HEARTS - D

Diagnosis and Management of Type 2 Diabetes
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Acknowledgements

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Abbreviations

ACE  angiotensin-converting enzyme
ACR  albumin-to-creatinine ratio
CVD  cardiovascular disease
eGFR estimated glomerular filtration rate
FPG  fasting plasma glucose
GAD  glutamic acid decarboxylase
GFR  glomerular filtration rate
HbA1c glycated haemoglobin
HHS  hyperosmolar hyperglycaemic state
PAD  peripheral artery disease
LOPS loss of protective sensation
NPH  neutral protamine Hagedorn
PCR  protein-to-creatinine ratio
RPG  random plasma glucose
T2DM type 2 diabetes mellitus
Diagnosis and management of type 2 diabetes

Part of the HEARTS technical package for cardiovascular disease management in primary health care

Diagnosis and management of type 2 diabetes is based on WHO guidance on diagnosis, classification and management of diabetes. It is aligned with the WHO Package of Essential Noncommunicable Disease Interventions in Primary Health Care (WHO-PEN). It can be used independently or in conjunction with the HEARTS technical package developed to improve cardiovascular health.

Target users may vary, based on context, existing health systems and national priorities.

The people who will find the modules most useful are:

- **National level** – Ministry of Health NCD policymakers responsible for:
  - developing strategies, policies and plans related to service delivery of diabetes
  - setting national targets on diabetes, monitoring progress and reporting

- **Subnational level** – Health/NCD programme managers responsible for:
  - planning, training, implementing and monitoring service delivery

- **Primary care level** – Facility managers and primary health care staff and trainers responsible for:
  - assigning tasks, organising training and ensuring the facility is running smoothly
  - clinical management of people with type 2 diabetes
  - collecting facility-level data on indicators of progress towards diabetes targets.
Introduction

Definition of diabetes

Diabetes mellitus, commonly known as diabetes, is a group of metabolic disorders characterized by the presence of hyperglycaemia in the absence of treatment. The heterogeneous aetiopathology includes defects in insulin secretion, insulin action, or both. The long-term specific complications of diabetes include retinopathy, nephropathy, and neuropathy. People with diabetes are also at increased risk of other diseases, including cardiac, peripheral arterial and cerebrovascular disease, cataracts, erectile dysfunction, and nonalcoholic fatty liver disease. They are also at an increased risk of some infectious diseases such as tuberculosis, and are likely to experience poorer outcomes.

Epidemiology and global burden of diabetes

Diabetes is found in every population in the world and in all regions, including rural parts of low- and middle-income countries. WHO estimates there were 422 million adults with diabetes worldwide in 2014. The age-adjusted prevalence in adults rose from 4.7% in 1980 to 8.5% in 2014, with the greatest increase in low- and middle-income countries.

Aetio-pathology of diabetes

The underlying characteristic common to all forms of diabetes is the dysfunction or destruction of pancreatic beta-cells. These cells are not replaced, as the human pancreas seems incapable of renewing beta-cells after the age of 30 years. Many mechanisms can lead to a decline in function or the complete destruction of beta-cells. These mechanisms include genetic predisposition and abnormalities, epigenetic processes, insulin resistance, auto-immunity, concurrent illnesses, inflammation, and environmental factors.

Most common types of diabetes and their risk factors

<table>
<thead>
<tr>
<th>Risk factors for type 2 diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>• overweight/obesity</td>
</tr>
<tr>
<td>• physical inactivity</td>
</tr>
<tr>
<td>• age</td>
</tr>
<tr>
<td>• diabetes in first degree relatives</td>
</tr>
<tr>
<td>• history of gestational diabetes</td>
</tr>
<tr>
<td>• cardiovascular disease and its risk factors</td>
</tr>
<tr>
<td>• ethnicity (South Asian, Afro-Caribbean, Hispanic)</td>
</tr>
</tbody>
</table>

The most common type of diabetes mellitus is type 2 diabetes (T2DM). The majority of people with T2DM are overweight or obese, which either causes or aggravates
insulin resistance. Many of those who are not obese by BMI criteria have a higher proportion of body fat distributed predominantly in the abdominal region, indicating visceral adiposity, compared to people without diabetes.

However, in some populations, such as Asians, beta-cell dysfunction appears to be a more prominent feature than in populations of European descent. This is also observed in thinner people from low- and middle-income countries such as India, and among people of Indian descent living in high-income countries.

### Risk factors for type 1 diabetes

- certain genetic haplotypes
- unknown environmental factors

Type 1 diabetes is much less common, the risk being highest in populations of European origin. This module is specifically for management of type 2 diabetes, as type 1 is usually not managed in primary care facilities.

