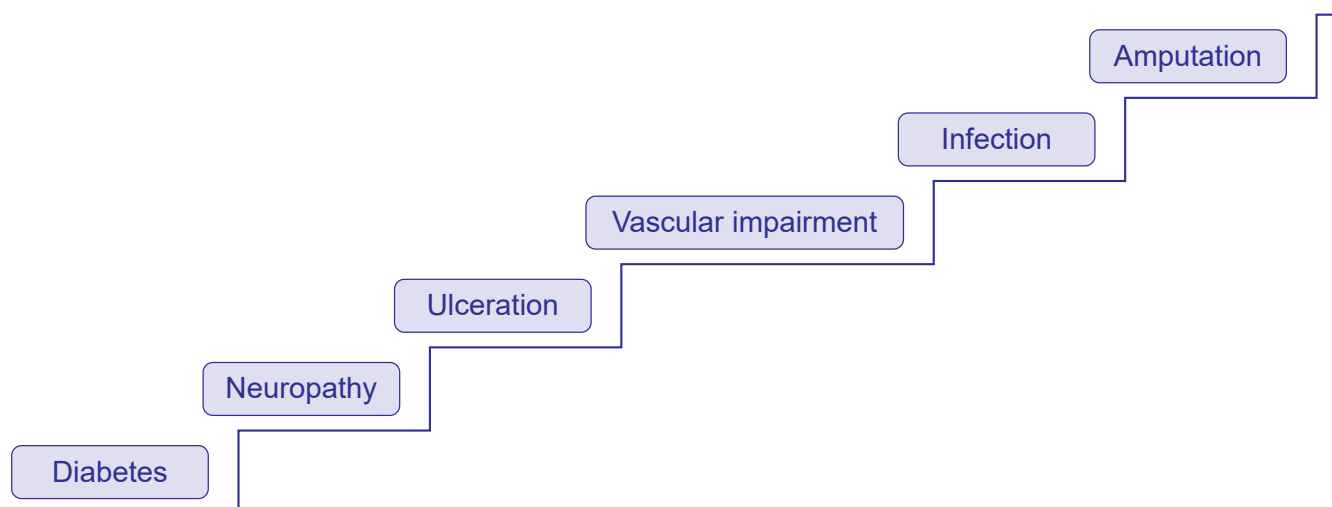


Figure 13.1 The stairway to an amputation in a person with diabetes

2. What is the pathogenesis of foot ulceration in diabetes?

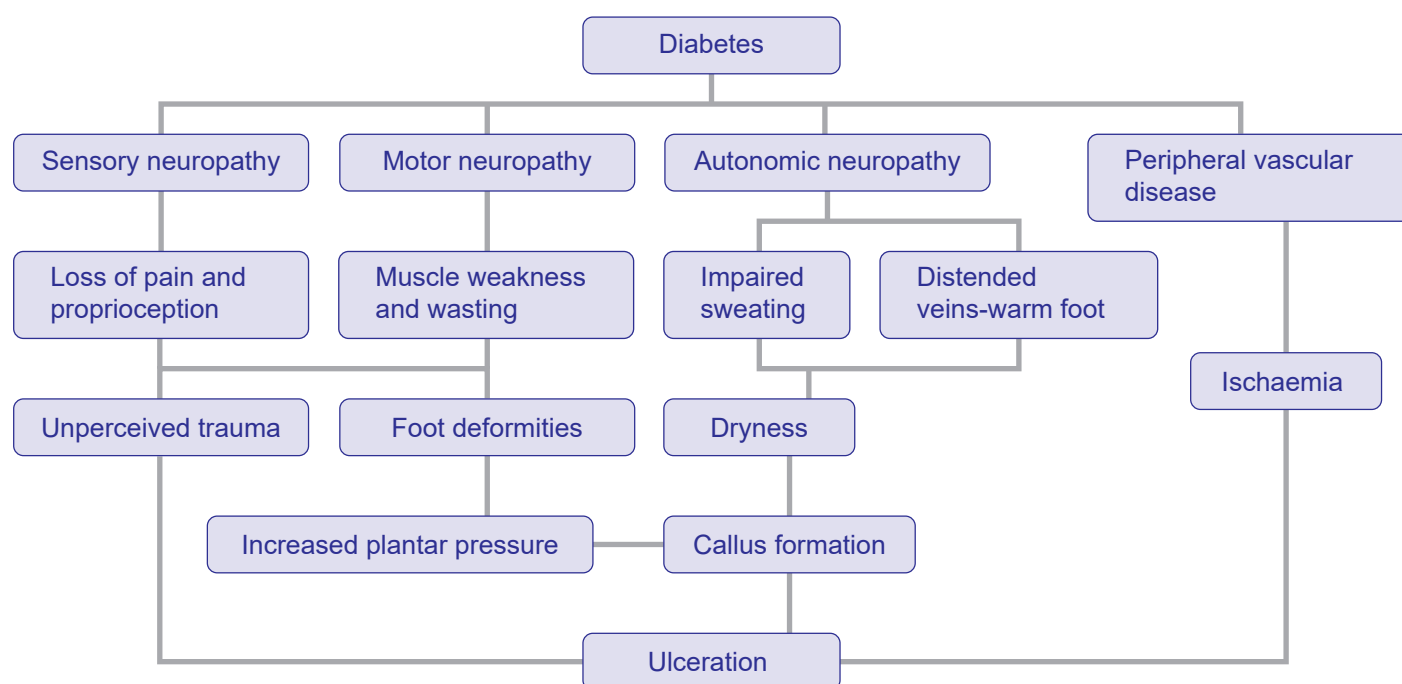


Figure 13.2 Pathogenesis of foot ulceration in diabetes

3. What are the risk factors for foot ulceration?

- Peripheral neuropathy
- Peripheral vascular disease
- Foot deformity
- Improper footwear
- History of foot ulceration, amputation
- Other complications of diabetes, including poor eye sight
- Diabetic kidney disease, especially on renal replacement therapy
- Other Comorbidities

- Poor glycemic control
- Longer duration of diabetes
- Older age
- Living alone

4. What are the important steps in screening for foot disease?

Foot screening checklist for clinical examination and guide to techniques are attached. (Annexure 13.1)

History:

- Demographic data: age, gender etc.
- Previous ulcer or lower extremity amputation
- Symptoms of ischemia (claudication and rest pain)
- Symptoms of neuropathy (numbness, pain etc.)
- Others – smoking status, social and financial situation, previous education of foot care, kidney disease

Examination:

- Inspection:
 - Skin: Skin colour, temperature, presence of callus or oedema, pre-ulcerative signs
 - Foot hygiene: Improperly cut toenails, superficial fungal infection
 - Bone/ joint: Deformities (eg: claw or hammer toes), abnormally large bony prominences, or limited joint mobility.
 - Footwear inspection: Check inside of footwear for safety.
- Vascular status:
 - Pedal pulses (dorsalis pedis pulse, posterior tibial pulse)
 - Ankle Brachial Pressure Index (ABPI) when available.
 - The presence of an ABPI 0.9-1.3 or a tri-phasic pedal pulse waveform largely excludes Peripheral Arterial Disease (PAD).
- Loss Of Protective Sensation (LOPS):
 - Pressure perception: Semmes-Weinstein 10-g monofilament (2/3 loss in pressure point considered as LOPS) (figure 13.3)
 - 10-g monofilament plus one of the following:
 - Vibration with 128-Hz tuning fork
 - Pinprick
 - Temperature perception
 - Biothesiometry - Vibration Perception Threshold (VPT) to detect early sensory loss

When monofilament or other methods are not available, use Ipswich touch test.

- Ipswich touch test
 - Lightly touch the tips of the toes of the patient with the tip of your index finger for 1 to 2 seconds (>2 sites loss of sensation when checking on 1st, 3rd, 5th toe tips – LOPS)



Figure 13.3 Sites of monofilament test

5. How can the basics of footcare can be ensured in a busy clinic?

- Following three steps can be performed within 3 minutes (1 minute each)

Table 13.1 Three minute foot care strategy for a busy clinic

History	Examination	Education
• Prior ulcer/ amputation • Numbness/ pain • Smoking • Current footcare	• Skin inspection • Deformities • Sensory (Ipswich touch test) • Pulses	• Daily foot examination • Dry after washing • Moisturise dry skin • Appropriate footwear • Proper nail cutting • Early medical advice in active foot disease

6. How frequently should people be screened for foot disease?

- At least once a year for all people with diabetes.
- More frequently based on risk category:
 - Every 3 – 6 months if the patient has any one risk factor (LOPS, PAD, deformity).
 - Every 1 – 3 months if there is a history of ulceration or amputation, or multiple risk factors combined.
 - Every 1 – 2 weeks or more often for those with active foot problems (ulcer, infection, Charcot foot).

7. How to assess and stratify a person's current risk of foot ulceration / amputation?

Risk stratification is based on:

- LOPS
- Foot deformity
- Limb ischaemia
- History of foot ulceration/ amputation
- Renal replacement therapy

Table 13.2 Risk stratification system and corresponding foot screening frequency

Risk Category	Risk characteristics	Frequency of follow up/	Interventions/ team involved
Low	No risk factors present except callus alone	Once a year	Education
Moderate	Deformity or Neuropathy or Non-critical limb ischaemia	Once every 3 - 6 months	Initiate basic podiatry care referral and education
High	Previous ulceration or previous amputation or On renal replacement therapy or Neuropathy and non-critical limb ischaemia or Neuropathy in combination with callus and/ or deformity	Once every 1 - 3 months	Intense podiatry care
Active diabetic foot problem	Ulceration or Spreading infection or Critical limb ischaemia or Gangrene or suspicion of an acute Charcot arthropathy or An unexplained hot, red, swollen foot with or without pain	Once in every 1- 2 weeks or frequently	Urgent foot clinic / foot MDT/ surgical referral

8. What are the steps in preventing development of foot ulcers?

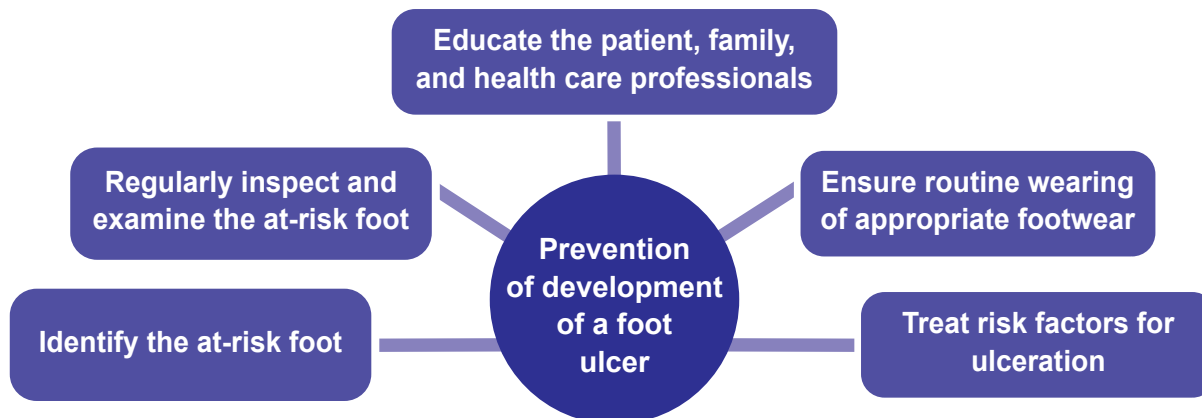


Figure 13.4 Steps in prevention of foot ulcers in people with diabetes

9. What are the main aspects in educating the patient, family and healthcare professionals about foot care?

- Inspect feet daily.
- Wash feet daily with water (neither hot nor cold), and dry them carefully, especially between the toes.
- Use emollients to lubricate dry skin but not between the toes.
- Avoid walking barefoot or in thin-soled slippers indoor or outdoor.
- Wear proper footwear after visually inspecting and manually feeling inside.
- Cut toenails straight across, preferably by another person who could see properly, under good lighting.
- Participate in routine foot checkup clinic as recommended based on the risk category.
- Seek medical advice, if foot is warmer, red, if a blister, cut, scratch, callus, nail infection or ulcer has developed (do not use chemical agents or plasters to remove corns and calluses).
- Avoid self-treatment of corns or calluses with sharp tools or chemicals
- Involve family/ caregivers when self-care is difficult
- Ensure healthcare providers reinforce these messages regularly

10. What are the features of appropriate footwear?

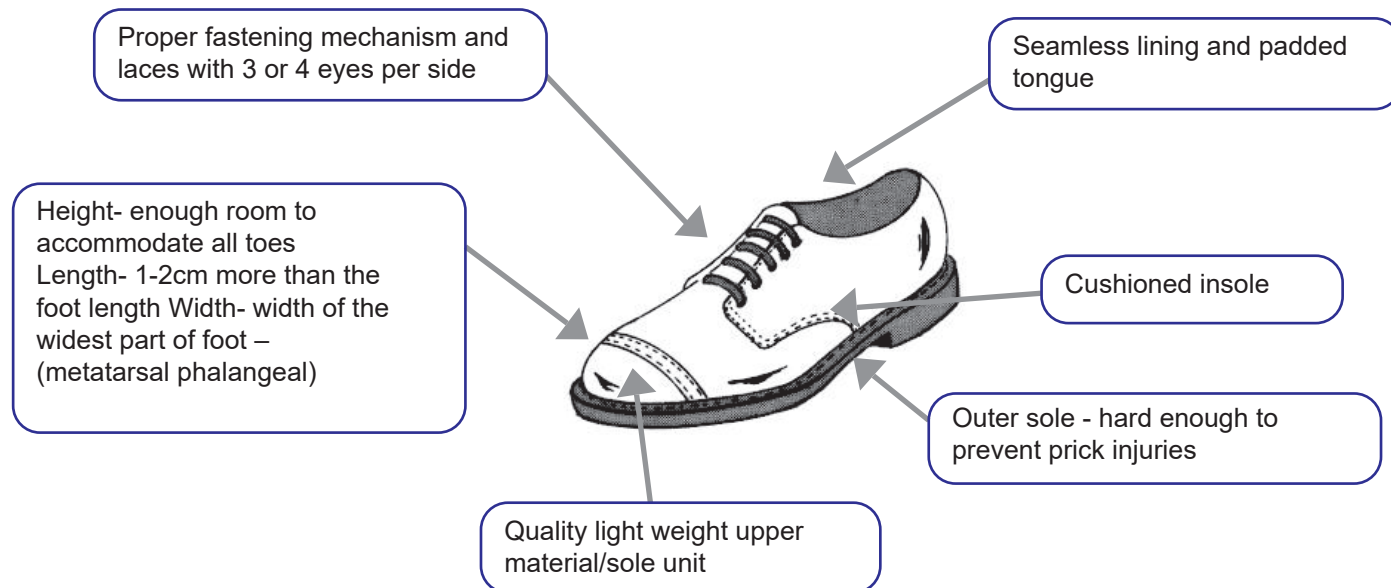


Figure 13.5 Characteristics of good footwear

- Fitting should be checked in standing position during the latter part of the day to accommodate possible oedema
- If needed refer the patient for special footwear (advice and/or construction), possibly including extra-depth shoes, custom made shoes, insoles, or orthoses.

11. How to treat risk factors for ulceration?

- Shaving callus
- Protecting blisters or draining them if necessary
- Appropriately treating ingrown or thickened nails
- Prescribing antifungal treatment for fungal infections
- Surgical interventions (e.g., osteotomy/ tenotomy/ tendon lengthening and transfers) for deformities that cause recurrent ulcers
- Revascularisation of the ischaemic foot
- Therapeutic offloading footwear
- Glycemic optimization, smoking cessation, and lipid/ blood pressure control

12. When to refer an individual to acute services or to a specialty (vascular/ orthopaedic/ surgical)?

- Any new-onset ulceration
- Ulceration with fever or any signs of sepsis or ischemia
- Infection - deep-seated soft tissue or bone infection (with or without ulceration)
- Gangrene (with or without ulceration)
- Suspected acute Charcot arthropathy (hot, swollen, red foot, especially in a neuropathic foot)

If a person has a limb-threatening or life-threatening foot problem, refer immediately to acute services

13. How to assess a foot ulcer?

- Type of ulcer: neuropathic, neuro-ischaemic, or ischaemic
- Ulcer characteristics: cause (e.g., pressure or trauma), site, size, depth
- Probe to bone test if osteomyelitis is suspected
- Signs of infection: local (discharge, warmth, swelling) or systemic (fever, sepsis)
- Assessment of perfusion: pedal pulses, capillary refill, ABPI if needed
- Standardized scoring: use the SINBAD system to assess and monitor severity
- Photograph and document the ulcer at each visit for comparison
- Consider imaging (e.g., X-ray, MRI) if bone or deep tissue involvement is suspected

Table 13.3 SINBAD system of scoring severity of a foot ulcer.

Category	Description	Score
Site	Forefoot	0
	Midfoot and hindfoot	1
Ischaemia	Pedal blood flow intact: at least one palpable pulse	0
	Clinical evidence of reduced pedal flow	1
Neuropathy	Protective sensation intact	0
	Protective sensation lost	1
Bacterial infection	None	0
	Present	1
Area	Ulcer < 1 cm ²	0
	Ulcer ≥ 1 cm ²	1
Depth	Ulcer confined to skin and subcutaneous tissue	0
	Ulcer reaching muscle, tendon or deeper	1
Total score		(Out of 6)

Source: Ince P. et al. Use of the SINBAD classification system and score in comparing outcome of Foot Ulcer Management on three continents. *Diabetes Care* May 2008; 31(5): 964-967. <http://doi.org/10.2337/dc07-2367>

14. What are the main components of treating a foot ulcer?

- **Offloading** (the key to ulcer healing for plantar ulcers); (figure 12.6)
 - Plantar ulcers
 - non-removable casting to offload neuropathic, non-ischaemic, uninfected ulcers
 - Non-plantar ulcers - decide according to the type and location of the foot ulcer
 - removable ankle-high offloading device
 - footwear modifications
 - toe spacers
 - orthoses

**Figure 13.6** Methods used for off-loading foot ulcers in people with diabetes

1. TCC- total contact cast 2. removable boot 3. orthowedge 4. post-op shoe

- **Control of ischaemia**
 - Assess perfusion via pulses, ABPI, or imaging
 - Refer to vascular surgery for revascularization (angioplasty or bypass) if critical limb ischemia is suspected

- **Control of foot infection**
 - Evaluate for infection using clinical signs and probe-to-bone test
 - Take deep tissue samples or swabs for culture before starting antibiotics
 - Use empiric antibiotics then tailor based on culture and clinical response
 - Use imaging (X-ray or MRI) to assess for osteomyelitis
- **Wound debridement**
 - Regular inspection of the ulcer by a trained staff
 - Debride the ulcer and remove surrounding callus and repeat as needed
- **Wound dressings**
 - Select dressings to control excess exudation and maintain moist environment.
 - Consider negative pressure to help heal postoperative wound
- **Patient and family education**
 - Reinforce the importance of foot care during and after ulcer healing
 - Educate on early signs of recurrence
 - Ensure access to proper footwear and follow-up plans

- Recurrence rate of foot ulcer/ admission is high in those who had a previous ulcer.
- Make sure patient and family education, appropriate footcare and footwear are implemented after the cure of a foot ulcer to prevent recurrences.