**Classification of diabetes**

The WHO classification of diabetes is presented in Table 1. It prioritizes clinical care and guides health professionals in choosing appropriate treatments at the time of diabetes diagnosis, providing practical guidance to clinicians in assigning a type of diabetes to individuals at the time of diagnosis.
<table>
<thead>
<tr>
<th>Type of diabetes</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1 diabetes</td>
<td>Beta-cell destruction (mostly immune-mediated) and absolute insulin deficiency; onset most common in childhood and early adulthood.</td>
</tr>
<tr>
<td>Type 2 diabetes</td>
<td>Most common type, various degrees of beta-cell dysfunction and insulin resistance; commonly associated with overweight and obesity.</td>
</tr>
<tr>
<td>Hybrid forms of diabetes</td>
<td></td>
</tr>
<tr>
<td>Slowly evolving, immune-mediated diabetes of</td>
<td>Similar to slowly evolving type 1 in adults but more often has features of the metabolic syndrome, a single GAD autoantibody, and retains greater beta-cell function.</td>
</tr>
<tr>
<td>adults</td>
<td></td>
</tr>
<tr>
<td>Ketosis-prone type 2 diabetes</td>
<td>Presents with ketosis and insulin deficiency but later does not require insulin; common episodes of ketosis, not immune-mediated.</td>
</tr>
<tr>
<td>Other specific types</td>
<td></td>
</tr>
<tr>
<td>Monogenic diabetes:</td>
<td>Caused by specific gene mutations. Has several clinical manifestations requiring different treatment, some occurring in the neonatal period, others by early adulthood.</td>
</tr>
<tr>
<td>a) Monogenic defects of beta-cell function</td>
<td></td>
</tr>
<tr>
<td>b) Monogenic defects in insulin action</td>
<td>Caused by specific gene mutations. Has features of severe insulin resistance without obesity; diabetes develops when beta-cells do not compensate for insulin resistance.</td>
</tr>
<tr>
<td>Diseases of the exocrine pancreas</td>
<td>Various conditions that affect the pancreas can result in hyperglycaemia (trauma, tumour, inflammation, etc.).</td>
</tr>
<tr>
<td>Endocrine disorders</td>
<td>Occurs in diseases with excess secretion of hormones that are insulin antagonists.</td>
</tr>
<tr>
<td>Drug- or chemical-induced</td>
<td>Some medicines and chemicals impair insulin secretion or action, some can destroy beta-cells.</td>
</tr>
<tr>
<td>Infection-related diabetes</td>
<td>Some viruses have been associated with direct beta-cell destruction.</td>
</tr>
<tr>
<td>Uncommon specific forms of immune-mediated</td>
<td>Associated with rare immune-mediated diseases.</td>
</tr>
<tr>
<td>diabetes</td>
<td></td>
</tr>
<tr>
<td>Other genetic syndromes</td>
<td>Many genetic disorders and chromosomal abnormalities increase the risk of diabetes</td>
</tr>
<tr>
<td>sometimes associated with diabetes</td>
<td></td>
</tr>
<tr>
<td>Unclassified diabetes</td>
<td>Used to describe diabetes that does not clearly fit into other categories. This category should be used temporarily, when there is no clear diagnostic category, close to the time of diagnosis, in particular.</td>
</tr>
<tr>
<td>Hyperglycaemia first detected during pregnancy</td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus in pregnancy</td>
<td>Type 1 or type 2 diabetes first diagnosed during pregnancy.</td>
</tr>
<tr>
<td>Gestational diabetes mellitus</td>
<td>Hyperglycaemia below diagnostic thresholds for diabetes in pregnancy.</td>
</tr>
</tbody>
</table>
Clinical manifestations of diabetes and diagnostic criteria

Clinical manifestations
Diabetes may present with characteristic symptoms and signs (Table 2). It is estimated that a significant percentage of cases of type 2 diabetes (T2DM) (30% to 80%, depending on the country) are undiagnosed. The most severe clinical manifestations are ketoacidosis or a non-ketotic hyperosmolar state that may lead to dehydration, coma and, in the absence of effective treatment, death. However, symptoms in T2DM are often not severe, or may be absent, owing to the slow pace at which the hyperglycaemia is worsening. As a result, in the absence of biochemical testing, hyperglycaemia sufficient to cause pathological and functional changes may be present for a long time before a diagnosis is made, resulting in the presence of complications at diagnosis.

Type 1 diabetes is more likely to present with symptoms and its onset is typically in children and young adults. However, the type of diabetes cannot always be determined at diagnosis, and initial treatment decisions should be based on clinical presentation and plasma glucose values.

Table 2 Symptoms and signs of diabetes

<table>
<thead>
<tr>
<th>Symptoms of diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>• thirst</td>
</tr>
<tr>
<td>• frequent urination</td>
</tr>
<tr>
<td>• blurring of vision</td>
</tr>
<tr>
<td>• fatigue</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Signs of diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>• unintentional weight loss</td>
</tr>
<tr>
<td>• signs of acute metabolic deterioration</td>
</tr>
<tr>
<td>• clinical signs of chronic complications</td>
</tr>
</tbody>
</table>

(acute coronary disease, stroke, kidney disease, vision loss, diabetic foot)
Diagnostic criteria for diabetes

Diagnosis of diabetes is based on values of plasma glucose or glycated haemoglobin (HbA1c). Diagnostic cut-off values are presented in Table 3.

### Table 3 Diagnostic criteria for diabetes

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Diagnostic cut-off value</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting venous or capillary** plasma glucose</td>
<td>≥7.0 mmol/L (126 mg/dL)</td>
<td>Least costly but difficulties with ensuring a fasting state</td>
</tr>
<tr>
<td>2-hour post-load venous plasma glucose</td>
<td>≥11.1 mmol/L (200 mg/dL)</td>
<td>Cumbersome and costly, difficulties with ensuring a fasting state</td>
</tr>
<tr>
<td>2-hour post-load capillary** plasma glucose</td>
<td>≥12.2 mmol/L (220 mg/dL)</td>
<td>Cumbersome and costly, difficulties with ensuring a fasting state</td>
</tr>
<tr>
<td>Random plasma glucose</td>
<td>≥11.1 mmol/L (200 mg/dL)</td>
<td>To be used only in the presence of symptoms</td>
</tr>
<tr>
<td>HbA1c***</td>
<td>6.5% (48 mmol/mol)</td>
<td>• Less intra-individual variability than plasma glucose</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Does not require the fasting state but substantially more costly than glucose measurements</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Is an indirect method</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Can be inaccurate in some conditions (haemoglobinopathies, renal failure, some anaemias, conditions with rapid red blood cell turnover)</td>
</tr>
</tbody>
</table>

* Overnight fast of 8–14 hours.
** If laboratory measurement is not available, point of care, (“finger stick”) devices can be used (they report glucose values in capillary plasma).
*** Plasma glucose is preferred in people with symptoms who are suspected of having type 1 diabetes.

### Diagnostic tests

Venous plasma glucose is the standard method for measuring and reporting. However, in recognition of the widespread use of capillary sampling, especially in low-resource settings, values for capillary plasma glucose are provided for post-load glucose values. *Fasting values for venous and capillary plasma glucose are identical.*

Glucose should be measured immediately after collection, otherwise the blood sample should be collected into a container with glycolytic inhibitors, immediately centrifuged to separate the plasma, and frozen until analysis.

- In asymptomatic people, repeat the test to confirm the diagnosis, preferably with the same test, as soon as practicable on a subsequent day.
- If plasma glucose ≥18 mmol/L (325 mg/dL), or symptoms are present, measure urine ketones to assess degree of metabolic disturbance.
- If plasma glucose measurement is not possible, urine glucose testing can be used to confirm suspicion of diabetes in people with symptoms. A negative urine test does not exclude diabetes, but it excludes severe hyperglycaemia.
Management of diabetes

Type 2 diabetes is a progressive illness, with insulin secretion decreasing over time. Introduction of oral hypoglycaemic agents (OHA) will often be necessary in patients treated with diet and physical activity only, and further intensification with insulin might be needed as the illness progresses and OHAs are not sufficient to control glycaemia.

Blood glucose management protocol

The blood glucose management protocol is recommended for patients with established or newly diagnosed type 2 diabetes (see Fig. 1). A simplified blood glucose management protocol and key actions on complications are presented in Fig.2.

Non-pharmacological management

A healthy diet to achieve or maintain normal body weight and regular physical activity are the mainstay of diabetes management.

- People with diabetes should be advised to eat a healthy balanced diet that is applicable to the general population.
- Overweight patients should be advised to reduce weight by reducing their food (calorie) intake.
- All patients should be advised to practise regular daily physical activity appropriate for their physical capabilities (e.g. walking). Most adults should engage in at least 150 minutes of moderate or vigorous-intensity aerobic activity per week, spread over at least 3 days.
- All patients should be advised on avoidance of tobacco use and harmful use of alcohol.

Pharmacological management

Control of blood glucose levels (glycaemia)

Initial treatment:

- Metformin does not cause weight gain or hypoglycaemia and is the recommended initial treatment for people who do not achieve the desired glycaemic control with diet and physical activity. Increase the dosage gradually according to the diabetes protocol.
- A second-generation sulfonylurea (preferably gliclazide) can be used as initial (first-line) treatment when metformin is contraindicated or not tolerated. Sulfonylureas may cause weight gain or hypoglycaemia.
- Other pharmacological agents have not been shown to be superior to metformin or sulfonylurea for glycaemic control and long-term outcomes as initial treatment.
Metformin

Metformin is contraindicated in:

- people with chronic kidney disease (estimated glomerular filtration rate (eGFR) <30 mL/minute/1.73m²)
- people with severe reduced liver function
- people with acute cardiac insufficiency
- people with respiratory insufficiency
- people who abuse alcohol
- people with history of lactic acidosis

Intensification of treatment when metformin alone fails to control glycaemia:

- Add a second-generation sulfonylurea (preferably gliclazide) to metformin in patients with inadequately controlled glycaemia on metformin, along with diet and physical activity.
- In hyperglycaemic patients with symptoms, give a sulfonylurea or refer for insulin treatment.
- Hypoglycaemia is a possible side-effect of sulfonylurea, more frequent with glibenclamide than with gliclazide.

Glibenclamide

Glibenclamide is not recommended in:

- people aged 60 years or older
- people with severe liver disease
- in patients for whom hypoglycaemia is a concern (people who are at risk of falls, who have impaired awareness of hypoglycaemia, who live alone)
- people who drive or operate machinery as part of their job.

Intensification of treatment when metformin and sulfonylurea fail to control glycaemia:

- Refer for insulin treatment or add human insulin¹ to oral medication (Annex 1).
- If insulin is unsuitable², a DPP-4 inhibitor, SGLT-2 inhibitor or a thiazolidinedione (TZD) may be added, but these are not recommended for routine use due to high cost and, with the exception of SGLT-2 inhibitors, uncertain benefit.

¹ Insulin analogues are not recommended for routine use as they are substantially more costly than human insulin and there is considerable uncertainty over their benefits, especially in people with type 2 diabetes.

² Insulin treatment could be unsuitable when it is more costly than oral agents, or when circumstances make its use difficult (e.g. persons who live alone and are dependent on others to inject them with insulin).
Fig. 1 Protocol for control of blood glucose in type 2 diabetes*

TEST ADULTS who have symptoms of diabetes with fasting or random plasma glucose (FPG or RPG)
TEST ASYMPTOMATIC ADULTS who are 40+ years old and BMI ≥25 (FPG)

FPG ≥7 mmol/L and <18 mmol/L or RPG ≥11.1 mmol/L
IF ASYMPTOMATIC repeat test on subsequent day

FPG ≥15 mmol/L and SYMPTOMATIC or FPG/RPG >18 mmol/L, regardless of symptoms

Counsel on diet and physical activity and adherence to medicines at all visits

REVIEW IN 1 MONTH (or immediately if symptoms appear)

IF goal not achieved BEGIN METFORMIN 500 mg daily

IF goal not achieved INCREASE METFORMIN to 1000 mg 1 x daily

IF goal not achieved INCREASE METFORMIN to 1000 mg 2 x daily

IF goal not achieved ADD glipizide 80 mg 1 x daily

IF goal not achieved ADD glipizide 80 mg 2 x daily

IF goal not achieved despite adherence to medication and diet and physical activity, REFER to higher level of care or BEGIN INSULIN (see insulin protocol)

Urine ketones ≥2+

GIVE METFORMIN 1000 mg 2 x daily and GLICLAZIDE 80 mg 2 x daily
Counsel on diet, physical activity and adherence to medication

No improvement

Improvement

CONTINUE treatment

IF goal achieved or frequent hypoglycaemia, consider reducing or stopping glipizide in people who were on 2000 mg metformin and 160 mg glipizide from diagnosis

REVIEW IN 1 MONTH

IF goal NOT achieved

REFER to higher level of care

* Derived from WHO-PEN 2013
Control of blood pressure and blood lipids

Blood pressure should be measured at every visit. Hypertension treatment is indicated in people with diabetes when systolic blood pressure ≥130 mmHg or diastolic blood pressure ≥80 mmHg. Statins are recommended for all people with type 2 diabetes 40 years old or older, but only if this does not negatively impact access to glucose-lowering and blood pressure-lowering medication.