15. How to identify and manage Charcot arthropathy?

- Suspect acute Charcot arthropathy,
 - Redness, warmth, swelling or deformity (in particular, when the skin is intact), especially in the presence of peripheral neuropathy or renal failure.
 - Offer non-weight-bearing treatment until definitive treatment can be started by the multidisciplinary footcare service/ orthopaedic team.
- Refer the person within one working day to the specialised multidisciplinary foot care service or to orthopaedic team.
- Confirm the diagnosis
 - Imaging - weight-bearing X-ray of the affected foot and ankle
 - Consider an MRI if the X-ray is normal but suspicious of Charcot arthropathy
- Treatment
 - Offer a non-removable offloading device (total contact cast)
 - If a non-removable device is not advisable because of clinical, or personal circumstances, consider treatment with a removable offloading device.
 - Bisphosphonates have been studied in small, randomised trials (RCT) as a treatment in acute Charcot neuroarthropathy. However, there is lack of good quality evidence to recommend this treatment.
- Offer long term care with offloading device (rocker bottom shoes)

16. What are the main specialty teams involved in an ideal multidisciplinary footcare service?

It should be led by a named healthcare personal and should consist of skilled specialists from following specialties:

- Endocrinology/ General medicine
- Podiatry
- Diabetes specialist nursing
- Vascular surgery
- Orthopaedic surgery
- Biomechanics and orthoses
- Interventional radiology
- Casting
- Wound care

References:

1. Schaper NC, van Netten JJ, Apelqvist J, et al, IWGDF Editorial Board. Practical Guidelines on the prevention and management of diabetic foot disease (IWGDF 2019 update). *Diabetes/metabolism research and reviews*. 2020 Mar;36:e3266.
2. Monteiro-Soares M, Russell D, Boyko EJ, et al, International Working Group on the Diabetic Foot (IWGDF). Guidelines on the classification of diabetic foot ulcers (IWGDF 2019). *Diabetes/metabolism research and reviews*. 2020 Mar;36:e3273.
3. NICE guidelines 2015 - Diabetic foot problems: Prevention and Management (NG19) (updated in 2019)
4. American Diabetes Association. 11. Microvascular Complications and Foot Care: Standards of Medical Care in Diabetes—2025: *Diabetes Care* 2025; 44 (Suppl. 1): S151–S167.
5. American Diabetes Association. 12. Retinopathy, Neuropathy, and Foot Care: Standards of Care in Diabetes—2025. *Diabetes Care*. 2025 Jan;48(Suppl 1):S252–S265. doi:10.2337/dc25-S012. Available from: https://diabetesjournals.org/care/article/48/Supplement_1/S252/157552/12-Retinopathy-Neuropathy-and-Foot-Care-Standards

14. DIABETIC EMERGENCIES

A. Hypoglycaemia

1. How to diagnose hypoglycaemia in people with diabetes?

- Blood glucose (BG) < 70 mg/dL (3.9 mmol/L)
- During a hospital admission, any blood glucose less than <70 mg/dl should be treated.

2. What are the common medications that cause hypoglycaemia?

- Insulin
- Sulfonylurea
 - Hypoglycaemia is the commonest side-effect of insulin and sulfonylurea therapy because of their modes of action.
 - Metformin, pioglitazone, DPP-4 inhibitors, acarbose, SLGT-2 inhibitors and GLP-1 analogues prescribed without insulin or an insulin secretagogue (sulfonylurea and repaglinide) are unlikely to result in hypoglycaemia.

3. How to classify hypoglycaemia in clinical practice?

Table 14.1 Classification of hypoglycaemia

Category	Glycaemic criteria / description
Level 1 / mild	Glucose <70 mg/dL (3.9 mmol/L) and ≥54 mg/dL (3.0 mmol/L)
Level 2 / moderate	Glucose <54 mg/dL (3.0 mmol/L)
Level 3 / severe	Altered mental and/or physical status requiring assistance for treatment of hypoglycaemia

- Level 1 hypoglycemia – threshold for adrenergic responses (autonomic)
- Level 2 hypoglycemia – threshold for neuroglycopenic symptoms – requires immediate action to resolve event.

4. What are the common symptoms of hypoglycaemia?

Table 14.2 Symptoms of hypoglycaemia

Autonomic	Neuroglycopenic	General
Sweating Palpitations Shaking Hunger	Abnormal behaviour Speech difficulty Incoordination Seizures Drowsiness Confusion/ loss of consciousness	Headache Nausea Malaise

5. How to assess hypoglycemic risk ?

- Hypoglycaemia history should be assessed at every clinical encounter/ hypoglycaemic event frequency, severity, precipitants, symptoms, approach to treatment
- Correlate home glucose readings (if possible) with symptoms and treatment (patients may experience and treat hypoglycaemic symptoms without checking thereby treating normal glucose values)
- Those at risk for hypoglycaemia – Ask for impaired hypoglycaemia awareness at least annually
- Consider an individual's risk for hypoglycaemia when selecting diabetes medications and glycaemic goals.
- Use of CGM is beneficial for individuals at high risk for hypoglycaemia.
- Screen older adults with diabetes for cognitive function decline – may be due to hypoglycaemia

Table 14.3 Assessment of hypoglycaemia risk among individuals treated with insulin, sulfonylureas, or meglitinides

Category	Major risk factors	Other risk factors
Clinical & biological	- Recent moderate/ severe hypoglycaemia (past 3–6 months) - Intensive insulin therapy - Impaired awareness of hypoglycaemia - End-stage kidney disease - Dementia or cognitive impairment	- Multiple mild hypoglycaemia episodes - Basal insulin therapy - Age ≥ 75 years - Female sex - High glycaemic variability - Polypharmacy - Heart disease or stroke - Moderate kidney disease - Neuropathy, retinopathy - Depression or severe mental illness
Social & economic	- Food insecurity - Low income - Housing instability - Religious fasting	- Low health literacy - Alcohol or drug misuse

- High risk - Any 1 major risk factor or multiple other risk factors.
- Use clinical judgment to assess risk.
- These individuals may need individualised targets, simplified regimens, or CGM/ SMBG support.
- Be especially cautious in elderly, those with kidney disease, or those with poor social support.

6. What is impaired awareness of hypoglycaemia (IAH)/ hypoglycaemia unawareness and how should it be treated?

- Reduced Intensity of hypoglycaemic symptoms, altered nature of symptoms or complete loss of symptoms when BG drops below 70 mg/dL in patients with diabetes.
- It increases the risk of severe hypoglycaemia and death.
- Recurrent treatment related hypoglycaemia lowers the BG threshold to trigger protective counter regulatory mechanisms and cause IAH.
- Avoid hypoglycaemia for 2-3 weeks by giving careful attention to BG readings and insulin doses to restore awareness.
 - Setting higher glycaemic goals in short term
 - Increased home BG monitoring

7. What are the glycaemic targets for people with diabetes in hospital?

- Joint British Diabetes Societies (JBDS) for Inpatient care suggest a target blood glucose level of 6.0 - 10.0 mmol/L (108 - 180 mg/dl)
- The American Diabetes Association suggest less stringent target blood glucose level of 7.8 - 10.0 mmol/L (140 - 180 mg/dl) for people on insulin therapy.
- It is important to emphasise the glycaemic target should be adjusted according to the person's individual circumstances; a frail, elderly person with multiple comorbidities may benefit from less stringent glycaemic control.

8. What is looming hypoglycemia?

- While the treatment threshold for hypoglycaemia remains at less than 4.0 mmol/L, for people with diabetes in hospital, it may be appropriate to act proactively to prevent hypoglycaemia at higher blood glucose levels.
- A blood glucose value between 4.0 to 6.0 mmol/L (72 - 108 mg/dl) could indicate looming hypoglycaemia; particularly if repeated episodes showing a particular pattern of glucose measurements below 6.0 mmol/L were occurring.

9. How to manage hypoglycaemia?

Step 1: Acute management

Treat promptly with 15-20 g of quick acting carbohydrate to return their BG to normal range

Table 14.4 Sources of 15-20 g of quick acting carbohydrates

Oral	Intravenous	Intramuscular
3-4 heaped tea spoons of glucose	200 ml of 10% dextrose	1 mg of glucagon
3-4 heaped tea spoons of sugar dissolved in water (not effective if on acarbose)	100 ml of 20% dextrose	(Not useful in patients with depleted glycogen reserves e.g., impaired hepatic function/ excessive alcohol consumption)
150-200 ml of pure fruit juice (e.g., orange juice)		

- Avoid,
 - Foods containing added fat (eg., chocolate) or protein
 - 50% dextrose solution: increase the risk of extravasation injury and post-treatment over shoot of BG

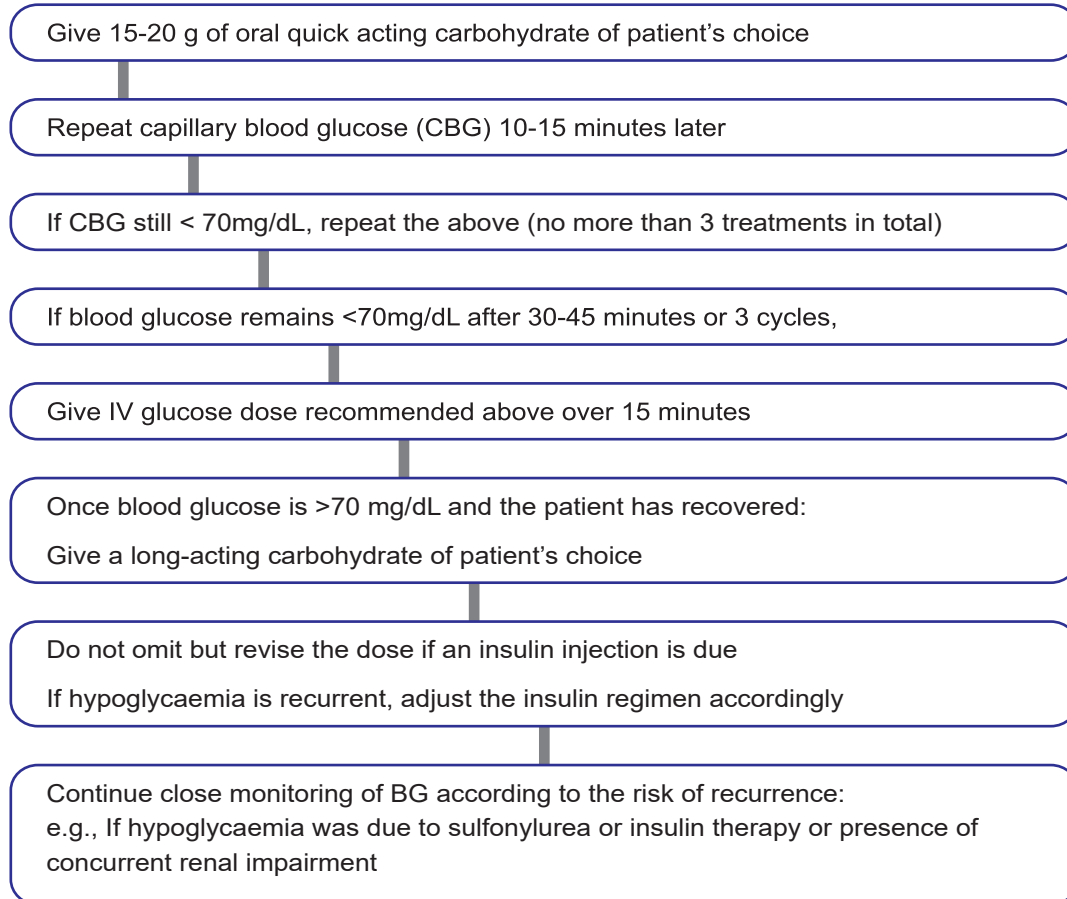
Step 2: Prevention of recurrence

Once BG returns to normal, give a long-acting carbohydrate as a snack or as a part of a main meal,

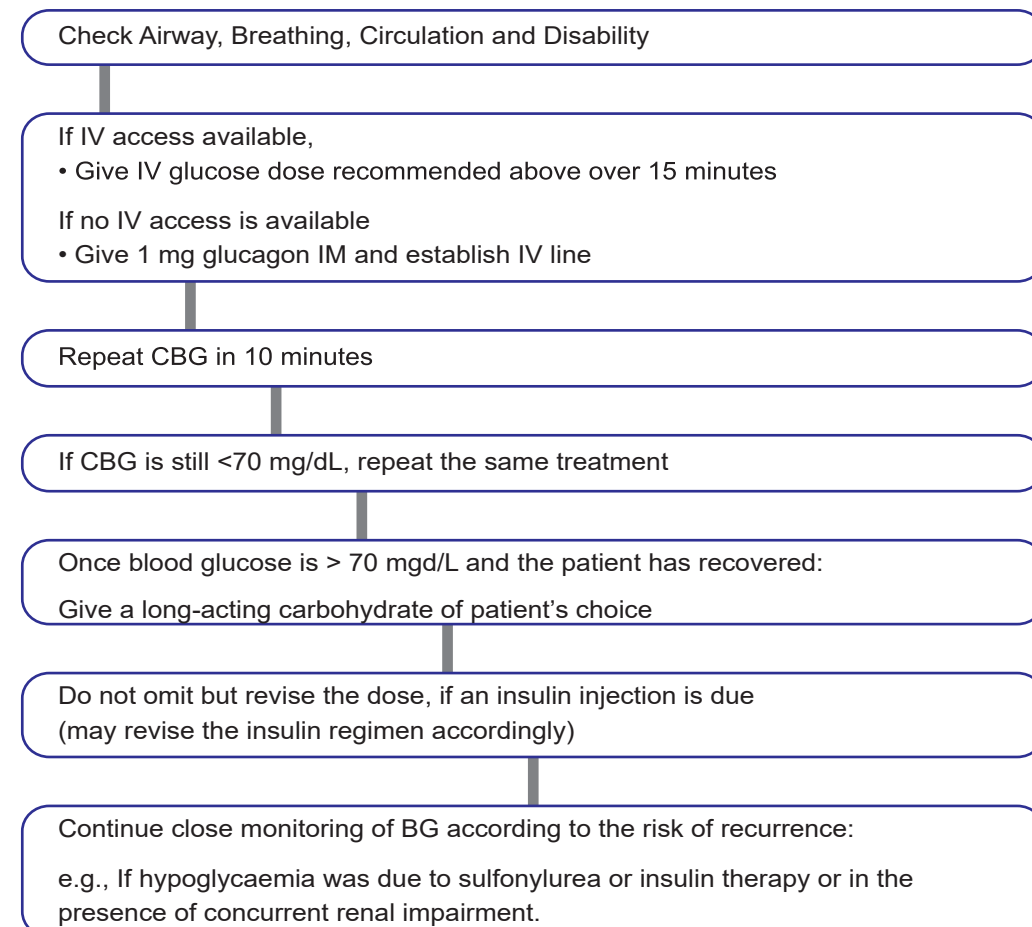
- Two biscuits
- One slice of bread
- 200-300 ml glass of milk
- Normal meal if due (must contain carbohydrate)

Management algorithms

1. Conscious, oriented and able to swallow



2. Having altered level consciousness and/or seizures



10. How to prevent hypoglycaemia in the clinical setting?

- Inquire about all hypoglycaemic episodes and take action to prevent further episodes.
- Revise glucose lowering agents (insulin, sulphonylurea).
- Individualise glycaemic targets (i.e., less stringent targets for elderly).
- Educate on prevention of hypoglycaemia with exercise.
- Use frequent capillary glucose monitoring to identify hypoglycaemia episodes.
- Consider newer BG monitoring technologies (e.g., CGM).

B. Diabetic ketoacidosis (DKA)

11. What is DKA?

- DKA is characterised by the triad of diabetes/ hyperglycaemia, metabolic acidosis and ketosis

D - Diabetes/ hyperglycemia - Blood glucose ≥ 200 mg/dL **OR** prior history of diabetes

K – Ketosis - Blood β -hydroxybutyrate ≥ 3.0 mmol/L **OR** urine ketone strip 2+ or greater

A - Metabolic Acidosis - pH < 7.3 and/ or bicarbonate (HCO_3^-) < 18 mmol/L

Increased mortality associated with DKA is predominantly due to the precipitating illnesses (i.e., pneumonia, myocardial infarction, sepsis) and rarely due to metabolic derangements (i.e., hypokalaemia).

12. What are the precipitating factors for DKA?

- Infections (commonest)
- Discontinuation of insulin/ inadequate insulin
- Type 1 diabetes/ absolute insulin deficiency
- Medications – steroids, SGLT2 inhibitors, atypical antipsychotics, immune checkpoint inhibitors
- Pancreatitis
- Intercurrent illness – myocardial infarction, stroke, trauma
- Psychological stress
- Alcohol/ substance use

13. What are the clinical features of DKA?

- Symptoms
 - Nausea, vomiting, diffuse abdominal pain are frequent symptoms ($>50\%$)
 - Hyperosmotic symptoms (polyuria, polydipsia and weight loss)
 - Change in mental status (reduced alertness or profound lethargy or coma)
- Signs
 - Poor skin turgor, Kussmaul respiration, tachycardia, and hypotension
 - Fever due to an underlying infection (patients can be normothermic or even hypothermic due to peripheral vasodilation)
 - Fruity odour of breath (due to acetoacetate)

14. What is euglycaemic ketoacidosis?

- DKA with near normal blood glucose levels (<200 mg/dL)
- Causes
 - Treatment with SGLT2 inhibitors – majority of euglycaemic DKA
 - Prevented by following sick day rules – stop SGLT2i on admission
 - After DKA resolution – do not initiate or continue SGLT2i
 - Reduced food intake
 - Insulin injections immediately prior to admission

- Pregnancy
- Conditions with impaired gluconeogenesis (heavy alcohol abuse, liver failure)
- Manage with 5 -10% dextrose added to IV fluids at the same time as insulin infusion

Caveats in diagnosis of DKA

- Venous blood gas is adequate in diagnosis and management of DKA.
- Anion gap is not recommended as a diagnostic criterion - high anion gap metabolic acidosis common in DKA ($AG > 10-12 \text{ mEq/l}$), but $> 1/3$ of patients present with mixed anion gap acidosis.
- Mixed anion gap acidosis is due to;
 - Hyperglycaemia induced osmotic diuresis loss of $\text{HCO}_3^- \rightarrow$ metabolic acidosis
 - Vomiting $\rightarrow$ loss of gastric $\text{HCl} \rightarrow$ metabolic alkalosis
 - Compensatory respiratory alkalosis with Kussmaul breathing
 - Hyperchloremic normal anion gap acidosis – after resolution of DKA - due to high volumes of normal saline that is used in DKA (this is mistaken for persistent DKA)
- Best test for ketonaemia is direct venous/ capillary β - hydroxybutyrate, which measures the main metabolic product in DKA.
 - The nitroprusside test (commonly used in urine ketone body tests, detects acetoacetate). It does not detect β - hydroxybutyrate and could underestimate the severity of DKA.
 - Urine β hydroxybutyrate is converted to acetoacetate on recovery of DKA - therefore urine ketone bodies can be persistently positive despite true resolution of DKA.
 - False positive urine ketone bodies with captopril, valproate use.
 - Other conditions causing ketonaemia: starvation ketosis, alcoholic ketoacidosis, ketosis of pregnancy, hyperemesis gravidarum