Referral criteria

Criteria for referral to higher levels of care are presented in Section 4.

Monitoring of glycaemic control

Glycated haemoglobin (HbA1c) is commonly used in clinical practice to monitor glycaemic control, as it provides a measure of average plasma glucose over the preceding 8 to 12 weeks.

If HbA1c is not available:

- Fasting plasma glucose (FPG) values can be used to assess glycaemic control and inform treatment.
- More informative is a combination of FPG and postprandial plasma glucose (2 hours after breakfast).
- Most informative is a glucose profile with several pre- and postprandial measurements throughout the day.
- The least informative is random plasma glucose (RPG).

Targets for glycaemic control

Blood glucose values closer to normal substantially reduce the risk of microvascular complications. The downside of tight blood glucose control is a potentially dangerous side-effect of the treatment (hypoglycaemia). It is recognized that an individualized approach is preferred when setting treatment targets. Patients with a short life expectancy and those with advanced complications or serious comorbidities are unlikely to benefit from efforts to achieve near-normal glycaemia.

- The majority of patients can be expected to aim for an HbA1c of 7.0% (53 mmol/mol).
- The HbA1c target can be relaxed (e.g. to <8% or <64 mmol/mol) in people with frequent severe hypoglycaemia, advanced complications or low life-expectancy.
- Patients treated with diet, physical activity and metformin (very low risk of hypoglycaemia) should be encouraged to achieve a lower HbA1c target.
- If HbA1c measurement is not available or there is concern over its validity, an FPG value of ≤7.0 mmol/L (126mg/dL) and a postprandial PG value of ≤9.0 mmol/L (160 mg/dL) can serve as surrogates.
3 Prevention and management of complications of diabetes

Acute complications of diabetes

Two important acute complications are hypoglycaemia and hyperglycaemic emergencies.

Hypoglycaemia

Hypoglycaemia (abnormally low blood glucose) is a frequent iatrogenic complication in diabetic patients, occurring particularly in patients receiving sulfonylurea or insulin. It can cause loss of consciousness and coma and is potentially life-threatening. Severe hypoglycaemia is defined as hypoglycaemia, during which the patient is unable to self-treat by ingestion of carbohydrates. There is no universally agreed plasma glucose cut-off point for hypoglycaemia, as symptoms and signs can occur at different thresholds. It is most frequently defined at plasma glucose of ≤3.9 mmol/L (70 mg/dL), when it should be managed even if there are no symptoms and signs.

Table 4 Symptoms and signs of hypoglycaemia

<table>
<thead>
<tr>
<th>Symptoms of hypoglycaemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>• headache</td>
</tr>
<tr>
<td>• hunger</td>
</tr>
<tr>
<td>• irritability, anxiety</td>
</tr>
<tr>
<td>• paraesthesias</td>
</tr>
<tr>
<td>• palpitations</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Signs of hypoglycaemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>• sweating</td>
</tr>
<tr>
<td>• trembling</td>
</tr>
<tr>
<td>• difficulty in speaking</td>
</tr>
<tr>
<td>• confusion</td>
</tr>
<tr>
<td>• ataxia</td>
</tr>
<tr>
<td>• stupor</td>
</tr>
<tr>
<td>• pallor</td>
</tr>
<tr>
<td>• seizures</td>
</tr>
<tr>
<td>• coma</td>
</tr>
</tbody>
</table>
Management of hypoglycaemia

Hypoglycaemia is managed by ingestion of carbohydrate if the patient is able to swallow, or by i.v. administration of hypertonic glucose.\(^1\)

- **If the patient is able to ingest food or drink**, they should ingest 15–20 g of glucose. If glucose is not available, give oral carbohydrate that contains 15–20 g of rapidly absorbing forms of glucose (e.g. sugar-sweetened soft drink, 1–2 teaspoons of sugar, 5–6 hard candies, cup of milk). Plasma glucose levels typically increase by 2.8 mmol/l (50 mg/dL) within ~15 minutes; repeat the treatment if hypoglycaemia persists.

- If rapidly absorbing glucose is not available, it can be substituted by any foods containing carbohydrate (e.g. bread, rice, potato).

- Follow by a small meal with complex carbohydrate.

- **Unconscious patients, those with plasma glucose ≤2.8 mmol/L (50 mg/dL) and those unable to to ingest drink** should be given hypertonic glucose (dextrose) intravenously (20–50 mL of 50% glucose over 1–3 minutes). If this concentration is not available, substitute with any hypertonic glucose solution.

- Food should be provided as soon as the patient is able to ingest food safely.

- Discuss hypoglycaemia risk factors with the patient (skipping meals, physical activity more intense than usual, alcohol ingestion) and adjust medication if necessary.

**Hyperglycaemic emergencies**

Diabetic ketoacidosis (DKA) and hyperosmolar hyperglycaemic state (HHS) are life-threatening conditions with somewhat different biochemical features (Table 5). Although uncommon, DKA can occur in people with type 2 diabetes.

Symptoms and signs of DKA and HHS:

- Frequent symptoms and signs of DKA are nausea, vomiting and abdominal pain.

- Severe cases of DKA can present with Kussmaul’s breathing.

- Changes in sensorium in DKA range from alertness to stupor or coma, depending on the severity.

- Patients with HHS typically present with altered consciousness (stupor or coma).

**Table 5 Biochemical characteristics of DKA and HHS measurable in primary care facilities**

<table>
<thead>
<tr>
<th></th>
<th>DKA</th>
<th>HHS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Plasma glucose level</strong></td>
<td>≥13.9 mmol/L (250 mg/dL), occasionally lower</td>
<td>≥33.3 mmol/L (600 mg/dL)</td>
</tr>
<tr>
<td><strong>Urine ketones</strong></td>
<td>Positive</td>
<td>Negative (or weakly positive)</td>
</tr>
</tbody>
</table>

\(^1\) Intramuscular injection of glucagon can also resolve hypoglycaemia, but it is rarely available in primary care.
Management of hyperglycaemic emergencies

Hyperglycaemic emergencies should be managed in hospital by correction of dehydration and electrolyte disbalance and administration of insulin.

- Refer to hospital all patients with suspected DKA or HHS. (DKA or HHS should be suspected in every ill patient with hyperglycaemia.)
- Correction of dehydration is the critical first step for transport. Hyperglycaemia slows gastric emptying, and oral rehydration might not be effective even in patients who are not vomiting.
- Infuse isotonic saline (0.9% NaCl) at a rate of 1000 mL in the first 2 hours. Continue with 1000 mL every 4 hours until reaching a hospital.

Screening and management of chronic complications of diabetes

Microvascular complications

Longstanding diabetes with uncontrolled blood glucose levels can lead to multiple organ damage, leading to diabetic retinopathy, nephropathy, neuropathy, and diabetic foot complications. These complications are typically clinically “silent” until far advanced.

Diabetic eye disease

- Diabetic retinopathy is a highly specific microvascular complication of diabetes. It is among the leading causes of blindness.
- Diabetes is also associated with an increased risk of other vision-threatening conditions, such as cataract and glaucoma.

Risk factors for diabetic retinopathy

- duration of diabetes
- poor glycaemic control
- hypertension
- diabetic kidney disease
- dyslipidaemia

Diagnosis of diabetic retinopathy

Signs and symptoms of diabetic retinopathy:

- Vision-threatening retinopathy and macular changes may be asymptomatic.
- Vision loss occurs at advanced stages.

Diabetic retinopathy can be diagnosed through the presence of specific retinal abnormalities on examination of the fundus after pupil dilation (microaneurysms, haemorrhages, venous beading, hard exudates, cotton-wool spots and neovascularization) and/or macular oedema (retinal thickening).

Retinal examination can be performed through ophthalmoscopy (direct or indirect), slit lamp biomicroscopy, or retinal fundus photography.

Diagnosis of diabetic macular oedema requires a three-dimensional assessment that is best performed by a dilated examination using slit-lamp biomicroscopy and/or stereo fundus photography.
Screening for and management of diabetic retinopathy

People with type 2 diabetes should be screened for retinopathy by a trained person upon diagnosis, and biennially thereafter, or as recommended by an ophthalmologist, for:

- visual acuity
- direct or indirect ophthalmoscopy (dilated pupils) or retinal fundus photography.

Patients reporting vision loss at any visit and those who have not had a retinal examination in the past two years should be referred to an ophthalmologist.

Good control of glycaemia as well as blood pressure and dyslipidaemia can slow the progression of diabetic retinopathy and macular oedema. Timely treatment of retinopathy with laser photocoagulation can reduce the risk of vision loss. In advanced proliferative retinopathy, some recovery of visual acuity can be achieved by vitrectomy.

Intravitreal injections of anti-vascular endothelial growth factor (anti-VEGF) can prevent some visual loss in diabetic macular oedema.

Diabetic kidney disease

Diabetic kidney disease is a microvascular complication of diabetes, with a characteristic histopathology. If untreated, it is characterized by a relentless decline in glomerular filtration rate (GFR), raised arterial blood pressure, and high risk of CVD and death. If left untreated, once the stage of proteinuria is reached it often ends in renal failure in about 5 to 7 years.

Diabetic kidney disease is defined by albuminuria and/or a decreased estimated glomerular filtration rate (eGFR).

**Diagnosis of diabetic kidney disease**

Symptoms and signs of diabetic kidney disease:

- The earliest clinical signs are elevated blood pressure and moderately increased urine albumin excretion (Table 6).
- Peripheral oedema occurs at a very late stage.
- The first symptoms are those of uraemia (nausea, itching, anorexia).

The diagnosis of diabetic kidney disease is made in patients diagnosed with diabetes upon:

- Estimated glomerular filtration rate (eGFR)\(^1\) of <60 mL/min per 1.73 m\(^2\) on at least two occasions, 1 to 3 months apart, and/or
- Presence of albuminuria in at least two urine samples, 1 to 3 months apart.

---

\(^1\) eGFR is calculated from serum creatinine, using an equation that has been validated for that population. Otherwise, the most commonly used equation, developed by the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI). It is based on serum creatinine, age, sex and race.
Urinary albumin excretion can be estimated in a spot urine sample using several tests (from most preferred to least preferred):

- urine albumin-to-creatinine ratio (ACR)
- urine protein-to-creatinine ratio (PCR)
- reagent strip ("dipstick") urine analysis for albumin or total protein with automated reading
- reagent strip ("dipstick") urine analysis for albumin or total protein with manual reading.

Table 6 Categories of albuminuria (measured by ACR)

<table>
<thead>
<tr>
<th>Category</th>
<th>mg/g</th>
<th>mg/mmol</th>
<th>dipstick</th>
</tr>
</thead>
<tbody>
<tr>
<td>normal to mildly increased</td>
<td>&lt;30</td>
<td>&lt;3</td>
<td>–</td>
</tr>
<tr>
<td>moderately increased</td>
<td>30–300</td>
<td>3–30</td>
<td>Trace/1+</td>
</tr>
<tr>
<td>severely increased</td>
<td>&gt;300</td>
<td>&gt;30</td>
<td>1+/2+</td>
</tr>
</tbody>
</table>

**Screening for and management of diabetic kidney disease**

People with type 2 diabetes should be screened once a year with either the albumin/creatinine ratio in a spot urine sample or with eGFR using serum creatinine (preferably both tests, for better prognostic ability).