15. How is DKA classified according to severity?

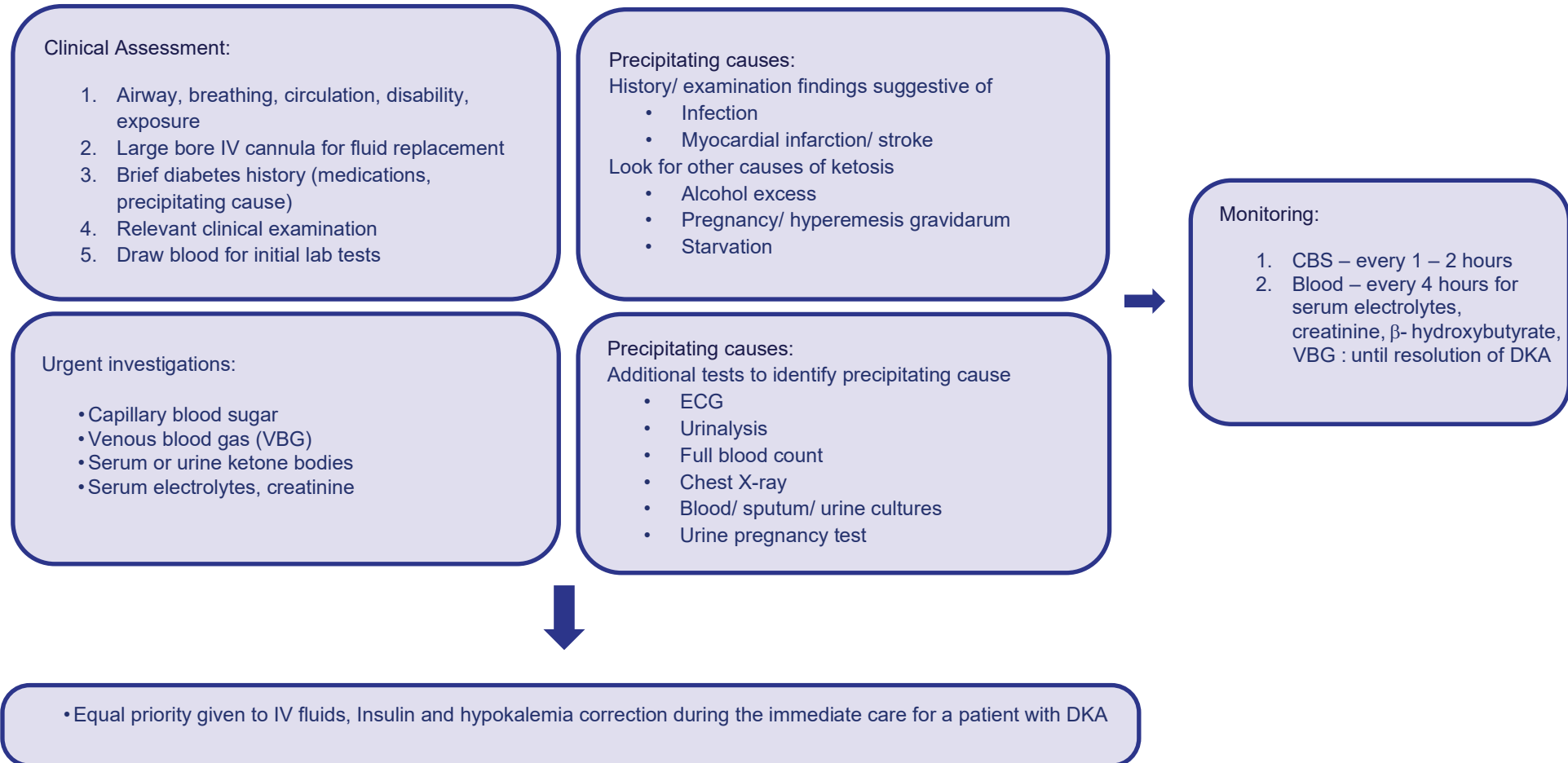
Table 14.5 DKA severity classification

	Mild DKA	Moderate DKA	Severe DKA
“D”: history of diabetes or elevated glucose level	Glucose $\geq 200 \text{ mg/dL}$	Glucose $\geq 200 \text{ mg/dL}$	Glucose $\geq 200 \text{ mg/dL}$
“K”: ketonaemia	$\beta\text{-HB } 3.0-6.0 \text{ mmol/L}$	$\beta\text{-HB } 3.0-6.0 \text{ mmol/L}$	$\beta\text{-HB } > 6.0 \text{ mmol/L}$
“A”: acidosis	pH 7.25-7.30 or $\text{HCO}_3^- 15-18 \text{ mmol/L}$	pH 7.0-7.25 $\text{HCO}_3^- 10 \text{ to } < 15 \text{ mmol/L}$	pH < 7.0 $\text{HCO}_3^- < 10 \text{ mmol/L}$
Mental status	Alert	Alert/ drowsy	Stupor/ coma
Suggested level of care	Regular or observation nursing unit	Step-down unit or intermediate care unit	Intensive care unit

This classification enables to identify the level of care/ management setting. Also mild DKA can be managed with subcutaneous rapid acting insulin (as an alternative to IV insulin)

16. How to manage DKA?

Step 1: Immediate management



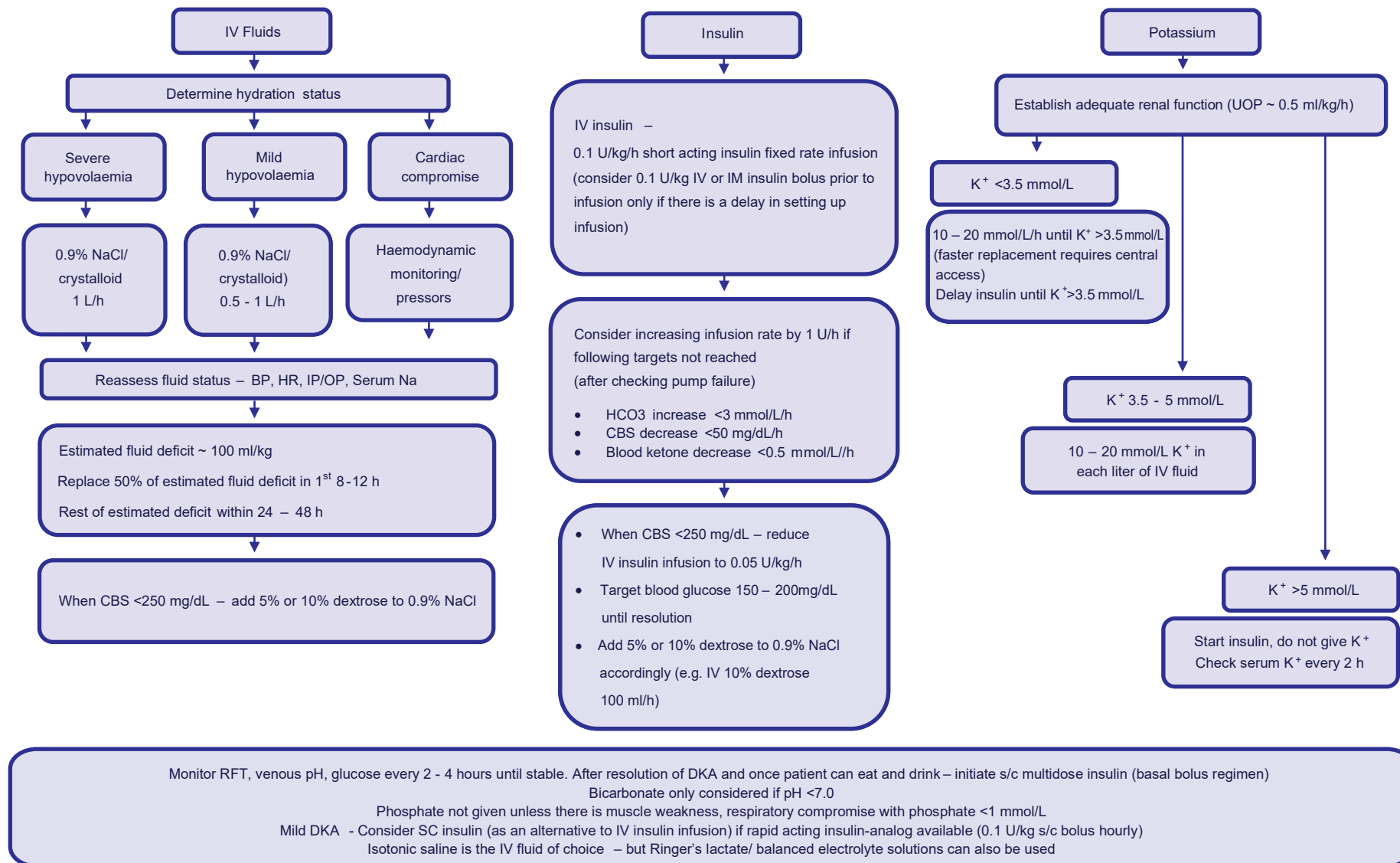


Figure 14.1 Management algorithm of DKA

17. What is the place for bicarbonate administration in DKA?

No advantage in improving clinical outcomes or for recovery of DKA

Possible detrimental effect – avoid unless pH < 7.0

- 100 ml of 8.4% NaHCO₃ in 400 ml sterile water – repeated every 2 h to achieve a pH >7.0

18. What is the place for basal insulin?

- Basal insulin while on fixed rate insulin infusion may be continued during IV insulin infusion
- Basal insulin can also be initiated while on IV insulin infusion – may reduce time to DKA resolution and prevent rebound hyperglycemia
- Can be given as once or twice daily doses, 0.15 – 0.3 U/kg/day

19. What are the criteria for resolution of DKA?

- Venous pH > 7.3 or bicarbonate > 18 mmol/L and
- Plasma/ capillary ketones <0.6 mmol/L
- Urine ketone bodies may persist to be positive even after resolution – therefore it is NOT taken as criteria for resolution
- Anion gap is not taken as a criterion - persistent acidosis with negative serum ketone bodies may be due to hyperchloremic metabolic acidosis following DKA resolution, due to excessive normal saline use.

20. What is the management after resolution of DKA?

- Start subcutaneous insulin 1 – 2 h prior to discontinuing IV insulin infusion
- Ensure insulin regimen provides 24h coverage
 - Basal insulin and rapid acting insulin analogs preferred
 - 3 methods are used at present to calculate total daily dose (TDD) – weight based estimates, preadmission insulin requirements, hourly IV insulin requirements

Weight based estimates

- 0.5 – 0.6 U/kg/d for total daily dose (TDD)
- 0.3 U/kg/d for those with risk of hypoglycemia

Preadmission insulin requirements

- Consider TDD of insulin according to the insulin regimen used prior to admission

Hourly IV insulin requirements

- Summation of stable hourly IV insulin requirements may help to estimate TDD
- Caution- insulin requirement is overestimated due to glucotoxicity

- If newly diagnosed T1DM – basal bolus regimen can be initiated after DKA resolution
 - Long acting basal insulin – 0.15 – 0.3 U/kg – once daily or in two divided equal doses
 - Rapid acting insulin – according to nutritional intake/ glucose levels (carbohydrate counting)
- Do not initiate/ continue SGLT2 inhibitors during hospitalisation
- Other non-insulin agents may be considered during hospitalisation or discharge of T2DM patients

21. What are the complications of DKA?

Table 14.6 Complications and suggested approaches in DKA

Complication	Mechanism	Suggested approach
Hypokalaemia	Serum K ⁺ will almost always fall when DKA is treated with insulin. Indicates severe total-body K ⁺ deficiency	Requires careful cardiac monitoring and more vigorous K ⁺ replacement because insulin lowers K ⁺ further and can provoke cardiac dysrhythmia. Monitor K ⁺ 4 hourly
Hyperkalaemia	Due to extracellular shift of potassium Caused by insulin deficiency, hypertonicity, acidosis and pre-renal AKI due to dehydration	Usually settles with rehydration and insulin unless there is renal impairment Monitor K ⁺ 4 hourly
Hyponatraemia (mild)	Reduced Na ⁺ due to osmotic flux of water from intracellular to extracellular space in the presence of hyperglycaemia	An increased or even normal Na ⁺ in the presence of hyperglycaemia might indicate profound degree of free water loss
Hypophosphataemia	Reduced phosphate due to transcellular shift, osmotic diuresis and reduced renal phosphate reabsorption in the proximal renal tubule (due to acidosis and hyperglycaemia) However, phosphate level may be normal or even increased on admission.	IV phosphate replacement is not routinely recommended but consider in; • Muscle weakness, respiratory/cardiac compromise with phosphate <1.0 mmol/L 20–30 mmol/l potassium phosphate can be added to replacement fluids • The maximal rate for safe phosphate replacement : 4.5 mmol/h (1.5 ml/h of potassium phosphate) (excessively rapid replacement may precipitate hypocalcaemia) No clinical outcome benefit from replacement.
Hypoglycaemia	With overzealous insulin regimens	Frequent blood glucose monitoring (1–2 hourly) and addition of IV dextrose once CBS is <250 mg/dL
Thrombosis	Pro thrombotic state – dehydration, underlying inflammatory process	Prophylactic LMW heparin should be used
Normal anion gap metabolic acidosis	With large volumes of normal saline used in resuscitation – chloride excess leading to hyperchloremic metabolic acidosis	Judicious use of normal saline/ use of balanced crystalloids
Cerebral oedema	Due to osmotic shifts in brain, hypoperfusion and inflammatory responses with rapid shifts in osmolarity – rare in adults (common in children)	Low threshold for brain imaging with changes in mental state Mannitol infusion and mechanical ventilation can be used in treatment
Acute kidney injury	Pre-renal AKI – more common with older adults, higher osmolarity and higher admission glucose levels	Monitor renal function tests daily, Rehydration

22. DKA in special populations

- Frail/ older adults – mixed DKA/HHS and isolated HHS more frequent than DKA, precipitating factors and concurrent diagnoses (CV events, infections) common, recurrent DKA can occur
 - ESKD – concurrent fluid overload, greater hyperglycemia/ hyperkalemia, lower ketone concentration – careful fluid/ K administration, greater risk of cardiac complications
- Pregnancy – 2% of pregnancies with pregestational DM develop DKA (<0.1% in GDM) – may have euglycemic DKA, mixed acid base disturbances with hyperemesis - need expert opinion (significant fetomaternal risk)

23. How to prevent DKA?

- Assess precipitating causes of DKA and take appropriate measures
- Close follow up 2-4 weeks after discharge
- Patient education (before discharge) regarding current event and overall diabetes management, structured diabetes education programs, education on insulin administration
- 'sick day advice' – adequate hydration and calorie intake, monitor blood sugar frequently (2-4hrly if possible), do not skip insulin
- Ensure adequate supply of insulin on discharge

C. Hyperosmolar hyperglycaemic state (HHS)**24. What is HHS?**

HHS is characterised by severe hyperglycaemia and dehydration in the absence of significant ketoacidosis.

25. What is the pathophysiology of HHS?

- Relative insulin deficiency → Severe hyperglycemia (>600 mg/dL or >33.3 mmol/L) due to increased hepatic glucose production and decreased glucose utilization by peripheral tissues.
- Osmotic diuresis → Severe dehydration.
- Altered mental status due to hyperosmolality (>320 mOsm/kg).
- Residual insulin prevents significant ketogenesis.

26. When to suspect HHS in the clinical setting?**Symptoms**

- History of polyuria, polydipsia, weight loss, vomiting, dehydration
- Altered mental status, i.e., lethargy, profound coma

Signs

- Poor skin turgor, tachycardia, and hypotension
- Focal neurologic signs (hemianopia and hemiparesis) and seizures (focal or generalised)
- Fever due to underlying infection (although infection is a common precipitating factor, patients can be normothermic or even hypothermic due to peripheral vasodilation)

27. How to diagnose HHS?

Hyperglycaemia - Plasma glucose ≥ 600 mg/dL (33.3 mmol/L)

Hyperosmolality - Calculated effective serum osmolality >300 mOsm/kg (calculated as $[2 \times \text{Na}^+ (\text{mmol/L}) + \text{glucose} (\text{mmol/L})]$) OR total serum osmolality >320 mOsm/kg $[(2 \times \text{Na}^+ (\text{mmol/L}) + \text{glucose} (\text{mmol/L}) + \text{urea} (\text{mmol/L}))]$

β -Hydroxybutyrate concentration <3.0 mmol/L OR urine ketone strip $<2+$

Absence of acidosis - pH ≥ 7.3 and bicarbonate concentration ≥ 15 mmol/L

28. How to manage HHS?

Immediate management

Table 14.7 Immediate management of HHS

Clinical assessment	• Airway, breathing, circulation, disability, exposure • Large bore IV cannula for fluid replacement • Brief diabetes history e.g., (medications, precipitating cause) • Relevant clinical examination • Draw blood for urgent investigations
Urgent investigations	• CBG • Venous blood gas (VBG) • Plasma glucose • Serum or urine ketones • Serum electrolytes, creatinine • Serum osmolality • An electrocardiogram • Precipitating causes - ECG/ UFR/CRP /Blood /urine /sputum cultures /chest X-ray
Monitoring	• Hourly observation of hemodynamic parameters • Capillary blood glucose testing - 1 – 2 h using a hospital calibrated glucose meter • Electrolytes, creatinine, serum osmolality – every 4 hours - until resolution of HHS

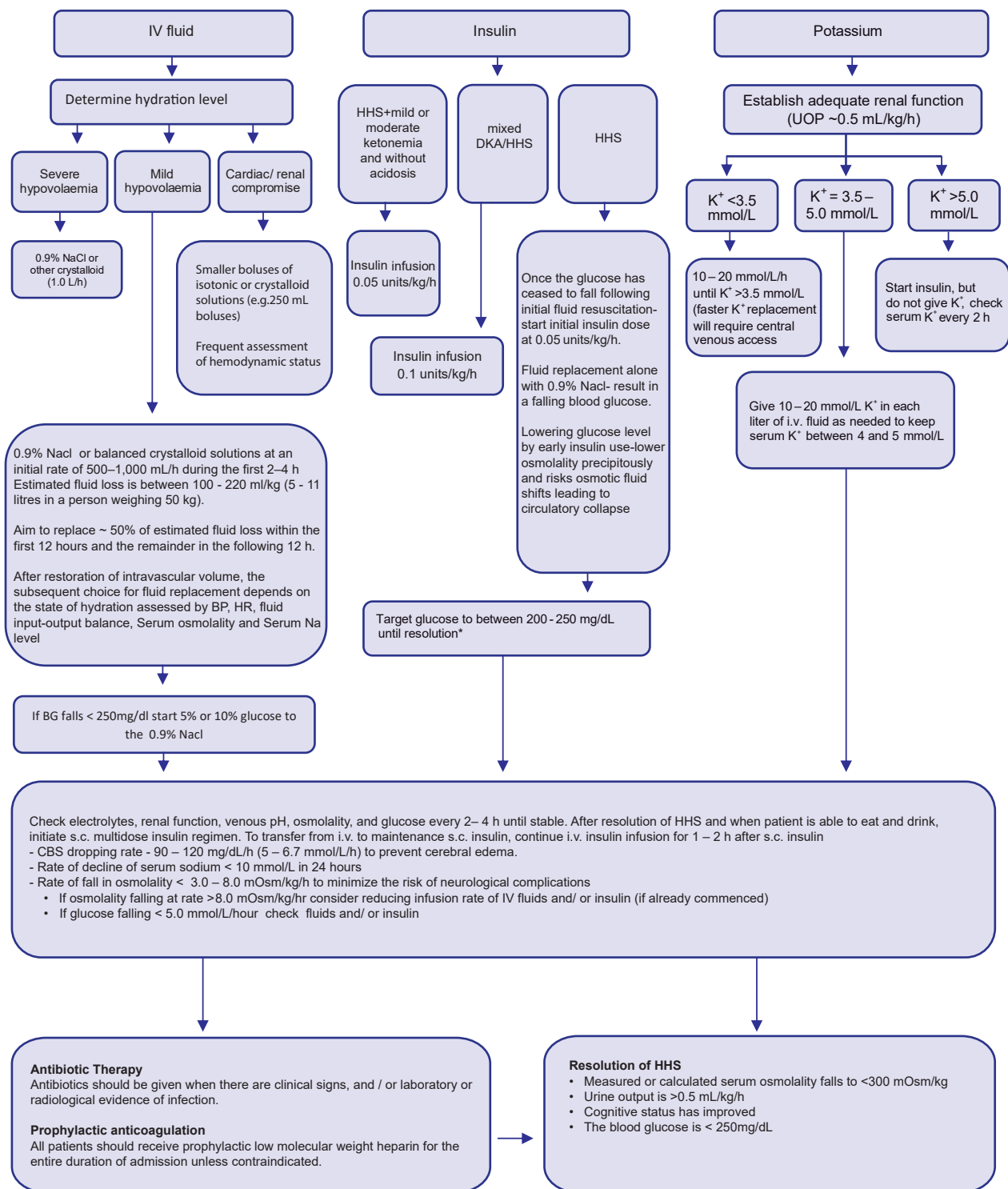


Figure 14.2 Management algorithm of HHS

29. What is the place for basal insulin?

- Basal insulin while on fixed rate insulin infusion may be continued during IV insulin infusion
- Can be given as once or twice daily doses, 0.15 – 3 U/kg/day.

30. What is the management after resolution of HHS?

- Start subcutaneous insulin 1 – 2 h prior to discontinuing IV insulin infusion
- Ensure insulin regimen provides 24h coverage
 - Basal insulin and rapid acting insulin analogs preferred
 - 3 methods are used at present to calculate total daily dose (TDD) – weight based estimates, preadmission insulin requirements, hourly IV insulin requirements

Patients with known diabetes may be given insulin at the dosage they were receiving before the admission

If inadequate baseline insulin therapy (i.e., high HbA1c) - change the dose accordingly

Weight-based formula - calculation using 0.5–0.6 units/kg/day

Risk factors for hypoglycaemia, including kidney failure or frailty a calculation using approximately 0.3 units/kg/day

Hourly intravenous insulin infusion rate

Summation of stable hourly insulin requirement may help to estimate TDD

Caution-insulin requirement over - estimated due to glucotoxicity.

References

1. Dhatariya Keran, Savage M, Claydon A. The management of diabetic ketoacidosis in adults. Joint British Diabetes Societies. 2013
2. American Diabetes Association. Diabetes Care in the Hospital: Standards of Medical Care in Diabetes—2025. Diabetes Care, 2025. 44(Supplement 1): p. S211-S220.
3. Dhatariya, K.K. and Umpierrez, G.E. (2017). Guidelines for management of diabetic ketoacidosis: time to revise? The Lancet Diabetes & Endocrinology, [online] 5(5), pp.321–323.
4. Acid–Base Problems in Diabetic Ketoacidosis. (2015). New England Journal of Medicine, 372(20), pp.1968–1970.
5. Umpierrez GE, Davis GM, ElSayed NA, et al. Hyperglycemic crises in adults with diabetes: a consensus report. Diabetes Care. 2024; 47(8):1257-1275.

15. DIABETES IN HOSPITALISED PATIENTS

A. Non-critically ill patients

1. How should a patient with diabetes be evaluated on admission to the hospital?

- Check random capillary blood glucose (CBG) on admission.
- Perform HbA1c if not done within past 3 months.
- Check fasting and post-prandial plasma glucose.
 - State the type of diabetes (type 1, type 2, gestational, pancreatogenic etc.) when known in the initial admission notes
 - Assess the diabetes self-management knowledge and behaviours on admission

2. How should a hospitalised adult without a previous diagnosis of diabetes be assessed?

- Perform opportunistic screening in all adults above 35 years on admission (refer to section 3).
 - In patients without prior diagnosis of diabetes, hyperglycaemia in hospitalized patients is defined when blood glucose levels > 140 mg/dL
 - Admission HbA1C > 6.5% in such patients suggests pre-existing diabetes

3. How frequently should CBG be monitored in patients with in-hospital hyperglycaemia?

- Test CBG before meals.
- Adjust monitoring frequency according to individual patient characteristics.

Table 15.1 Frequency of CBG monitoring in patients

More frequent	Less frequent
• Complex insulin regimens • High glucose variability	• Minimal glucose fluctuations • Regular eating patterns • Low risk of hypoglycaemia • Simple glucose lowering therapy regimen

- In patients using continuous glucose monitoring (CGM), it can be continued if clinically appropriate with confirmatory CBS measurements for insulin dosing decisions and hypoglycaemia assessment.

4. What are the CBG targets during hospitalisation?

- 100 - 180 mg/dL (5.6 - 10.0 mmol/L) for non-critically ill patients if this can be achieved without significant hypoglycaemia
- 140 - 180 mg/dL (7.8 - 10.0 mmol/L) for critically ill patients
- 110 - 140 mg/dL (6.1 - 7.8 mmol/L) for selected individuals (e.g., critically ill individuals undergoing cardiac surgery) if they can be achieved without significant hypoglycaemia
- 180 - 250 mg/dL (10.0 - 13.9 mmol/L) in patients with severe comorbidities (terminally ill individuals with short life expectancy, advanced kidney failure [and/or on dialysis], high risk for hypoglycaemia, and/or labile glycaemic excursions), and inpatient care settings where frequent glucose monitoring or close nursing supervision is not feasible
- Avoid hypoglycaemia

5. How should in-hospital hyperglycaemia be managed?

- If persistently hyperglycaemic (CBG >140 mg/dL):
 - Commence medical nutrition therapy.
 - Minimise medication causing hyperglycaemia.
 - Consider initiation of glucose lowering pharmacotherapy.
- Commence insulin if CBG is repeatedly > 180 mg/dL.
- Review and adjust patient's usual glucose lowering therapies (see below).

6. How to use insulin in hospitalised non-critically ill patients?

- Use basal bolus regimen (BBR) of insulin.
- Use isophane insulin (once or twice a day) or long-acting analogues for basal insulin requirement.
- Use human insulin or rapid acting insulin analogues as insulin boluses for prandial insulin requirement. These doses should align with the carbohydrate quantity of the meal.
- If oral intake is poor, consider administering prandial insulin immediately after meals, with the dose adjusted to the amount ingested.
- Pre-mixed insulin is not routinely recommended due to higher glucose fluctuations.
- Avoid sliding scale insulin regimen. It may be considered for not more than a day to gauge the insulin requirement in special situations.

7. How to determine the insulin dose?

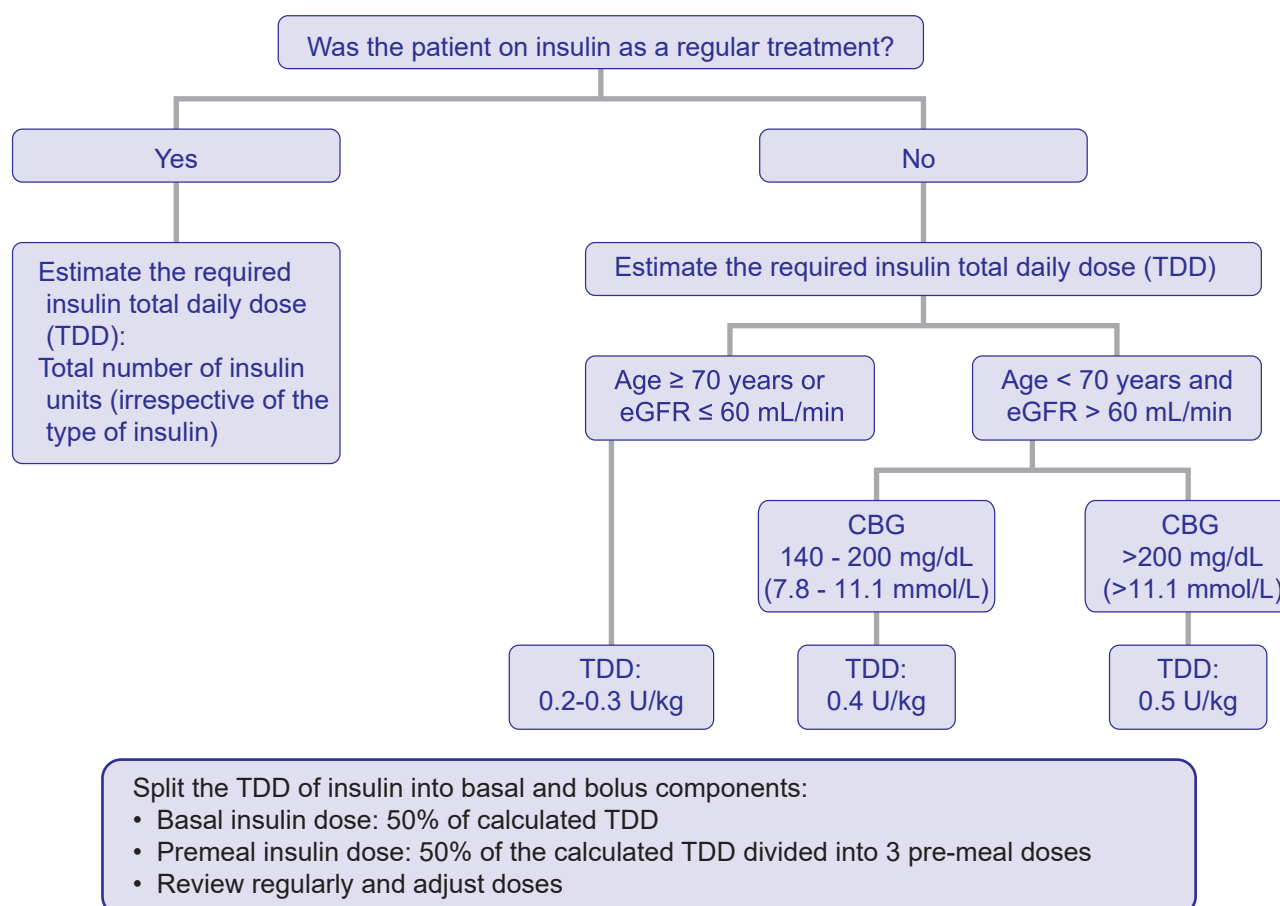


Figure 15.1 Determining the insulin dose requirement

8. How to manage persistent hyperglycaemia while on basal bolus regimen (BBR)?

- Add correction doses of short acting insulin to the premeal insulin bolus, according to pre-meal glucose level and likely insulin resistance (table 15.2)
- If the patient needs frequent addition of correction doses increase the basal insulin dose.
- If the patient is unable to take meals and blood glucose levels are higher than 180 mg/dL, give regular insulin 6 hourly or rapid acting insulin analogues 4 hourly. Decide the dose according to the 'insulin sensitive' column in table 15.2

Table 15.2 Correction insulin doses according to CBG values and insulin resistance

Blood glucose (mg/dL)	Correction dose (add to pre-planned premeal dose)		
	Insulin sensitive	Usual	Insulin resistant
140-180	2	4	6
181-220	4	6	8
221-260	6	8	10
261-300	8	10	12
301-350	10	12	14
351-400	12	14	16
>400	14	16	18

Insulin sensitive: TDD < 40 U; usual: TDD 40 - 80 U; insulin resistant: TDD > 80 U

9. How should oral glucose lowering drugs be modified?

Table 15.3 Use of oral glucose lowering drugs in hospitalised patients

Agent	Action
Metformin	Should be discontinued in patients at risk of lactic acidosis (acute kidney injury, severe organ impairment, hypoxia, shock) Should be discontinued 48 hours before an iodinated contrast procedure in patients with reduced eGFR (<60 mL/min per 1.73/m ²), history of liver disease, alcohol abuse, acute heart failure, or in those receiving intra-arterial contrast. Reassess renal function before restarting
Sulphonylurea	Caution in severe illness
SGLT2i	Avoid in severe illness
DPP-4i	Discontinue saxagliptin in heart failure
Thiozolidinedione	Discontinue pioglitazone in heart failure

10. What is the role of medical nutrition therapy (MNT) in hospitalised adults?

- Provide adequate calories to meet the increased metabolic demands of acute illness
- Minimize glycaemic fluctuations
- Address personal food preferences
- Needs to be individualised according to patient's needs

11. In which instances, patients are at high risk of hypoglycaemia and requires dose adjustments or frequent monitoring?

- Sudden reduction of corticosteroid dose
- Reduced oral intake or emesis
- Reduced infusion rate of intravenous dextrose
- Unexpected interruption of enteral or parenteral feeding
- Altered ability of patient to report symptoms of hypoglycaemia
- Inappropriate timing of short or rapid acting insulin doses in relation to meals

B. Critically ill patients

12. When should blood glucose be tested?

Perform random CBG in all patients hospitalised with a critical illness, irrespective of previous diagnosis of diabetes

13. What is the CBG target in a critically ill patient with diabetes / newly detected hyperglycaemia?

- 140-180 mg/dL (7.8 - 10.0 mmol/L)

14. How frequently should the CBG be monitored?

- In patients on intravenous insulin infusion, monitor CBG hourly until it is stable.
- Thereafter, monitor less frequently (2-4 hourly)

15. How to use insulin in critically ill hospitalised patients?

- Variable Rate Intravenous Insulin Infusion (VRIII). Follow unit protocols / algorithms to titrate infusion rate to maintain BG in the desired range. (annexure 15.1)
- In suitable patients, administration of low dose subcutaneous basal insulin analogue (0.15-0.3 units/kg) in addition to insulin infusion may reduce the duration of insulin infusion and length of hospital stay and prevent rebound hyperglycaemia without increased risk of hypoglycaemia

16. How should blood glucose control be achieved in patients with assisted feeding?

- Use NPH insulin (8-12 hourly) or long-acting analogues (insulin detemir 12 hourly or insulin glargine 24 hourly) to provide basal insulin requirement
- Decide insulin administration method for nutritional needs according to pattern of feeding (table 15.4)
- If continuous enteral feeding is interrupted, immediately commence intravenous 10% dextrose 50 mL/hour and titrate to avoid hypoglycaemia and select an appropriate insulin regimen
- Review the glucose control and adjust overall insulin doses daily

Table 15.4 Insulin therapy for patients on assisted feeding

	Bolus enteral tube feeding	Continuous enteral feeding	Intravenous feeding
Basal insulin requirement	Continue basal doses of insulin in all patients with T1D or insulin treated patients with T2D Do not stop basal insulin in patients with T1D even if feedings are discontinued		
Nutritional insulin requirement	Give 1 unit of regular insulin subcutaneously per 10 - 15 g of carbohydrates, before each main feed	Calculate daily nutritional insulin requirement (1 unit of regular insulin per 10 - 15 g of carbohydrate in daily meal plan) Provide this insulin requirement as intermediate acting insulin (2-3 divided doses) or a long-acting analogue (2 divided doses of insulin detemir or once daily dose of insulin glargine) administered subcutaneously*	Add 1 unit of regular insulin per 10 -15 g of carbohydrate into the intravenous nutrition infusion. This will safely avoid hypoglycaemia if the parenteral nutrition is interrupted.
Correctional insulin doses	Add correctional doses to pre-meal boluses according to table 15.2	Give regular insulin every 6 hourly or rapid acting insulin analogues every 4 hourly, subcutaneously. Decide the dose according to table 15.2, "insulin sensitive" column.	

* For patients receiving nocturnal tube feeding, NPH insulin administered with the initiation of feeding to cover this nutritional load

17. How to plan discharge?

Insulin

- Convert VRIII to BBR of subcutaneous insulin once the patient is taking meals orally.
- Switching from VRIII to BBR is best done at the breakfast or lunch.
- Administer subcutaneous basal insulin 2 hours before intravenous infusion is discontinued, with the aim of minimizing rebound hyperglycemia till the subcutaneous insulin action takes place.
- Monitor blood glucose for next 24 hours after withdrawing VRIII (at least 4 - 6 hourly).
- For transitioning, the total daily dose of subcutaneous insulin may be calculated based on the insulin infusion rate during the prior 6 – 8 hours when stable glycemic goals were achieved, based on prior home insulin dose, or following a weight-based approach
- Decide on home insulin regimen (BBR vs pre-mixed insulin twice a day vs basal insulin only) considering glucose control, risk of hypoglycaemia and patient's preference. It is best to maintain the home treatment regimen 1- 2 days prior to discharge.

Non-insulin therapies

- Patient's usual oral hypoglycaemics and GLP-1 RAs can be restarted if the patient had a good control with HbA1c of less than 7.5%. Restart usual treatment when a meal is due.
- Patient may need additional oral hypoglycaemic drugs if the initial control is poor with HbA1c more than 7.5%.

Patient education and follow-up

- Review the knowledge and address the following areas prior to hospital discharge: understanding related to the diabetes diagnosis, SMBG, prevention of hypoglycaemia and sick-day management.
- Crosscheck the patient's medications to ensure that no long term medications were stopped and to ensure the safety of new prescriptions.
- Schedule an outpatient followup visit with the patient's diabetes care provider in 2 - 4 weeks following discharge. Review earlier if glucose control is not optimal at discharge or if medications were changed.

C. Patients undergoing surgery

18. What are the components of preoperative assessment in a patients with diabetes?

- Diagnosis and type of diabetes
- Usual diabetes medications
- Current glycaemic control
- HbA1C level within last 3 months
- Evidence of end organ damage

19. What are the glucose targets for patients undergoing elective surgery?

- HbA1c - usually <8%
- Random plasma glucose – 100 - 180 mg/dL (depending on risk of hypoglycaemia, comorbidities, etc)

20. How to monitor glucose levels in the preoperative period?

- Monitor premeal CBG three times a day, before the three main meals.
- During the period of fasting, monitor CBG 4 - 6 hourly.

21. How to optimise the oral glucose lowering therapies in the preoperative period?

- Withhold the morning dose of oral glucose lowering therapies on the day of surgery.
- Commence insulin if preoperative CBG values are >180 mg/dL repeatedly (see below).
- Metformin should be withheld on the day of the surgery
- SGLT2 inhibitors should be discontinued 3 - 4 days before surgery

22. What are the factors and precautions to consider in patients on GLP-1 RA undergoing surgery?

- primary indication of these medications (e.g., diabetes or weight loss)
- current glycaemic management
- type of surgery or procedure and its urgency
- type of anaesthesia
- consideration of preoperative gastric ultrasound to quantify gastric contents
- implementation of full stomach precautions due to increased aspiration risk in surgery due to delayed gastric emptying.

23. How to adjust insulin therapy in the preoperative period?

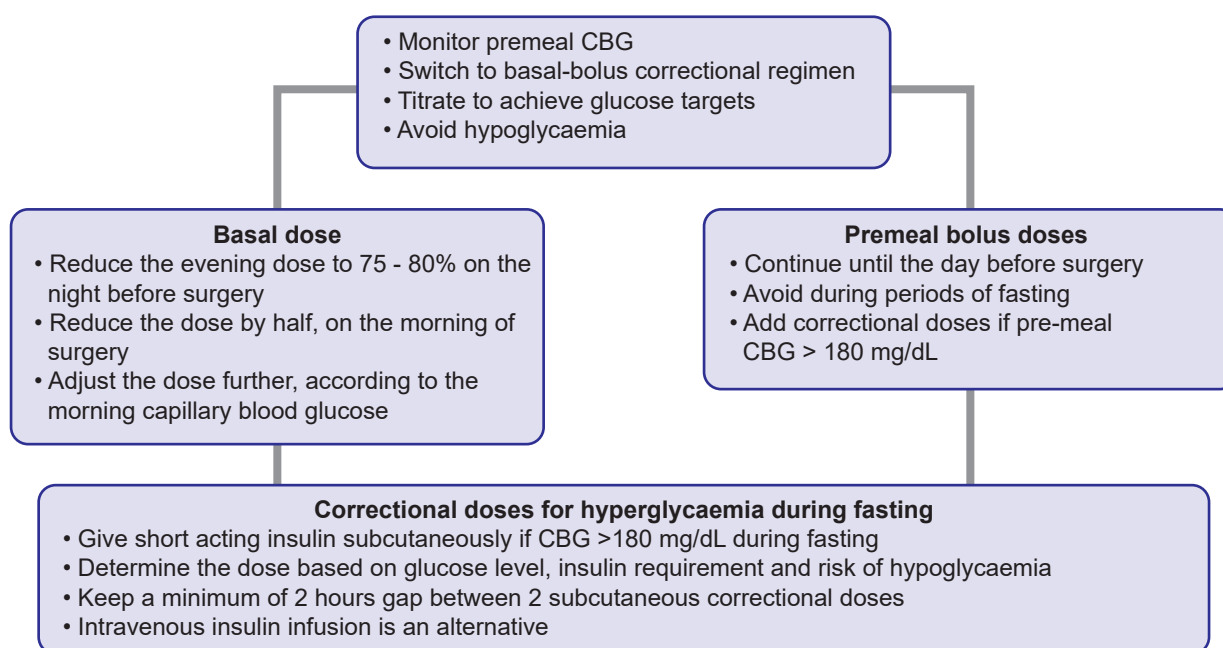


Figure 15.2. Adjusting insulin therapy in the immediate preoperative period

24. How to commence insulin in the preoperative period?

- Use basal bolus correctional regimen (see above)
- Determine the dose depending on age, insulin sensitivity, renal function and CBG values. Higher doses will be needed in patients with infections, obesity, planned for prolonged surgery or on glucocorticoid treatment.
- In the immediate preoperative stage if there is hyperglycaemia:
 - Short acting insulin is useful in short duration surgeries (<4 hours).
 - If prolonged fasting periods / more than one skipped meal is expected, consider intravenous fluids containing dextrose, potassium and variable rate insulin infusion (annexure 15.1).
(To set up VR/III - add 50 units of soluble insulin to 49.5 ml 0.9% saline. This will make 1 unit/mL insulin solution)
 - Titrate the dose according to the CBG values.