Patients with moderately and severely increased albuminuria and those with GFR <60 ml/min/1.73 m² if combined with albuminuria should be referred for specialist assessment (see Section 4).

To delay onset and slow the progression of diabetic kidney disease:

- Aim for good glycaemic control but adjust for hypoglycaemia risk.
- Maintain blood pressure levels at <130/80 mmHg with an angiotensin-converting enzyme (ACE) inhibitor and add thiazide diuretic if required.
- Modify other major CVD risk factors (dyslipidaemia, smoking).

End-stage renal disease requires renal replacement therapy (haemodialysis, peritoneal dialysis, renal transplant).

**Diabetic neuropathy**

Nerve damage in diabetes is a group of disorders with diverse clinical manifestations. The most common forms are distal symmetrical peripheral neuropathy, which is predominantly sensory, and autonomic neuropathy, which affects the autonomic nervous system. Loss of protective sensation in peripheral neuropathy is a predisposing condition in the pathway leading to foot ulcer and amputation.

Risk factors for diabetic neuropathy

- duration of diabetes
- poor glycaemic control
- age
**Diagnosis of diabetic neuropathy**

Symptoms and signs of diabetic neuropathy:

- **Peripheral neuropathy:**
  - sensory loss
  - unsteadiness
  - sensory symptoms (pain, unpleasant sensation of burning, tingling or numbness).

- **Autonomic neuropathy:**
  - lack of awareness of hypoglycaemia
  - orthostatic hypotension and resting tachycardia
  - diarrhoea, constipation and fecal incontinence
  - erectile dysfunction, urinary incontinence and bladder dysfunction.

Neuropathy can be present without symptoms.

Peripheral neuropathy is considered probable if there is a combination of two or more of the following: sensory symptoms, decreased distal sensation, or unequivocally decreased or absent ankle reflexes. (See Annex 2 for details.) The diagnosis of autonomic neuropathy requires tests that are not usually performed in primary care facilities.

**Management of diabetic neuropathy**

Specific treatment for the underlying nerve damage is not available. There are several treatment options for pain control, with adjuvant benefits or side effects to consider.

- Exclude causes of peripheral neuropathy other than diabetes (alcohol, chemotherapy, vitamin B12 deficiency, hypothyroidism, renal disease, malignancies, HIV infection).
- Refer patients with painful peripheral neuropathy to specialized care for pharmacological management of pain.
- Refer patients with suspected autonomic neuropathy to specialized care.
- Improve glycaemic control.

**Foot problems in diabetes**

Diabetes dramatically increases the risk of lower extremity amputation. Diabetic foot lesions often result from simultaneous presence of several risk factors, with peripheral diabetic neuropathy playing a central role. It leads to an insensitive and sometimes deformed foot, which can cause an abnormal walking pattern and abnormal biomechanical loading of the foot. The resulting high pressure in some areas of the foot leads to the formation of calluses, further increase in abnormal loading and eventually ulceration.

A diabetic foot ulcer is a localized injury to the skin and/or underlying tissue below the ankle. People with diabetes often have peripheral artery disease as a result of accelerated atherosclerosis. Consequent ischemia impairs wound healing. Many diabetic foot ulcers occur in patients with combined neuropathy and ischaemia and are most often caused by trauma from inappropriate footwear and/or walking barefoot with insensitive feet. Disruption of protective skin allows for colonization.
of subcutaneous tissues by microbes and in many cases the wound becomes clinically infected, requiring antimicrobial treatment and often some form of surgical intervention.

Annex 2 provides details on prevention, assessment and management of diabetic foot.

**Macrovascular complications**

Coronary heart disease, cerebrovascular disease and peripheral vascular disease are a major cause of morbidity and mortality in people with diabetes.

**Blood pressure control**

Blood pressure lowering in people with diabetes reduces the risk of microvascular and macrovascular complications. Control of blood pressure in people with diabetes often requires more than one medication. Thiazide diuretics and ACE inhibitors are recommended to achieve a target blood pressure of <130/80 mmHg. If this is not achievable, refer to a higher level of care. *Other protocols from HEARTS-E are also an option.*

**Control of blood lipids**

Some improvement in lipid profile can be achieved with a healthy diet and physical activity. Statins can reduce the risk of CVD events in people with diabetes. Statins are recommended for all patients of 40 years of age and older with diabetes. If this is not feasible, use statins in patients at highest risk of CVD events (e.g. patients with CVD, patients with nephropathy and/or patients with a high risk on a CVD risk prediction chart).

**Antiplatelet treatment**

Use of antiplatelet treatment is recommended only for secondary prevention of CVD events, even among patients with diabetes. The recommendation is for 75–100 mg of acetylsalicylic acid daily to be prescribed to all people with diabetes who have survived a CVD event and have no history of major bleeding.
**Screening for Chronic Complications**

- Measure blood pressure at every scheduled visit, review medication as per hypertension protocol
- REFER for dilated-pupil retinal exam upon diagnosis, and every two years thereafter, or as per ophthalmologist recommendation
- Examine feet for ulcers at every visit. REFER to higher level of care if ulcer present
- Assess risk of lower limb amputation annually (foot pulses, sensory neuropathy by monofilament, presence of healed or open ulcers, calluses). REFER to higher level of care if ulcer present or pulse absent
- Test for proteinuria annually. REFER to higher level of care if positive.

**Management of Acute Complications**

*Severe hypoglycaemia* (plasma glucose <50 mg/dl or 2.8 mmol/l) or signs:
- If conscious, give a sugar-sweetened drink
- If unconscious, give 20–50 ml of 50% glucose (dextrose) IV over 1–3 minutes.