Table 15.5 Modifications of medications in the perioperative period

Medicine	Day prior to surgery	Patient for am surgery	Patient for pm surgery
Once daily long acting insulin (morning)	No dose adjustment	Give 80% of dose	Give 80% of dose
Once daily long acting insulin (evening)	Give 80% of dose	No dose adjustment	No dose adjustment
Twice daily long acting	- No dose adjustment for morning dose - Give 80% of the evening dose	- Give 80% of the morning dose - No dose adjustment for evening dose	- Give 80% of the morning dose - No dose adjustment for evening dose
Twice daily premixed insulin	No dose adjustment	- Give 50% of morning dose - If taking normal meal – give usual evening dose - If taking small meal – give 50% of evening dose - If not eating – give basal only component of the evening dose	- Give 50% of morning dose - If taking normal meal – give usual evening dose - If taking small meal – give 50% of evening dose - If not eating – give basal only component of the evening dose
Short acting insulin with meals	No dose adjustment	- Omit morning dose if no breakfast is eaten - Omit lunchtime dose if not eating - If taking normal – give usual evening dose - If taking small meal – give 50% of evening dose - If not eating – omit the evening dose	- Usual morning dose with breakfast - Omit lunchtime dose if not eating - If taking normal – give usual evening dose - If taking small meal – give 50% of evening dose - If not eating – omit the evening dose
Acarbose	Take as usual	Omit morning dose if not eating	Give morning dose if eating
Metformin	Take as usual	- If taken once or twice a day – take as usual - If taken three times per day – omit lunch time dose	- If taken once or twice a day – take as usual - If taken three times per day – omit lunch time dose
Sulphonylurea	Take as usual	- Omit morning dose - If taken twice daily – take evening dose if eating	Do not take
Pioglitazone	Take as usual	Take as usual	Take as usual
DPP4 inhibitors	Take as usual	Take as usual	Take as usual
GLP 1 RA	Take as usual	Take as usual	Take as usual
SGLT2 inhibitors	Omit	Omit	Omit

Source: Adapted from Guideline for Perioperative Care for people with Diabetes mellitus undergoing elective and emergency surgery by Centre for Perioperative Care, December 2022

25. How to manage a patient with type 1 diabetes preoperatively?

- Continue usual insulin doses and adjust according to CBG level.
- Consider switching to basal bolus regimen if not already on.
- Do not stop basal insulin as it will precipitate diabetic ketoacidosis.
- In case of ill patients, exclude diabetic ketoacidosis. If present, reschedule the surgery and manage DKA (section 14).
- See figure 15.2 for dose adjustments in the immediate preoperative stage and during fasting.

26. How to maintain glycaemic control in the intraoperative period?

- Monitor blood glucose levels hourly/ less frequently depending on the clinical circumstances.
- Maintain blood glucose within the target range (110 -180 mg/dL).
- Intravenous infusion of insulin depending on the blood glucose levels in case of prolonged surgery (>4hours) or prolonged starvation period (more than one missed meal): (annexure 15.1)
- Introduce intravenous dextrose infusion with potassium if the patient becomes hypoglycaemic.

27. How to manage a patient with diabetes in the postoperative period?

- If intravenous insulin infusion is used, continue with dextrose infusion until patient can take a meal.
- Give subcutaneous short acting insulin 30 min before stopping the insulin infusion.
- If the patient was previously on insulin, use the same regimen.
- If the patient was not on insulin previously, but was commenced in the immediate preoperative period, determine the subcutaneous insulin doses as follows:
 - Estimate the TDD of insulin (equivalent to the cumulative predicted insulin dose received in the past 24 hours. Adjust according to level of glucose control. Anticipate changes in insulin requirement with changing clinical status)
 - Administer 40 - 50% of TDD as basal insulin dose.
 - Divide the rest into premeal boluses.
 - Give correctional doses according to premeal CBG (table 15.2)
 - Review the glucose status and adjust basal and bolus doses regularly
- Oral glucose lowering therapies can be recommenced from the evening of the day of surgery, provided oral intake is permitted.

References

1. Intensive versus Conventional Glucose Control in Critically Ill Patients. (2009). *New England Journal of Medicine*, 360(13), 1283–1297. doi:10.1056/nejmoa0810625
2. Diabetes Care in the Hospital: Standards of Medical Care in Diabetes—2025. American Diabetes Association-Diabetes Care 2025
3. Pasquel, F., Lansang, M., Dhatariya, K. and Umpierrez, G., 2021. Management of diabetes and hyperglycaemia in the hospital. *The Lancet Diabetes & Endocrinology*, 9(3), pp.174-188
4. Aldam P, Levy N, Hall GM. Perioperative management of diabetic patients: new controversies. *British Journal of Anaesthesia*. 2014.
5. Duggan EW, Carlson K, Umpierrez GE. Perioperative Hyperglycemia Management: An Update. *Anesthesiology*. 2017.
6. Radhakutty A, Burt MG. Management of endocrine disease: Critical review of the evidence underlying management of glucocorticoid-induced hyperglycaemia. *European Journal of Endocrinology*. 2018.
7. Weerakkody, M.I. and Somasundaram, N.P., 2012. Glucocorticoid induced hyperglycaemia. *Sri Lanka Journal of Diabetes Endocrinology and Metabolism*, 2(1), pp.25–27. DOI: <http://doi.org/10.4038/sjdem.v2i1.4330a>
8. Centre for Perioperative Care. (2021). Guideline for perioperative care for people with diabetes mellitus undergoing elective and emergency surgery. Centre for Perioperative Care.

16. DIABETES IN SPECIAL GROUPS

A. Adolescents with type 2 diabetes

T2D in youth is different not only from T1Ds but also from T2D in adults and has unique features, such as a more rapidly progressive decline in beta cell function and accelerated development of diabetes complications.

1. Why should adolescents be screened for T2D?

- Undiagnosed T2D is rare in the adolescent population, but is increasing due to increasing rates of obesity in this population (figure 16.1).
- Median age of diagnosis of youth onset T2D is 13.5 years. The median age of onset is 1 year later in boys than in girls.

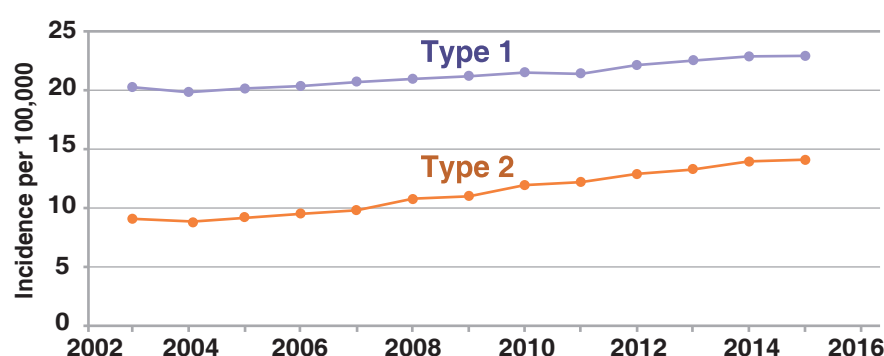


Figure 16.1 Trends in the incidence of type 1 and type 2 diabetes among youth < 20 years of age.

(Source: Centers for disease control and prevention (CDC). Available free of charge from <https://www.cdc.gov/diabetes/research/reports/children-diabetes-rates-rise.html>. Accessed on 10.02.2022)

2. When should an adolescent be screened for T2D?

- Screen children / adolescents after 10 years of age / after puberty (whichever is earlier) if at increased risk
 - Obesity (BMI $\geq$ 95th centile) / overweight (BMI $\geq$ 85th centile))
 - Strong family history of diabetes (possibly mediated by shared genetics, lifestyle, and environmental factors)
 - Foetal and early life influences (maternal gestational diabetes)
 - Adverse social determinants of health
- If initial screening is negative, repeat screening at a minimum of 2 year intervals and more frequently if BMI is increasing.

3. How to diagnose diabetes in adolescents?

- Apply adult diagnostic criteria to diagnose diabetes in adolescents (refer to section 3). Fasting plasma glucose, 2-h plasma glucose during 75g oral glucose tolerance test and HbA1C can be used to test for diabetes and prediabetes.
- Determine the type of diabetes (refer to section 2). Panel of pancreatic autoantibodies should be done to exclude the possibility of autoimmune type 1 diabetes.
- Screen adolescents with T2D for its complications after the diagnosis (refer to section 7).

4. What lifestyle interventions should be implemented in adolescents with T2D?

- Family education.
Weight loss in those with overweight or obesity – at least 7-10% decrease of weight should be targeted through developmentally and culturally appropriate comprehensive lifestyle programs.
- Dietary modification:
 - Restrict sugar-sweetened soft drinks and juices
 - Increase fruit, non-starchy vegetables, legumes, whole grains, nuts and seeds intake.

- Eat less meat and refined grains
- Reduce the use of processed, pre-packaged, and convenience foods.
- Reduce sedentary time, including TV, computer-related activities, texting, and video games. Screen time should be limited to less than 2 hours a day.
- Increase physical activity
Encourage at least 60 minutes of moderate to vigorous physical activity daily (with muscle and bone strength training at least 3 days/week)
Encourage engaging in sports.

5. What pharmacological therapies are available for treatment of T2D in adolescents?

- Metformin, insulin, GLP1 RA and empagliflozin can be used safely among adolescents.
- Determine initial treatment based on severity of hyperglycaemia:
 - Presenting with DKA/ HHS: basal bolus insulin regimen
 - HbA1c >8.5% or blood glucose > 250 mg/dL without DKA/ HHS symptomatic with polyuria/ polydipsia/ nocturia/ weight loss : basal insulin and metformin
 - HbA1c < 8.5% without DKA/ HHS: metformin monotherapy
- In patients not achieving glycaemic goals, maximize non insulin therapy (metformin, GLP-1 RA, empagliflozin) before initiating or intensifying insulin.
- In patients meeting glycaemic goals based on blood glucose monitoring, insulin can be tapered over 2-6 weeks by decreasing insulin dose 10-30% every few days.
- Regularly review and titrate the dose considering progression of puberty and rapid changes in weight in adolescents.

6. What is the role of metabolic surgery in adolescents?

- It can be considered in class 2 obesity or higher and who have elevated HbA1C and/ or serious comorbidity despite lifestyle and pharmacological treatment.
- However, long-term data on the rates of complications, reoperations, nutritional deficiencies, and diabetes recurrence are still needed.

7. What other challenges need specific assessment in the management of T2D among adolescents?

- Higher risk of
 - Mental health challenges - anxiety disorders, eating disorders, social isolation
- Transition to adult care
 - Need to work within a multidisciplinary team during transition
 - Allow transition from insulin therapy to newer oral or injectable medications

B. Older adults

8. Why should treatment of diabetes of elderly differ from that of young?

- Older adults are likely to have,
 - Different pharmacodynamics and pharmacokinetics of drugs
 - Co-morbidities including impaired renal and liver functions
 - Increased risk of hypoglycaemia and dehydration
 - Memory impairment
 - Higher risk of falls

9. How to assess an older adult to plan diabetes management?

- Assess medical, psychological, functional and social geriatric domains.
- Screen annually for geriatric syndromes (i.e., cognitive impairment, depression, urinary incontinence, falls, polypharmacy, and persistent pain)
- Individualise screening for diabetes complications.
 - Pay particular attention to complications that would lead to functional impairment.
- Assess hypoglycaemia risk at all routine visits.

Screen for early detection of mild cognitive impairment or dementia for adults over 65 years of age annually. Mini-Mental State Examination, Mini-Cog and the Montreal Cognitive Assessment can be used.

10. How to determine treatment targets in older adults?

- Individualise glucose targets:
 - Consider comorbidities, functional status, cognitive function, life expectancy and risk of hypoglycaemia (refer to table 16.1)
- Individualise blood pressure targets:
 - Consider adverse effects, postural hypotension and risk of falls
- Individualise treatment of cardiovascular risk factors (e.g., aspirin and statin therapy):
 - Consider the timeframe of benefit and risks

11. How should pharmacotherapy be approached?

- Use medication classes with low risk of hypoglycaemia.
- Consider adverse effects of medications
 - Hypoglycaemia with sulfonylureas and insulin
 - Volume depletion with SGLT2i
 - Gastrointestinal adverse effects of metformin and GLP-1 RA
- Consider less intensive and simple regimes with lower risk of hypoglycaemia and lower pill burden if it can be achieved within the individualised HbA1c target (refer to table 16.1).

12. How can a complex insulin regimen be simplified in a frail patient?

- Give the basal insulin dose in the morning.
- In patients receiving premixed insulin twice a day
 - Convert to once-a-day morning basal regimen, or,
 - Consider a higher proportion in the morning.
- Consider de-escalating to oral glucose lowering therapies where possible.

Table 16.1 Treatment goals for glycaemia, blood pressure, and dyslipidaemia in older adults with diabetes

Patient characteristics	Rationale	Reasonable HbA1c goals	Fasting or premeal glucose (mg/dL)	Bedtime glucose (mg/dL)	Blood pressure mmHg	Lipids
Healthy (few chronic illnesses, intact cognitive and functional status)	Longer remaining life expectancy	<7.0 - 7.5%	80 - 130	80 - 180	<130/80	Statin unless contraindicated or not tolerated
Complex / intermediate (multiple coexisting chronic illnesses or ADL impairment or mild-moderate cognitive impairment)	Intermediate life expectancy, high treatment burden, hypoglycaemia vulnerability, falls risk	<8.0%	90 - 150	100 - 180	<130/80	Statin unless contraindicated or not tolerated
Very complex / poor health (end-stage chronic illnesses or moderate-severe cognitive impairment or severe ADL impairment)	Limited remaining life expectancy makes benefit uncertain	Avoid reliance on HbA1c; Avoid hypoglycaemia and symptomatic hyperglycaemia	100 - 180	110 - 200	<140/90	Consider likelihood of benefit with statin

Source: adapted from ADA standards of medical care in diabetes- 2025

C. Chronic liver disease

13. Why is diabetes management important in patients with chronic liver disease?

- About 30% of patients with liver cirrhosis have overt diabetes. It is associated with an increased risk of hepatic complications and death.

14. What are the changes in glucose homeostasis takes place in chronic liver disease

- Risk of hypoglycaemia is increased by reduced metabolism of insulin by the liver and reduced gluconeogenic capacity of the liver especially in decompensated liver disease.
- Requirement for insulin can be increased due to insulin resistance, hyperglucagonaemia and decreased hepatic clearance of portal vein glucose.

15. What are the diagnostic tests used in chronic liver disease for the diagnosis of diabetes?

- Child A chronic liver disease: HbA1C, fasting plasma glucose and oral glucose tolerance test can be used.
- Child B and C chronic liver disease: fasting plasma glucose and oral glucose tolerance test can be used.
- Child B and C chronic liver disease or anaemia: HbA1C is not recommended due to the risk of underestimation leading to false negative results

Table 16.2 Monitoring of glycaemic control in chronic liver disease

Child Pugh stage	Assessment of glycaemic control	Glycaemic target
A	HbA1C	Personalized HbA1C target as in chapter 8
B	Self monitoring of blood glucose	Preprandial plasma glucose 100-200 mg/dL
C	Self monitoring of blood glucose	Preprandial plasma glucose 100-200 mg/dL

16. Are non-insulin hypoglycaemic drugs safe in chronic liver disease?

Table 16.3 Non-insulin hypoglycaemic drugs in patients with cirrhosis.

Medication / class	Child-Pugh stage			Comments
	A	B	C	
Metformin	Can use	Use with caution / Avoid use	Avoid use	Maximum dose 1500 mg/day Some studies suggest metformin reduce hepatocellular carcinoma occurrence in patients with cirrhosis
Sulfonylureas	Use with caution / Avoid use	Use with caution / Avoid use	Avoid use	Greater risk of hypoglycaemia
Pioglitazone	Can use	Avoid use	Avoid use	Avoid when liver enzymes >3 times ULN or if patient has oedema
SGLT2i	Can use	Use with caution	Avoid use	Caution is advised in hypotension and dehydration
GLP1-RA	Can use	Use with caution	Avoid use	
DPP-4i	Can use	Use with caution	Avoid use	Linagliptin is metabolized in the liver.
Alpha glucosidase inhibitors	Can use	Can use	Avoid use	

17. How should insulin be used?

- Insulin can be used at any stage of chronic liver disease regardless of degree of liver function impairment.
- The dose of insulin should be titrated to reduce risk of hypoglycaemia.
- Recommended insulin regimen is basal insulin alone or combined with prandial insulin
- Insulin analogues are preferred in patients with chronic liver disease as their pharmacokinetics are less affected and confer a lower risk of hypoglycaemia.

18. What is the management of metabolic dysfunction- associated steatotic liver disease?

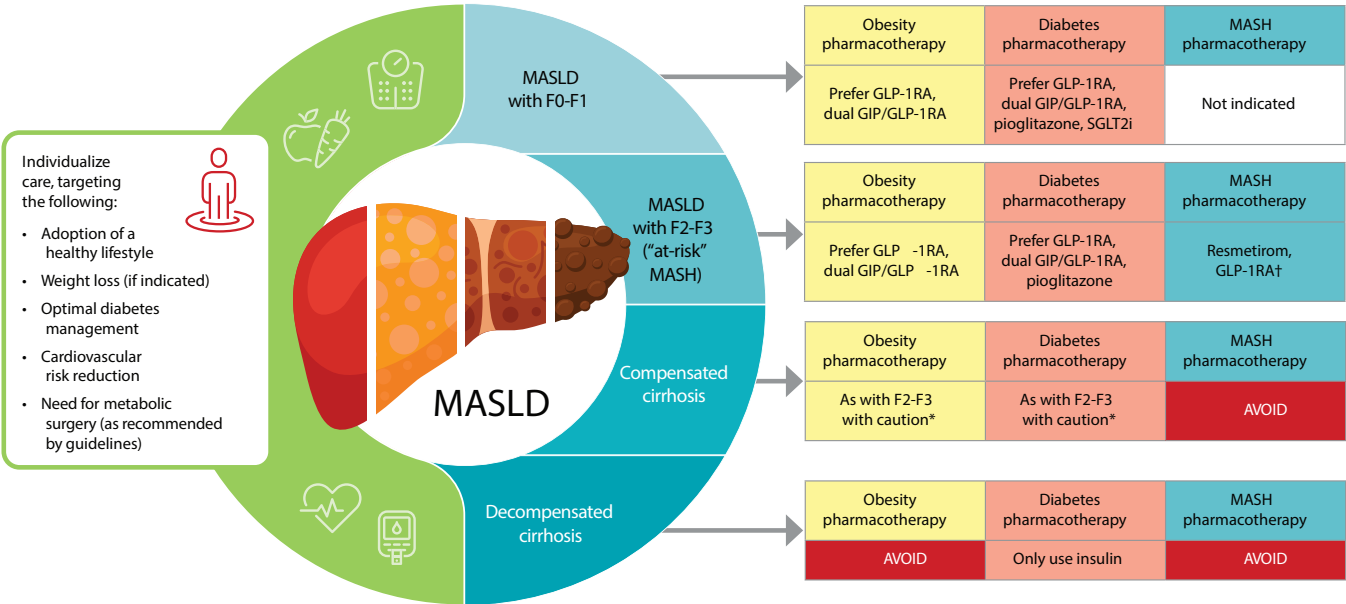


Figure 16.2 MASLD treatment algorithm for individuals with prediabetes or diabetes.

Source: A Consensus Report of the American Diabetes Association. Diabetes Care 20 June 2025; 48 (7): 1057–1082. <https://doi.org/10.2337/dci24-0094>

D. Patients receiving glucocorticoids

19. What are the risk factors for developing hyperglycaemia with steroid therapy?

- Pre-existing type 1 or type 2 diabetes
 - Family history of type 2 diabetes
 - Previous gestational diabetes
 - Previous impaired fasting glucose or impaired glucose tolerance
 - Polycystic ovary syndrome
 - Obesity
- Blood glucose level rises 4–8 hours after the administration of glucocorticoids
 - 50% individuals on glucocorticoids develop new-onset hyperglycaemia. 36% of them will progress to have steroid induced diabetes, persisting after its discontinuation.