*Severe hyperglycaemia* (plasma glucose >18 mmol/l (325 mg/dl) and urine ketone 2+) or signs and symptoms of severe hyperglycaemia:
- Set up intravenous drip 0.9% NaCl 1 litre in 2 hours; continue at 1 litre every 4 hours, REFER to hospital.

**Goal for glycaemic control**

<table>
<thead>
<tr>
<th>Fasting</th>
<th>Plasma glucose**</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤7.0 mmol/l (126 mg/dl)**</td>
<td></td>
</tr>
</tbody>
</table>

* REFER to table on diagnostic values for other tests which can be used to diagnose diabetes.
* If they are more affordable than insulin, DPP4-inhibitors, SGLT2-inhibitors or pioglitazone can be used before insulin in cases of treatment failure with metformin and gliclazide. Introduce and titrate insulin treatment according to local practices.
** HbA1c should be used where available.
† Consider less stringent glycaemic control in patients with frequent severe hypoglycaemia, advanced complications, serious comorbidities and/or limited life expectancy.
4 Criteria for referral to higher levels of care

Urgent (same day) referral should occur if one of the following is detected:

- urine ketones >2+, or no improvement in glycaemia ≥18 mmol/L after management with metformin and/or gliclazide
- suspicion of ketoacidosis or HHS (see page 19)
- hypoglycaemia unresolved by treatment (see page 19)
- clinical suspicion of type 1 diabetes in newly diagnosed patient
- symptoms/signs of coronary heart disease and stroke
- recent deterioration of vision
- blood pressure >200/>110 mmHg
- blood pressure >180/>110 mmHg with headache, shortness of breath, blurred vision, changed mental state, nausea, vomiting, reduced urine output
- infected foot ulcer with or without symptoms of systemic infection; gangrene
- critical limb ischaemia
- anuria or eGFR <30 mL/min/1.73 m².

Non-emergency referral should occur if one of the following is detected:

- glycaemia treatment goal is not achieved despite compliance to treatment with oral medication (and insulin)
- eGFR 30–59 mL/min/1.73 m²
- moderately and severely increased albuminuria
- symptoms and signs of peripheral vascular disease
- blood pressure >130/80 mmHg despite treatment with two medications
- total cholesterol >8 mmol/L (310 mg/dL).
5 Monitoring of processes and outcomes

A monitoring system to assess the effectiveness of diabetes management should include regular monitoring of biochemical parameters and occurrence of complications due to diabetes.

Biochemical parameters:

- Glycated haemoglobin (HbA1c) provides a measure of average plasma glucose over the preceding 8 to 12 weeks. The majority of patients can be expected to aim for a HbA1c of ≤7.0% (53 mmol/mol)
- If HbA1c measurement is not available or there is concern over its validity, an FPG value of ≤7.0 mmol/L (126mg/dL) and a postprandial PG value of ≤9.0 mmol/L (160 mg/dL) can serve as surrogates.

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Number of patients being treated for diabetes</strong></td>
<td>Number of patients and number of new patients with diabetes</td>
</tr>
<tr>
<td><strong>Control rate among people treated for diabetes</strong></td>
<td>Numerator: number of patients with diabetes with good glycaemic control at the last clinical visit in the last 6 months (HbA1c &lt;7.0% (53 mmol/mol), or FPG &lt;7.0 mmol/L (126mg/dL) and (if available) a postprandial PG value &lt;9.0 mmol/L (160 mg/dL) Denominator: number of patients with diabetes in the facility during the last 6 months</td>
</tr>
<tr>
<td><strong>Complications due to diabetes:</strong></td>
<td>Numerator: number of new diabetes complications in the past year Denominator: number of patients with diabetes in the past year</td>
</tr>
<tr>
<td>- diabetic foot</td>
<td>Frequency of reporting: monthly</td>
</tr>
<tr>
<td>- nephropathy</td>
<td></td>
</tr>
<tr>
<td>- retinopathy</td>
<td></td>
</tr>
<tr>
<td>- neuropathy</td>
<td></td>
</tr>
<tr>
<td>- cardiovascular diseases</td>
<td></td>
</tr>
</tbody>
</table>
6 Resources

For more detail on the topics discussed here, see the following publications.


Annex 1: Protocol for treatment of type 2 diabetes mellitus with insulin

**START** with 10 units intermediate-acting insulin (NPH) at bedtime
- Continue metformin and sulfonylurea
- Counsel patient on hypoglycaemia
- Counsel patient on symptoms of hyperglycaemia

**REVIEW** in 3 days

**IF** FBG > 7 mmol/L
- **INCREASE INSULIN** dosage by 1–2 units

**REVIEW** in 3 days

**CONTINUE INCREASING DOSAGE** by 1–2 units at 3-day intervals until
- FBG = 4–7 mmol/L
- Do not increase insulin if nocturnal hypoglycaemia occurs

**IF** FBG < 4 mmol/L or if nocturnal hypoglycaemia occurs
- **REDUCE INSULIN** by 1–2 units

**IF** FBG 4–7 mmol/L and daytime hypoglycaemia occurs
- **REDUCE** morning gliclazide by 40 mg
- **STOP** morning gliclazide if daytime hypoglycaemia continues

**IF** FBG > 7 mmol/L AND nocturnal hypoglycaemia occurs,
- **REFER** to higher level of care

**IF** stabilized
- **REVIEW** with HbA1c in 3 months

**IF** HbA1c > 7.5%
- **REFER** to higher level of care for intensification
Annex 2: Prevention, assessment and management of diabetic foot

What are diabetic foot problems?

Diabetic foot problems are amongst the most common, costly and severe complications of diabetes. The term “diabetic foot complications” encompasses the conditions of diabetic foot ulcer (i.e., a full-thickness epithelial defect below/distal to the ankle) and diabetic foot infections (i.e., any soft-tissue or bone infection occurring in the diabetic foot, including osteomyelitis).