20. How should steroid induced hyperglycaemia (SIHG) be managed in hospitalised adults?

- Assess risk of developing SIHG in all patients when starting on glucocorticoids.
- Perform HbA1c to exclude pre-existing diabetes.
- Monitor BG after starting steroids and treat accordingly (figure 16.3)
- CBG targets are as follows:
 - 100 - 180 mg/dL is ideal
 - 70 - 200 mg/dL is acceptable
 - 100 - 270 mg/dL and symptom relief for patients receiving end-of-life care.

21. How to manage patients with diabetes who are commenced on steroid treatment?

- Monitor CBG 4 times a day (before main meals and at bedtime).
- See figure 16.4 for adjustments of treatment.
- In all patients with T1D and in patients with insulin treated T2D, rule out diabetic ketoacidosis (DKA), especially if CBG > 250 mg/dL or critically ill or develops clinical features of DKA.

22. How to plan discharge of a patient treated with steroid-induced hyperglycaemia while in hospital?

For all patients

- Educate on SMBG, risk of hypoglycaemia with steroid dose reduction and need for dose titration of glucose lowering therapies.

Previously treated diabetes

- Titrate the doses of glucose lowering therapies to maintain desired glycaemic targets.
- Educate the patient on medication adjustments.
- Review within 1-2 weeks with SMBG chart or postprandial plasma glucose values.
- Review earlier if recurrent hypoglycaemia or hyperglycaemia develops.

Newly commenced on glucose lowering therapies

- If steroids are stopped, discontinue glucose lowering therapies. Review SMBG or PPPG $\pm$ FPG in 2-4 weeks. If hyperglycaemia persists, commence treatment for new onset diabetes
- If steroid therapy is continued, continue newly commenced glucose lowering therapies and review SMBG or PPPG $\pm$ FPG in 1-2 weeks.
- Review earlier if recurrent hypoglycaemia or hyperglycaemia develops.

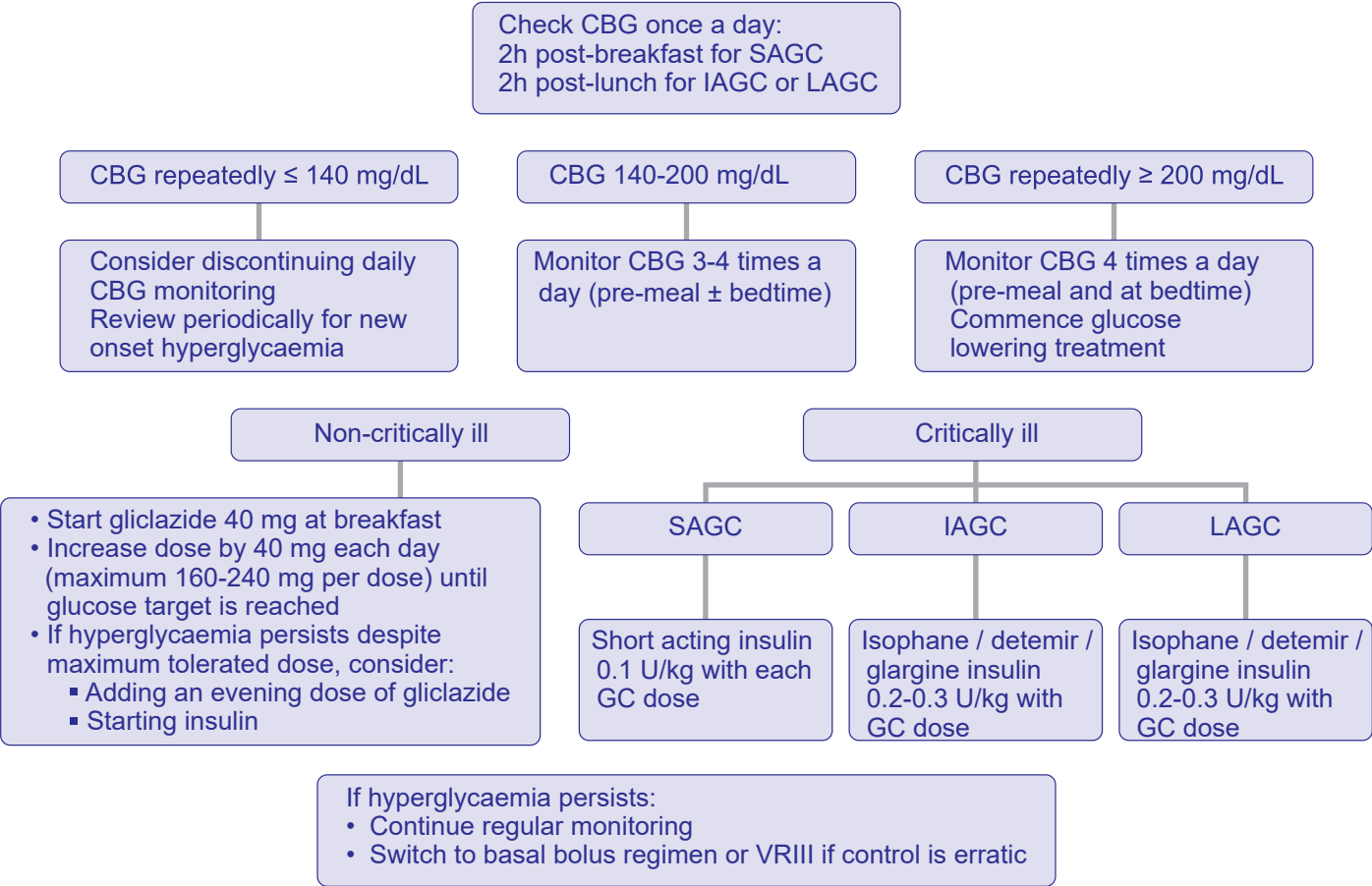


Figure 16.3 Management of SIHG in hospitalised patients without previously treated diabetes

CBG: capillary blood glucose; GC: glucocorticoid; IAGC: intermediate acting glucocorticoids (e.g., prednisolone, methylprednisolone); LAGC: long-acting glucocorticoids (e.g., dexamethasone); SAGC: short-acting glucocorticoids (e.g., hydrocortisone), VRIII: variable rate intravenous insulin infusion.

(Adapted from Diabetes UK Position Statements Management of hyperglycaemia and steroid (glucocorticoid) therapy: a guideline from the Joint British Diabetes Societies (JBDS) for Inpatient Care group and Aberer et al. A Practical Guide for the Management of Steroid Induced Hyperglycaemia in the Hospital. J. Clin. Med. 2021, 10, 2154)

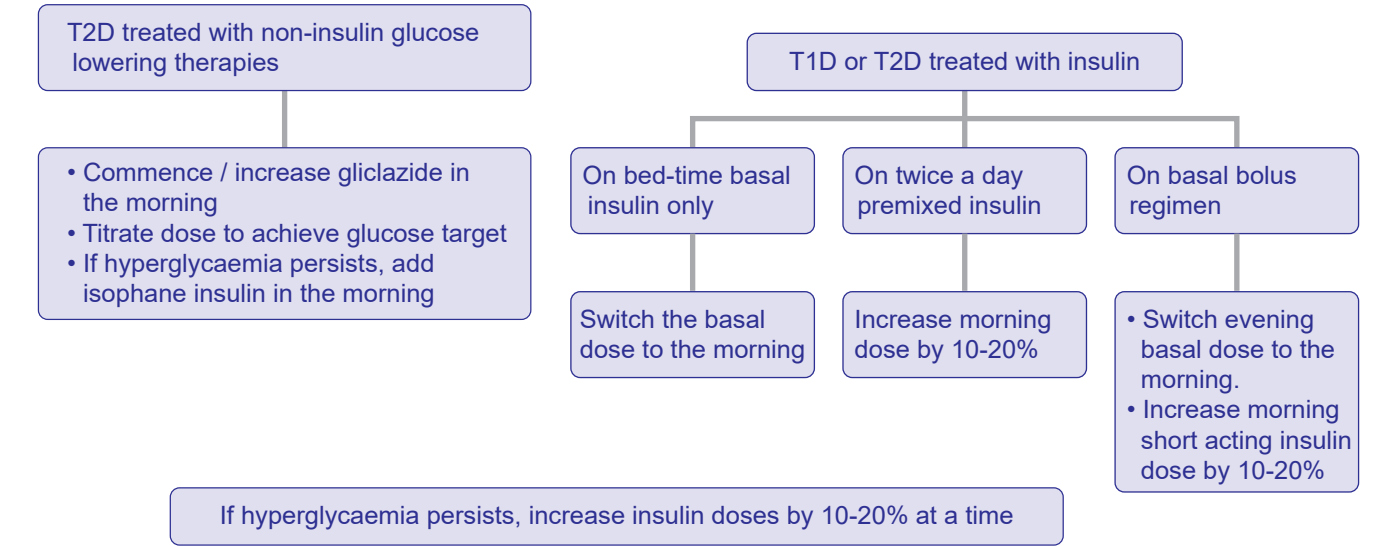


Figure 16.4 Management of hyperglycaemia in people with pre-existing diabetes commenced on steroids

(adapted from Diabetes UK Position Statements Management of hyperglycaemia and steroid (glucocorticoid) therapy: a guideline from the Joint British Diabetes Societies (JBDS) for Inpatient Care group)

23. How should SIHG be managed in non-hospitalised adults commenced on glucocorticoids?

- Screen for risk of developing SIHG before starting steroids.
- In individuals at risk of developing SIHG, encourage daily capillary glucose monitoring (before and 2h after lunch).
- If this is not feasible, consider reviewing post-prandial plasma glucose within a few days or hospital admission if acutely ill or having symptoms of hyperglycaemia.
- If SIHG develops, commence gliclazide or insulin (refer to figure 16.3).

E. Religious concerns**i. Buddhist monastics****24. What factors need to be considered when treating monastic monks with diabetes?**

- Cultural and religious background should be explored actively and respected during management.
- Treatment should be tailor-made to suit their dietary and lifestyle habits.
- Most Buddhist monks take only two main meals before dawn and at noon. There is a risk of hypoglycaemia during fasting stage and risk of hyperglycaemia and obesity due to consumption of large main meals. Some tend to consume high calorie drinks between noon and dawn. As they depend on alms offered by the devotees, there is difficulty in opting for healthy food and hence regulating calorie intake.
- Meditation practises among Buddhist monks which have benefits in managing chronic disorders, should be encouraged.

25. What are the lifestyle and pharmacological strategies recommended when treating monastics with diabetes?**Table 16.4** Strategies for glucose control in Buddhist monastics with diabetes

Diet	
	A consistent diet based on the healthy plate model should be adhered to. Both monastics and devotees who prepare meals need to be educated on a healthy diet and food preparation techniques. Consumption of high calorie drinks from noon to dawn should be avoided. They may consume sugar free drinks which are allowed by Vinaya rules such as tea, lime, lemon, Ceylon olive etc.
Physical Activity	
	Advise to engage in regular physical activities such as brisk walking and sweeping at least 150 min per week
Medications	
Metformin	First line therapy Low risk of hypoglycaemia Not necessarily given with meals May increase up to maximum tolerated dose Extended preparation may be used once or twice daily
Sulphonylurea	Increased risk of hypoglycaemia May use gliclazide or glimepiride which has low hypoglycaemic risk Need to be given with meals If night dose is given, ensure adequate calorie intake
DPP-4i	Low risk of hypoglycaemia Weight neutral Not necessarily given with meals Once daily dose May increase up to maximum tolerated effective dose

SGLT2i	Low risk of hypoglycaemia Ensure adequate fluid intake to prevent dehydration and ketoacidosis during the prolonged fast Inquire about genital mycotic infections, urinary tract infections and prostatic symptoms before prescribing
Insulin	Self-monitoring of blood glucose is encouraged Long acting insulin analogues can be given at any time of the day as basal insulin Rapid acting insulin can be added to the main meals Premixed insulin morning dose can be given with the meal Ensure adequate calorie intake if adding insulin at night
GLP-1 RA	Low risk of hypoglycaemia Once daily oral and once daily or once weekly subcutaneous injectable preparations Not needed to be given with meals

26. When to discourage their usual eating patterns?

Monastic monks may not be able to adhere to their rituals in instances such as acute illness, end stage kidney disease and during periods of poor blood glucose control. Any such changes to their eating pattern are better made in agreement with the monks, respecting their beliefs.

ii. Ramadan fasting

27. How to assess and manage a patient pre-Ramadan?

- Commence assessment six to eight weeks before the start of Ramadan
- Consider these aspects:
 - type, duration and control of diabetes
 - presence of comorbidities
 - prior experience in managing diabetes during Ramadan fasting
 - ability to self-manage diabetes
 - contraindications for fasting
- Education - risk identification, role of SMBG, when to break the fast, when to exercise, fluids and meal planning and medication adjustment during fasting.

28. Who are the very high-risk patients in whom fasting is generally not recommended?

- Severe hypoglycemia within the 3 months prior to Ramadan
- Severe hyperglycemia with average fasting or premeal plasma glucose > 300 mg/dL or HbA1c >10%
- A history of recurrent hypoglycemia or hypoglycemia unawareness
- Diabetic ketoacidosis/ hyperosmolar hyperglycemic state within the 3 months prior to Ramadan
- Acute illness
- Performing intense physical labour
- Pregnancy
- Chronic dialysis
- Patients with significant dementia or cognitive deficits

29. How to monitor blood glucose during Ramadan fasting?

- Practice seven-point SMBG :
 - Pre-dawn meal (suhoor)
 - Morning
 - Mid-day
 - Mid-afternoon
 - Pre-sunset meal (iftar)
 - 2 hours after iftar
 - At any time when there are symptoms of hypoglycemia/ hyperglycemia or feeling unwell.

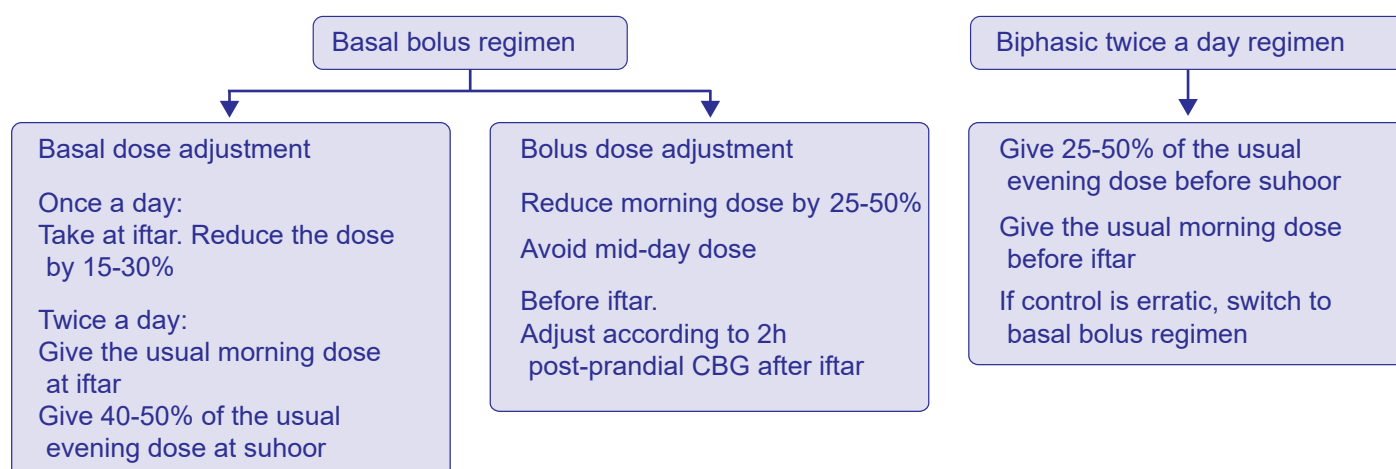
30. When should fasting be discontinued?

- All individuals should break their fast if:
 - blood glucose is less than 70 mg/dL (recheck within 1 hour if blood glucose is within 70 – 90 mg/dL)
 - blood glucose is more than 300 mg/dL
 - symptoms of hypoglycemia, hyperglycemia or dehydration
 - any acute illness occurs

31. What dietary advice should be given to people with diabetes during Ramadan fasting?

- Divide the daily calories between suhoor and iftar, plus one to two snacks if necessary
- Ensure meals are well balanced
 - 45-50% complex carbohydrates e.g., barley, wheat, oats, millet, semolina, beans, lentils
 - 20 – 30% proteins
 - < 35% fat (preferably mono-and poly unsaturated)
- Include low glycemic index, high fiber foods that release energy slowly before and after fasting e.g., granary bread, beans, rice
- Include plenty of fruits, vegetables, and salads
- Minimise foods that are high in saturated fats e.g., ghee, samosas, pakoras
- Avoid sugary desserts
- Use small amount of oil when cooking e.g., olive, canola oil, rapeseed oil
- Keep hydrated between sunset and sunrise by drinking water and other non-sweetened beverages
- Avoid caffeinated and sweetened drinks

32. How to adjust the insulin doses in an adult during Ramadan fasting



- Adjust doses regularly guided by CBG values.

29. How to adjust the sulfonylurea doses in patients with diabetes during Ramadan fasting?

Table 16.5 Sulphonylurea dose adjustment during Ramadan fasting

Once daily dosing	Prescribe at iftar	In individuals with well-controlled BG levels, the dose may be reduced
Twice daily dosing	Iftar dose could remain the same	In individuals with well-controlled BG levels, the suhoor dose should be reduced
Older generation SU	Carries a higher risk of hypoglycemia and should be avoided (e.g., glibenclamide)	Newer generation SU such as gliclazide, gliclazide MR, glimepiride should be used instead

30. How to adjust the doses of other oral glucose lowering therapies during Ramadan fasting?

Table 16.6 Non- sulphonylurea oral hypoglycaemic agent adjustment during Ramadan fasting

Metformin	Prescribe at meal times. May combine noon and evening doses into a single dose at iftar. Slow-release preparations need no adjustments.
Acarbose	Prescribe to take with meals.
Thiazolidinediones	Due to the low risk of hypoglycemia with pioglitazone, no dose modification is required during Ramadan, but dose should be taken at iftar.
GLP-1 RA	Titrate the dose prior to Ramadan (at least 2-4 weeks), no further treatment modifications are required during Ramadan.
DPP4i	Do not require treatment modifications during Ramadan.
SGLT2i	Low risk of hypoglycemia. No dose adjustments are required during Ramadan.

F. Patients with tuberculosis

Diabetes increases the risk of developing active tuberculosis by three-folds.

Tuberculosis worsens glycaemic control in people with diabetes.

31. How to approach diabetes management in patients with tuberculosis?

- Glycaemic target of HbA1C <7% is generally recommended. More lenient HbA1C target (<8%) might be assigned to people with markedly elevated glucose levels.
- Tailored advice on healthy diet and regular physical activity should be provided as appropriate.
- Metformin is the recommended initial glucose-lowering medication. A second-generation sulfonylurea (preferably gliclazide) may be used as initial first-line treatment when metformin is contraindicated or not tolerated.
- Initiate insulin therapy using basal bolus regime or premixed insulin
 - Higher insulin doses may be required initially (e.g., 1.0 U/kg/day)
 - Insulin requirement will decrease when tuberculosis improves (e.g., 0.5 U/kg/day)
 - Titrate according to individual's oral intake
- Screen for adrenal insufficiency if hypoglycaemia occurs frequently
- Prescribe pyridoxine to prevent neuropathy if isoniazid is used
- Consider oral hypoglycaemics when the patient shows clinical improvement.
- Eye examination is recommended before and during TB treatment.

32. What drug interactions should be considered before prescribing treatment for diabetes?

- Rifampicin is a potent hepatic enzyme-inducer. It accelerates the metabolism of sulfonylureas and metformin, and lowers their plasma levels. Therefore, it may cause hyperglycaemia in patients who are treated with these drugs.
- Glucose lowering effect of metformin may be increased by rifampicin.
- Isoniazid antagonizes the action of sulfonylureas and worsens the glycaemic control. It also impairs the release and action of insulin leading to hyperglycaemia.
- Monitor blood glucose when on these drugs.

33. What are the side effects of tuberculosis treatment that should be looked out for in patients with diabetes?

- Ethambutol may increase ocular neuropathy. Linezolid may worsen retinopathy or maculopathy.
- Linezolid, isoniazid, cycloserine can exacerbate optic neuritis and peripheral neuropathy.

G. Patients with HIV infection**34. How to diagnose diabetes in people living with HIV?**

- Perform fasting plasma glucose prior to anti-retroviral therapy (ART) initiation, at the time of switching antiretroviral therapy, 4 to 6 months after starting ART or switching ART.
- If initial testing is negative, fasting plasma glucose should be checked annually.
- However, HbA1c may underestimate the glycaemic status.

Consider OGTT in patients with prediabetes.

HbA1c can underestimate the level of glycaemia in the following circumstance, due to low grade haemolysis:

- CD4 counts <200 cells/mm³
- Increased mean cell volume (MCV); (greater discordance with higher MCV)
- ART use
 - Protease inhibitors
 - Azidothymidine (AZT)

35. What are the pharmacological considerations in people living with diabetes and HIV?

Table 16.7 Pharmacological considerations in prescribing glucose lowering therapies for people on ART

Drug class	Effect
Metformin	Dolutegravir increases metformin concentration Maximal recommended dose in people receiving dolutegravir is 1000mg daily
Sulfonylurea	No special consideration
DPP-4i	Gliptins have molecular target on immune cells. However no evidence that gliptin use changes CD4 or HIV RNA
SGLT2i	If UDP-glucuronosyl transferase enzyme inducers (ritonavir) are co-administered with canagliflozin, consider increasing the dose to 300mg
Thiazolidinediones	When used with CYP2C8 inhibitors (protease inhibitor), pioglitazone levels may increase Pioglitazone might have beneficial effect on HIV-associated lipodystrophy syndrome (HALS)
GLP-1 RA	No special consideration

36. How to select statins in people living with HIV and diabetes?

Multiple drug interactions between statin therapy and antiretroviral therapy exist. Evaluate the risk in individual patients.

Table 16.8 Risk of interactions between statins and anti-retroviral therapies

Low risk	Uncertain risk	High risk – avoid use
Pravastatin Fluvastatin Pitavastatin	Atorvastatin Rosuvastatin Fibrate in combination with statins	Simvastatin Lovastatin

- For people with HIV who have low-to-moderate (<20%) 10 year atherosclerotic cardiovascular disease (ASCVD) risk estimate
- Age 40-75 years with 5 - 20% ASCVD risk, recommend initiating moderate intensity statin therapy, which includes
 - atorvastatin 20 mg once daily
 - rosuvastatin 10 mg once daily
- Age 40-75 years with <5% ASCVD risk, consider initiating moderate intensity statin after considering other HIV related risk factors which increase ASCVD risk
- Age < 40 years – insufficient data to recommend for or against statin therapy for primary prevention of ASCVD

H . End of life care

37. What factors need to be considered when managing patients with diabetes who are receiving end of life care?

- Ensure that effective symptom control is provided.
- Avoid metabolic decompensation and diabetes-related emergencies:
 - Frequent hypoglycaemia
 - Diabetic ketoacidosis
 - Hyperosmolar hyperglycaemic state
 - Persistent symptomatic hyperglycaemia
- Avoid foot complications and pressure sores in frail, bedbound individuals with diabetes.
- Avoid symptomatic clinical dehydration.
- Ensure dignity and quality of life

38. How should glucose control be monitored and what is the glycaemic target?

- The aim is to prevent hyperglycaemia or hypoglycaemia. Glucose target may be kept between 110mg/dL and 270 mg/dL.
- In patients with organ failure try to make them free of hypoglycaemia and persistent symptomatic hyperglycaemia.
- Monitoring of these patients should target the minimum required investigations to reduce the distress and inconvenience to the patient and care givers.
- Plasma blood glucose or CBG can be measured infrequently to maintain blood glucose levels in the expected range.
- HbA1c is usually not indicated.
- When venepuncture or finger-stick is not welcome by the patient or difficult to perform, testing urine sugar may be acceptable.

39. What are the important factors in pharmacological management?

- Minimise pill burden
- Drugs used for long-term cardiovascular benefits are best withdrawn.
- Change drug doses according to renal function.
- Choose the simplest regime when switching to insulin; once or twice daily insulin.
- Basal insulin may be combined with oral hypoglycaemics.
- Consider using analogue insulin if risk of hypoglycaemia is high.
- Do not stop basal insulin in people with type 1 diabetes.

Table 16.9 Use of glucose lowering therapy in end-of-life care

Agent	Consideration
Metformin	No risk of hypoglycaemia. Dose reduction or withdrawal needed in patients with loss of appetite and gastrointestinal disturbances. Review the dose according to the renal function. Avoid when eGFR <30 mL/min and in critical illness due to risk of lactic acidosis.
Sulfonylureas	High risk of hypoglycaemia, especially with lower food intake and renal dysfunction. Use lower doses of short acting agents with lower risk of hypoglycaemia (gliclazide, glimepiride). Reduce doses in renal failure and liver dysfunction
DPP-4i	Avoid in pancreatic disease. Adjust dose and withdraw sitagliptin when needed in renal disease. Linagliptin does not need dose adjustment in renal impairment.
Thiazolidinediones	Causes fluid retention and worsens heart failure. Avoid in patients with oedema or heart failure
SGLT2i	Avoid in severe renal impairment (eGFR<30 mL/min). Stop if concerns of dehydration, foot ulceration or risk of genital infections arise. Risk of ketoacidosis would be higher in individuals with starvation or any acute illness.
Insulin	Dose adjustment with reduced oral intake and organ impairment to avoid hypoglycaemia. Basal only or pre-mixed twice daily regimes preferred over basal bolus for convenience. Even in type 1 diabetes basal only may be adequate to prevent diabetic ketoacidosis in terminal stages with reduced oral intake
GLP-1 RA	Dose reduction or withdrawal in patients with gastrointestinal disturbances, loss of appetite and loss of weight. Avoid in pancreatic disease

40. When should withdrawal of diabetes pharmacotherapy be considered?

- A dying patient (type 1 diabetes would be an exception)
- Frequent hypoglycaemia with small doses of medications
- When patient is withdrawn from therapeutic feeding and hydration
- Very low calorie intake
- Diabetes is controlled with minimal doses of oral hypoglycaemic agents

41. What are the dietary and nutritional recommendations?

- Food restriction is discouraged. Ensure adequate calorie intake.
- Allow any food item of their preference.
- Considering the small volume of food consumption, high calorie meals including sugary food can be allowed despite their adverse impact on glycaemic control.
- Nutrition consultation would be useful. Medication regime including timing can be adjusted to suit the feeding times.

42. What are the measures to prevent hypoglycaemia?

- Pay attention to anorexia and weight loss.
- Adjust doses of insulin and other oral hypoglycaemic agents.
- Adjust treatment according to deterioration of renal and liver functions.
- Avoid complex regimes of medications.

References

1. Praveen PA, Kumar SR, Tandon N. Type 2 diabetes in youth in South Asia. *Curr Diab Rep*. 2015 Feb; 15 (2):571
2. ISPAD Clinical Practice Consensus Guidelines 2018: Type 2 diabetes mellitus in youth.
3. Screening, assessment and management of type 2 diabetes mellitus in children and adolescents: Australasian Paediatric Endocrine Group guidelines 2020.
4. Older Adults: Standards of Medical Care in Diabetes—2025, American Diabetes Association, *Diabetes Care* 2025 Jan; 44(Supplement 1): S168-S179. <https://doi.org/10.2337/dc21-S012>
5. Boursier, Jerome, Rodolphe Anty, Claire Carette, Bertrand Cariou, Laurent Castera, Cyrielle Caussy, Helene Fontaine et al. "Management of diabetes mellitus in patients with cirrhosis: An overview and joint statement." *Diabetes & Metabolism* 47, no. 5 (2021): 101272.
6. Roberts A, James J, Dhatariya K; Joint British Diabetes Societies (JBDS) for Inpatient Care. Management of hyperglycaemia and steroid (glucocorticoid) therapy: a guideline from the Joint British Diabetes Societies (JBDS) for Inpatient Care group. *Diabet Med*. 2018 Aug;35(8): 1011-1017. doi: 10.1111/dme.13675.
7. Aberer, F.; Hochfellner, D.A.; Sourij, H.; Mader, J.K. A Practical Guide for the Management of Steroid Induced Hyperglycaemia in the Hospital. *J. Clin. Med*. 2021, 10, 2154. <https://doi.org/10.3390/jcm10102154>
8. Adikari, S.B., Wickramage, S.P., Kalupahana, N.S., Pussepitiya, D.M.U.R.K. and Antonypillai, C.N., 2020. Dietary Adjustments: A Dilemma Faced by Both Buddhist Monastics with Diabetes and the Clinicians Managing Them. *Sri Lanka Journal of Medicine*, 29(2), pp 11-15 DOI: <http://doi.org/10.4038/sljm.v29i2.220>
9. Sanjay Kalra et al. Diabetes Management and the Buddhist Philosophy: Toward Holistic Care. *Indian J Endocrinol Metab*. Nov-Dec 2018; 22(6):806-811. doi: 10.4103/ijem.IJEM_285_17.
10. Diabetes and Ramadan practical guidelines 2021. International diabetes Federation in collaboration with diabetes and Ramadan (DAR) International alliance.
11. Recommendations for managing diabetes during Ramadan; update 2020, applying the principals of ADA/EASD consensus. *BMJ Open Diabetes research & care*. The South Asian health foundation (UK) guidelines for managing diabetes during Ramadan.
12. Diagnosing and Managing Diabetes in HIV-Infected Patients: Current Concepts. Anne K. Monroe, Marshall J. Glesby, and Todd T. Brown Division of General Internal Medicine, Johns Hopkins University School of Medicine, *HIV/AIDS • CID* 2015;60 (1 February)
13. Katherine Samaras, Endocrine disorders in people with HIV infection - HIV Management Guide for Clinical Care, Australasian Society for HIV, Viral Hepatitis and Sexual Health Medicine <https://hiv.guidelines.org.au/management/endocrine-disorders-in-hiv-infection-and-their-treatment/>
14. End of life care Standards of Medical Care in Diabetes—2025, American Diabetes Association, *Diabetes Care* 2025 Jan; 44 (Supplement 1): S168-S179. <https://doi.org/10.2337/dc21-S012>
15. End of life diabetes care. *Diabetes uk* 2018. https://www.diabetes.org.uk/resources-s3/2018-03/EoL_Guidance_2018_Final.pdf
16. Angelo, M., C. Ruchalski, and B.J. Sproge, An approach to diabetes mellitus in hospice and palliative medicine. *Journal of palliative medicine*, 2011. 14(1): p. 83-87.

17. DIABETES CARE MODEL FOR SRI LANKA

The 'chronic care model' is a framework design to improve the quality of care for individuals with chronic illnesses.

1. How should the 'Chronic Care Model' be adopted for diabetes care in Sri Lanka?

- Utilise team based care
- Set goals in management together with the patient
- Develop a care plan aligning with the established goals and clinical practice guidelines
- Use individualised Diabetes Self-Management Education and Support (DSMES)
- Regular reviews at a frequency suitable for the established plan
- Utilise community resources to optimise patient management
- Reassess and revise goals appropriately

2. Who are the healthcare providers that should be a part of the team?

- Endocrinologist/ physician and medical team
- Family practitioner/ medical officer of the relevant primary healthcare institute for shared care
- Medical nutrition team
- Diabetes Educator Nursing Officer (DENO) / Public Health Nursing Officer (PHNO)
- Psychologist
- Podiatrist/ Orthotist
- Physiotherapist
- Others depending on the patient's complications and comorbidities,
 - Ophthalmologist
 - Nephrologist
 - Cardiologist
 - General surgeon
 - Vascular surgeon
 - Orthopaedic surgeon
 - Psychiatrist
 - Gynaecologist

3. How could the diabetes care model be integrated into the national health system?

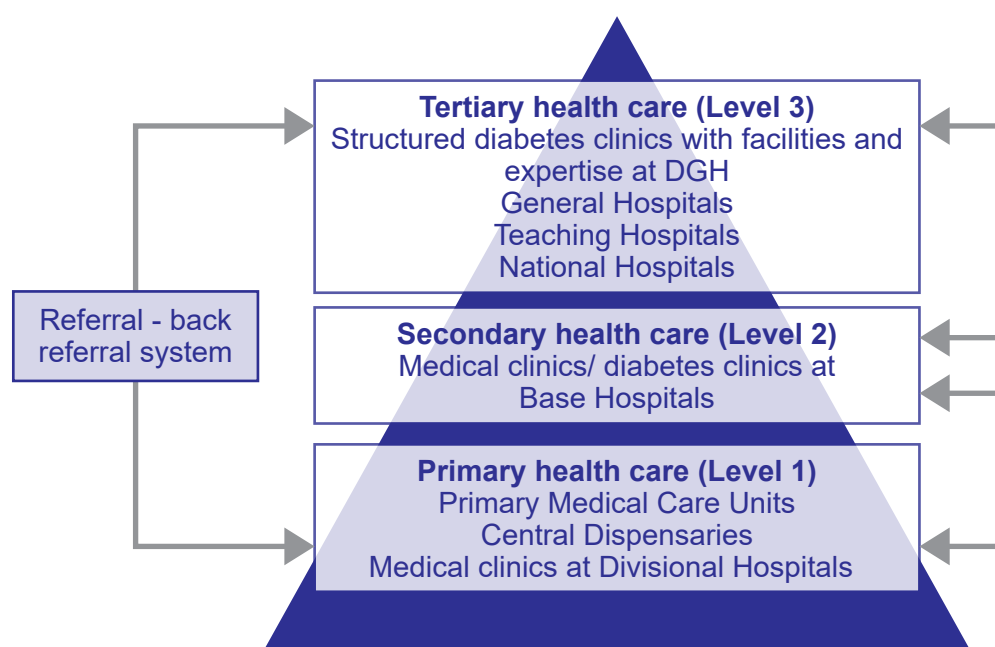


Figure 17.1 Suggested model for diabetes care in Sri Lanka

4. How could this model be utilised to improve patient care?

- Follow up the individual at one of the settings depending on the criteria mentioned below
- Use referral - back referral system as a dynamic method so the individual can move between the levels as and when a need arises
- Recognise level 1, 2, 3 facilities within a given healthcare cluster according to the geographical area to facilitate referral and back-referral system
- Refer individuals to higher levels when circumstances change (e.g., pregnancy, development of complications, suboptimal control etc.) or when facilities and/ or medicines are not available for satisfactory provision of care
- Refer back to the lower levels when circumstances change (e.g., following childbirth, following satisfactory glycaemic and metabolic control achieved) with a clear management plan including when to review again
- All individuals who are followed up at level 1 should be annually referred to a level 2 or 3 unit within the relevant healthcare cluster for end-organ screening, evaluation and optimisation.

5. What are the characteristics of patients likely to benefit from regular follow up at level 3 care?

- Type 1 diabetes
- Age < 20 years
- Hyperglycaemia in pregnancy
- On multiple daily doses of insulin
- Suboptimal glycaemic control despite usual care
- Recurrent hypoglycaemia/ any unexplained major hypoglycaemic episode/ hypoglycaemia unawareness
- Candidates for metabolic surgery
- Advanced complications related to diabetes requiring regular multidisciplinary approach
 - Diabetic kidney disease with urine albumin to creatinine ratio >300 mg/g or eGFR <30 mL/min/1.73m²
 - Disabling peripheral neuropathy/ autonomic neuropathy
 - Proliferative diabetic retinopathy/ maculopathy
 - Congestive cardiac failure/ ischaemic heart disease not controlled with first line therapy
 - Recent transient ischaemic attack/ stroke
 - High risk foot disease
 - Disabling occlusive arterial disease/ rest pain/ tissue loss of lower limbs.

6. How could the community health system in Sri Lanka assist in diabetes care?

- Establishment of 'diabetes support groups'.
 - Community health workers, government officers and volunteers can contribute to the functioning of these groups.
 - This would enable recognition of peer supporters and lay leaders who can assist people with diabetes.
- Provision of community diabetes care for needy people with poor social circumstances through the assistance of trained public health nursing officers.
 - assistance of trained public health nursing officers. Ensure timely follow up of individuals and minimising loss to follow up
 - assistance of trained public health nursing officers. Promotion of healthy lifestyle through individual level and group based programs
 - assistance of trained public health nursing officers. Assistance on insulin injections and blood glucose monitoring

7. How could social determinants of health be addressed through individualised diabetes care plans?

- Assess living conditions, financial state, access to food, home environment and health literacy of people
- Consider food insecurity and access to food when prescribing medical nutrition therapy
- Consider food insecurity, access to emergency services and social support when prescribing medicines with risk of hypoglycaemia including insulin
- Assess and address the effect of health literacy during diabetes education
- Address myths and beliefs related to diabetes care
- Acknowledge and be sensitive regarding cost-related medication non-adherence (medicines not available in government sector, regular self-monitoring of blood glucose etc.)
- Direct individuals with extreme social conditions to relevant local authorities for financial and other support
- Take additional steps to provide healthcare through local healthcare institutes for people with financial and social constraints

8. How can information technology be used in improving diabetes care?

- Use electronic health records to streamline hospital databases
- Establish electronic patient management systems to streamline patient consultations in order to minimise the waiting time
- Use remote management systems (telemedicine)
- Use artificial intelligence based systems to improve healthcare (e.g., trigger annual end-organ screening, trigger intensification of therapy)

References

1. American Diabetes Association. 1. Improving Care and Promoting Health in Populations: Standards of Medical Care in Diabetes—2025: Diabetes Care 2025; 44 (Suppl. 1): S7–S14.
2. Kayla L. Del Valle, Marie E Mc Donnell. Chronic Care Management Services for Complex Diabetes Management: a Practical Overview. Current Diabetes Reports. 2018. 18:135

Annexure 7.1**Diabetes new diagnosis/ new presentation check list**

	Specify/ mark with a tick	
Date of assessment		
Date of diagnosis		
Date of initiation of treatment		
Type of diabetes		
• T2D		
• T1D		
• Other		
Medications (with doses & frequency of administration)		
• Metformin		
• Sulphonylurea		
• DPP- 4i		
• SGLT2i		
• Insulin Type & doses		
• Other		
Eye screening	L	R
• No retinopathy		
• Mild non proliferative diabetic retinopathy (NPDR)		
• Moderate NPDR		
• Severe NPDR		
• Proliferative diabetic retinopathy (PDR)		
Early PDR		
High risk PDR		
Severe PDR		
• Clinically significant macular oedema (CSME)		
Foot assessment	Attached separately	
Other complications		
• Features of peripheral arterial disease		
• Ischaemic heart disease		
• Stroke/ transient ischaemic attacks		
• Erectile dysfunction		
Hypoglycaemia		
• Frequency and timing of day time hypoglycaemic episodes		
• Frequency and timing of night time hypoglycaemic episodes		
• Awareness in T1D/ insulin dependent T2D		
• Usual level of blood glucose at the onset of warning symptoms		
• Awareness about hypoglycaemia correction		

Insulin injection				
• Technique, storage				
• Sites – lipodystrophy/ lipohypertrophy				
Smoking	Y		N	
• Pack years				
Alcohol				
• Never				
• Social drinker				
• 2-3 times/ week				
• Daily >1/2 bottle of Arrack				
SMBG data (last 2 weeks)				
• Frequency of CBG monitoring				
• Fasting range				
• Post-prandial range				
Relevant co-morbidities				
• Hypertension				
• Metabolic liver disease				
• Dyslipidaemia				
• Hypothyroidism				
WHO 10 year CVD risk calculation				
In females if relevant;				
• Last menstrual period				
• Plans for fertility				
• Contraception				
Is immunisation up to date?				
Allergies				
Examination				
Height (m)				
Weight (kg)				
BMI (kg/m ²)				
Blood pressure				
Pulse rate				

Relevant investigations	Result	Date
HbA1c (%)		
FPG (mg/dl)		
PPPG (mg/dl)		
Lipid profile • Total cholesterol • LDL • HDL • TG • Non HDL		
Serum creatinine		
eGFR		
UFR		
Urine albumin: creatinine ratio		
Serum sodium		
Serum potassium		
TSH, fT4		
Coeliac screen		
Diabetes antibodies (if available/ relevant)		
C-peptide level with blood glucose (if available/ relevant)		
Education/counselling provided (mark with a tick)		
• Dietary management (including carbohydrate counting where relevant)		
• Physical activity		
• Insulin injection techniques		
• Hypoglycaemia detection & management		
• Sick day rules		

Annexure 7.2**Diabetes annual review check list**

	Specify/ mark with a tick			
Date of assessment				
Medications (with doses)				
• Metformin				
• Sulphonylurea				
• DPP- 4i				
• SGLT2i				
• Insulin Type & doses				
• Other				
Eye screening	L		R	
• No retinopathy				
• Mild non proliferative diabetic retinopathy (NPDR)				
• Moderate NPDR				
• Severe NPDR				
• Proliferative diabetic retinopathy (PDR)				
Early PDR				
High risk PDR				
Severe PDR				
• Clinically significant macular oedema (CSME)				
Frequency of eye screening (annually/ 6 monthly/ 3 monthly)				
Foot assessment	Attached separately			
Other complications				
• Features of peripheral arterial disease				
• Ischaemic heart disease				
• Stroke/ transient ischaemic attack				
Hypoglycaemia				
• Frequency and timing of day time hypoglycaemic episodes				
• Frequency and timing of night time hypoglycaemic episodes				
• Awareness in T1D/ insulin dependent T2D				
• Usual level of blood glucose at the onset of warning symptoms				
• Awareness about hypoglycaemia correction				
Insulin injection				
• Technique				
• Sites – lipodystrophy/ lipohypertrophy				
Smoking	Y		N	
• Pack years				

Alcohol		
• Never		
• Social drinker		
• 2-3 times/ week		
• Daily >1/2 bottle of Arrack		
SMBG data (last 2 weeks)		
• Frequency of CBG monitoring		
• Fasting range		
• Post prandial range		
WHO 10 year CVD risk calculation		
In females if relevant;		
• Last menstrual period		
• Plans for fertility		
• Contraception		
Is immunisation up to date?		
Examination		
Height (m)		
Weight (kg)		
BMI (kg/m ²)		
Blood pressure		
Pulse rate		
Relevant investigations	Result	Date
HbA1c (%)		
FPG (mg/dl)		
PPPG (mg/dl)		
Lipid profile		
• Total cholesterol		
• LDL		
• HDL		
• TG		
• Non HDL		
Serum creatinine		
eGFR		
UFR		
Urine albumin: creatinine ratio		
Serum sodium		
Serum potassium		
TSH, fT4		
Education/counselling provided (mark with a tick)		
• Dietary management (including carbohydrate counting where relevant)		
• Physical activity		
• Insulin injection techniques		
• Hypoglycaemia detection & management, sick day rules		

Annexure 7.3

NFS- NAFLD fibrosis score:

A non-invasive scoring system based on several lab tests that help to estimate the amount of scarring in patients with NAFLD.

Impaired fasting glucose/ diabetes:

Age :

AST :

ALT :

Platelet Count :

BMI :

Albumin :

Formula:

$-1.675 + 0.037 \times \text{age (years)} + 0.094 \times \text{BMI (kg/m}^2\text{)} + 1.13 \times \text{IFG/diabetes (yes = 1, no = 0)} + 0.99 \times \text{AST/ALT ratio} - 0.013 \times \text{platelet (}\times 10^9/\text{L)} - 0.66 \times \text{albumin (g/dl)}$

Explanation of result:

NAFLD Score < -1.455 = F0-F2 (advanced fibrosis could be excluded with high accuracy)

NAFLD Score $-1.455 - 0.675$ = indeterminate score

NAFLD Score > 0.675 = F3-F4 (the presence of advanced fibrosis could be diagnosed with high accuracy)

Reference:

Angulo P1, Hui JM, Marchesini G, Bugianesi E, George J, Farrell GC, Enders F, Saksena S, Burt AD, Bida JP, Lindor K, Sanderson SO, Lenzi M, Adams LA, Kench J, Therneau TM, Day CP. The NAFLD fibrosis score: a noninvasive system that identifies liver fibrosis in patients with NAFLD. *Hepatology*. 2007 Apr;45(4):846-54. PMID: 17393509.

Fib-4- Fibrosis 4 Score:

A non-invasive scoring system based on several lab tests that help to estimate the amount of scarring in the liver.

Age	
AST	
ALT	
Platelets	

Formula:

$(\text{Age} \times \text{AST}) / (\text{Plts} \times (\text{sqr (ALT)}))$

Explanation of result:

A FIB4 $> \text{or} = 2.67$ had an 80% positive predictive value and a FIB4 index $< \text{or} = 1.30$ had a 90% negative predictive value of advanced fibrosis

Reference

Shah, Amy G, Alison Lydecker, Karen Murray, Brent N Tetri, Melissa J Contos, and Arun J Sanyal. 2009. Comparison of noninvasive markers of fibrosis in patients with nonalcoholic fatty liver disease. *Clinical gastroenterology and hepatology*: the official clinical practice journal of the American Gastroenterological Association, no. 10 (June 10). doi:10.1016/j.cgh.2009.05.033. <http://www.ncbi.nlm.nih.gov/pubmed/19523535>.

ELF test- Enhanced liver fibrosis test:

A non-invasive blood test that assesses levels of three major components directly involved in liver matrix metabolism: hyaluronic acid (HA), procollagen III amino-terminal peptide (PIIINP), and tissue inhibitor of matrix metalloproteinase 1 (TIMP-1). A software calculates and report a unitless numerical score. The ELF score is well validated against biopsy proven liver fibrosis across a range of chronic liver diseases.

ELF Score	Severity assessment
<7.7	None to mild
≥ 7.7 - <9.8	Moderate
≥9.8	Severe
≥11.3	Cirrhosis

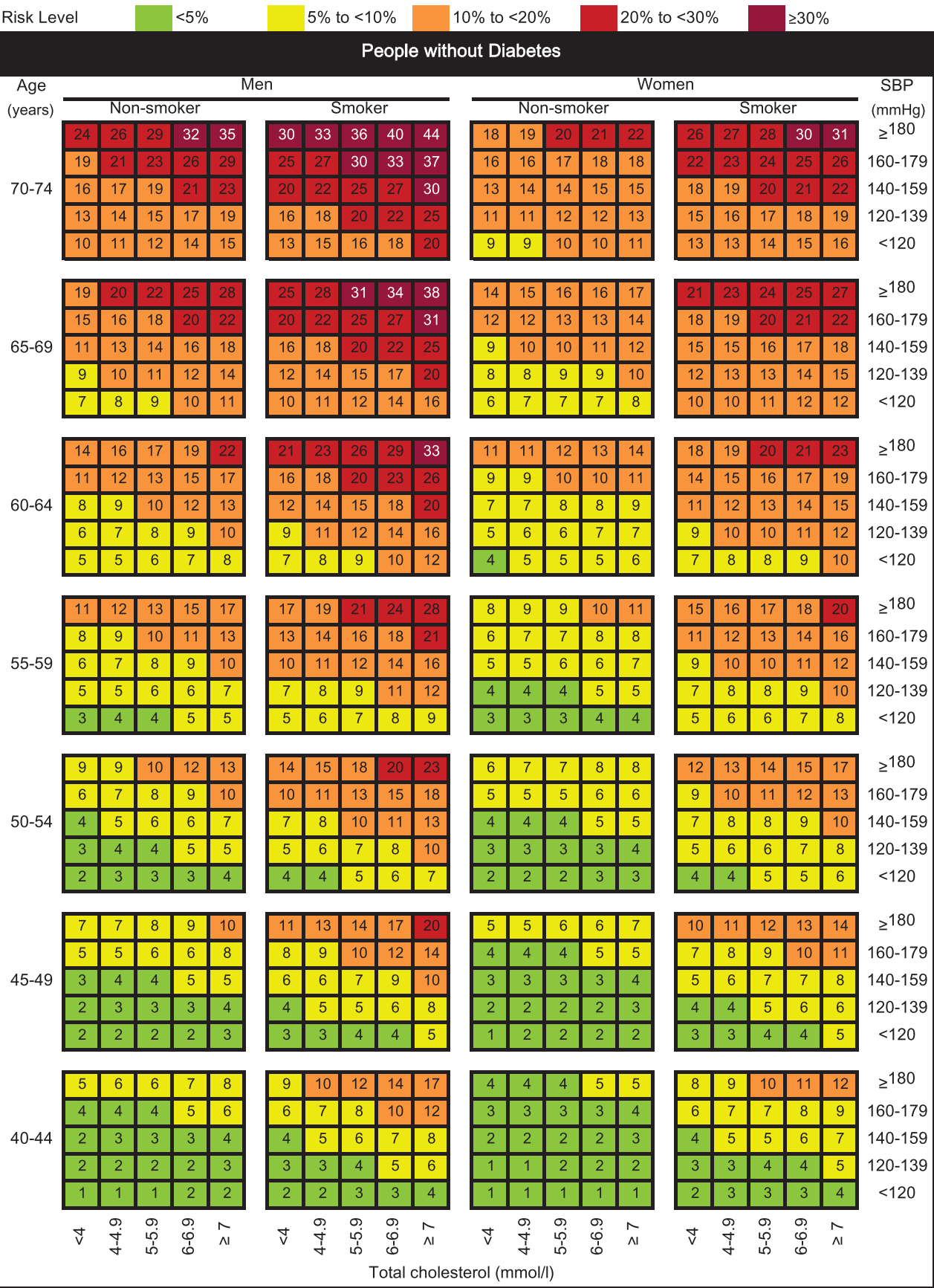
Reference

Guha IN, Parkes J, Roderick P, Chattopadhyay D, Cross R, Harris S, Kaye P, Burt AD, Ryder SD, Aithal GP, Day CP, Rosenberg WM. Noninvasive markers of fibrosis in nonalcoholic fatty liver disease: Validating the European Liver Fibrosis Panel and exploring simple markers. Hepatology. 2008 Feb;47(2):455-60. doi: 10.1002/hep.21984. PMID: 18038452.

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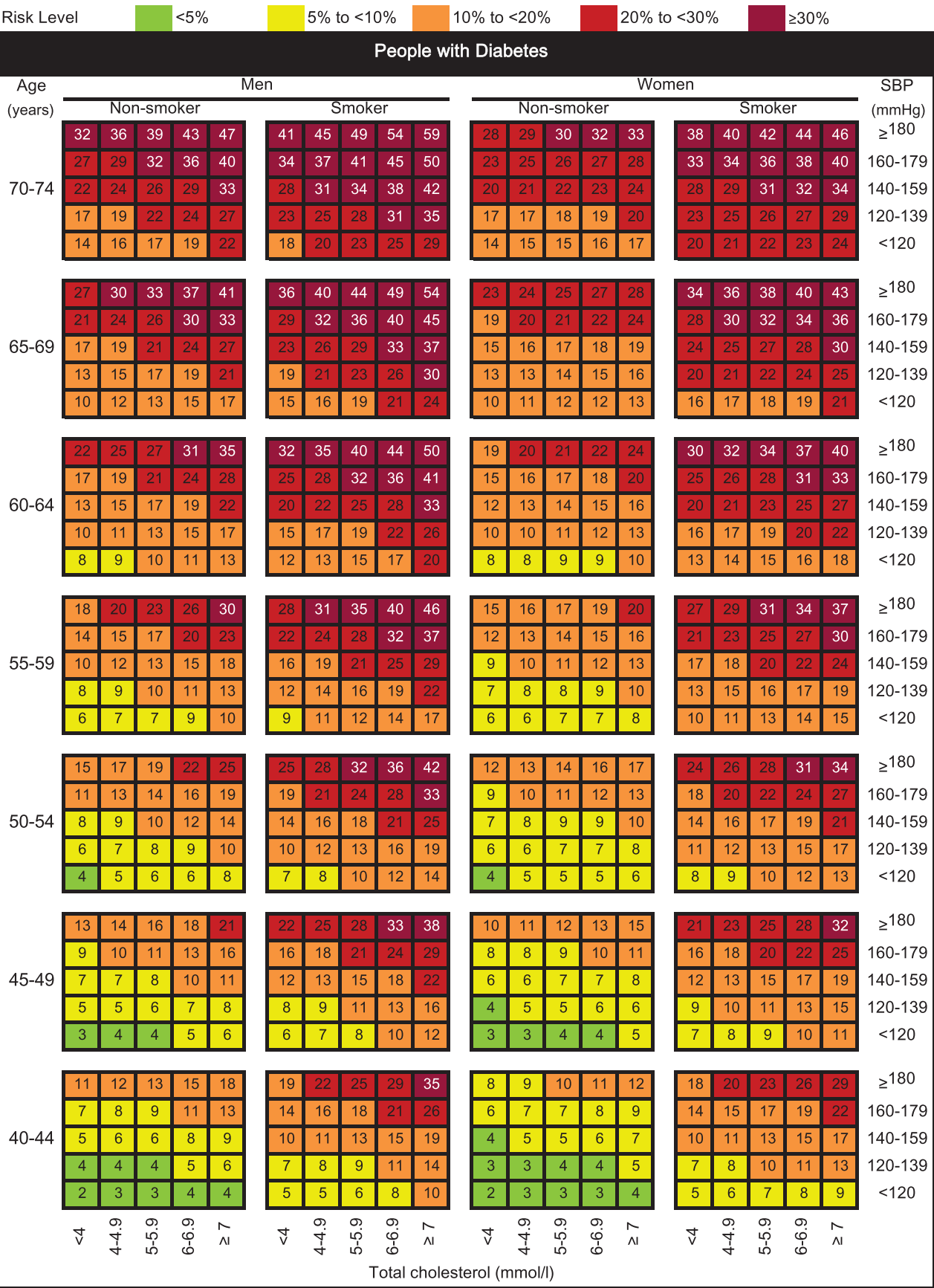
WHO cardiovascular disease risk laboratory-based charts
Southeast Asia

Indonesia, Cambodia, Lao PDR, Sri Lanka, Maldives, Myanmar, Malaysia, Philippines,
Thailand, Timor-Leste, Viet Nam, Mauritius, Seychelles.



WHO cardiovascular disease risk laboratory-based charts
Southeast Asia

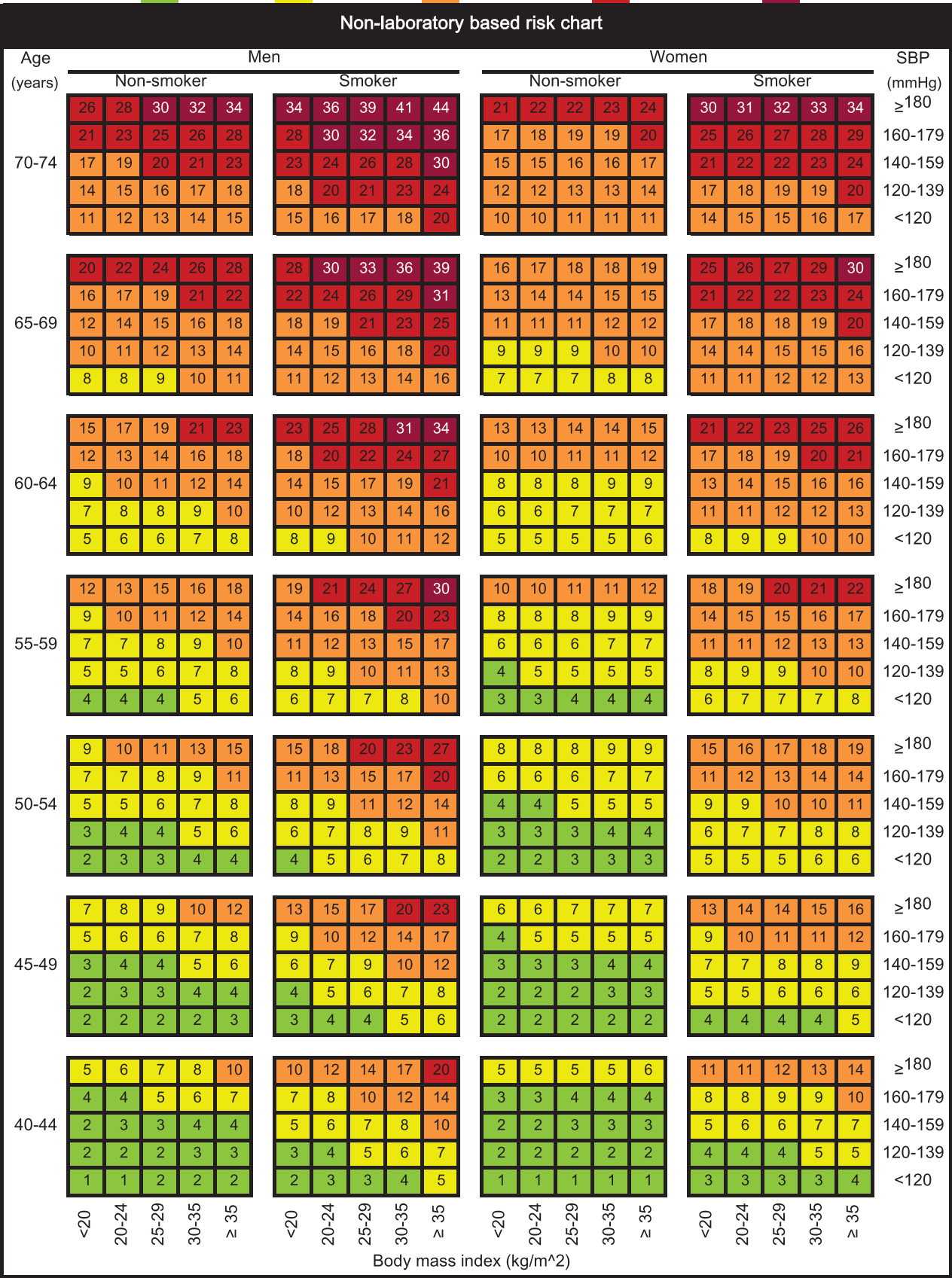
Indonesia, Cambodia, Lao PDR, Sri Lanka, Maldives, Myanmar, Malaysia, Philippines,
Thailand, Timor-Leste, Viet Nam, Mauritius, Seychelles.



WHO cardiovascular disease risk non-laboratory-based charts
Southeast Asia

Indonesia, Cambodia, Lao PDR, Sri Lanka, Maldives, Myanmar, Malaysia, Philippines,
Thailand, Timor-Leste, Viet Nam, Mauritius, Seychelles.

Risk Level <5% 5% to <10% 10% to <20% 20% to <30% ≥30%



Annexure 15.1
Variable Rate Intravenous Insulin infusion (VRIII) algorithm

Table 15A.1. Insulin infusion rates for patients on VRIII

Modified from: "Good In-patient Diabetes Service", Joint British Diabetes Societies for In Patient Care, July 2019. Available from <https://abcd.care/joint-british-diabetes-societies-jbds-in-patient-care-group> and the Diabetes UK website at <https://www.diabetes.org.uk/joint-british-diabetes-society>

Blood glucose (mg/dL)	Insulin infusion rate (units per hour)*		
	Reduced rate	Standard rate	Increased rate
70 – 140	0.5	1	2
141 – 220	1	2	4
221 – 300	2	4	6
301 – 380	3	5	7
381 – 450	4	6	8
> 450	6	8	10

*Most patients will require a starting insulin infusion at 'standard rate' plan. Patients with daily insulin requirements < 24 units/day or those who have repeated blood glucose values below 100 mg/dL or who shows too rapid blood glucose reduction should be switched to 'reduced rate' plan. Patients requiring insulin more than 100 units/day or having repeated measurements above 220 mg/dL will need 'increased rate' plan.