A diabetic foot ulcer is a localised injury to the skin and/or underlying tissue below the ankle. Disruption of protective skin allows for colonization of subcutaneous tissues by microbes and in many cases the wound becomes clinically infected, requiring antimicrobial treatment and often some form of surgical intervention.

Diabetic foot ulcers frequently result from a person with diabetes simultaneously having two or more risk factors, with diabetic peripheral neuropathy and peripheral artery disease usually playing a central role.

The neuropathy leads to an insensitive and sometimes deformed foot, often causing abnormal loading of the foot. In people with neuropathy, minor trauma (e.g., from ill-fitting shoes, or an acute mechanical or thermal injury) can precipitate ulceration of the foot. Loss of protective sensation, foot deformities, and limited joint mobility can result in abnormal biomechanical loading of the foot. This produces high mechanical stress in some areas, the response to which is usually thickened skin (callus). The callus then leads to a further increase in the loading of the foot, often with subcutaneous haemorrhage and eventually skin ulceration. Whatever the primary cause of ulceration, continued walking on the insensitive foot impairs healing of the ulcer.

Peripheral artery disease (PAD), generally caused by atherosclerosis, is present in up to 50% of patients with a diabetic foot ulcer. PAD is an important risk factor for impaired wound healing and lower extremity amputation. A small percentage of foot ulcers in patients with severe PAD are purely ischaemic; these are usually painful and may follow minor trauma.
Common risk factors for the development of foot ulcers in people with diabetes

- peripheral vascular disease
- neuropathy
- poor glycaemic control
- cigarette smoking
- diabetic nephropathy
- previous foot ulcerations/amputations

Symptoms

Patients can present with symptoms of peripheral neuropathy and or peripheral artery occlusion, such as:

- pain in the legs or cramping in the thighs or calves during physical activity
- tingling, burning, or pain in the feet
- loss of sense of touch or ability to feel heat or cold very well
- a change in the shape of feet over time
- dry, cracked skin on the feet
- a change in the color and temperature of the feet
- thickened, yellow toenails
- fungal infections between the toes
- a blister, sore, ulcer, infected corn, or ingrown toenail.

However, the absence of symptoms does not exclude diabetic foot problems.

Assessment and management of diabetic foot problems

Examination of the feet

Remove the patient’s shoes, socks, dressings, and bandages and carry out the following examination procedures:

Palpation of arteries

**Fig. 5 Dorsal pedis palpation**

*Dorsal pedis*: Feel in the middle of the dorsum of the foot just lateral to the tendon of extensor hallucis longus (extensor tendon of the great toe).

**Fig. 6 Posterior tibial artery palpation**

*Posterior tibial artery*: Feel midway between medial malleolus and tendon calcaneus.
Assessing loss of protective sensation (LOPS)

Assessment of LOPS is done with one of the following techniques:

a) Pressure perception: Semmes-Weinstein 10 g monofilament (if monofilament is not available, see “method c” below). Sensory examination should be carried out in a quiet and relaxed setting.

- First, apply the monofilament on the patient’s hands (or elbow or forehead) so that she or he knows what to expect. The patient must not be able to see whether or where the examiner applies the filament.
- Conduct the test on three sites on both feet (Fig. 6 and Fig. 7).
- The total duration – skin contact and removal of the filament should be approximately 2 secs.
- Apply the filament along the perimeter of, not on, an ulcer site callus or necrotic tissue.
- Do not allow the filament to slide across the skin or make repetitive contact at the test site.
- Apply the monofilament perpendicular to the skin surface. Apply sufficient force to cause the filament to bend or buckle.
- Press the filament to the skin and ask the patient whether they feel the pressure applied (Yes/No).
- Next, ask where they feel the pressure (right foot/left foot).
- Repeat this application twice at the same site but alternate this with one “mock” application in which no filament is applied.

b) Vibration perception: 128 Hz tuning fork. The proper method for using a 128 Hz tuning fork to check for vibratory sensation is as follows:

- First, apply the tuning fork on the patient’s wrist (or elbow or clavicle) to demonstrate what the sensation feels like.
- Ensure the patient cannot see whether or where the examiner applies the tuning fork.
- Apply the tuning fork to a bony part on the dorsal side of the distal phalanx of the first toe (or another toe if the hallux is absent).
- Apply the tuning fork perpendicularly, with constant pressure (Fig. 8).
- Repeat this application twice, but alternate this with at least one “mock”
application in which the tuning fork is not vibrating.

- The test is positive if the patient correctly answers at least two out of three applications, and negative if two out of three answers are incorrect.
- If the patient is unable to sense the vibrations on the toe, repeat the test more proximally (e.g., malleolus, tibial tuberosity).

c) When monofilament or tuning fork are not available, test tactile sensation with the light touch test. This simple test (also called the Ipswich Touch Test) can be used to screen for loss of protective sensation (LOPS), when the 10 g monofilament or 128 Hz tuning fork is not available. The test has reasonable agreement with these tests to determine LOPS, but its accuracy in predicting foot ulcers has not been established.

- Instruct the subject to close their eyes and to say “yes” when they feel the touch.
- The examiner lightly sequentially touches with the tip of her/his index finger the tips of the first, third, and fifth toes of both feet for 1–2 seconds. When touching, do not push, tap, or poke.
- LOPS is likely when light touch is not sensed in ≥2 sites.

Stratification and management of risk

Table 8 Stratification of level of risk of developing diabetic foot problems or the need for an amputation

<table>
<thead>
<tr>
<th>Risk level</th>
<th>Low risk</th>
<th>Moderate risk</th>
<th>High risk</th>
<th>Active foot problem</th>
</tr>
</thead>
<tbody>
<tr>
<td>Features</td>
<td>No risk factor except callus alone</td>
<td>Any of: • deformity • neuropathy • non-critical limb ischaemia</td>
<td>Any of: • previous ulcer • previous amputation • neuropathy with non-critical limb ischaemia • neuropathy with callus and/or deformity • non-critical limb ischaemia with callus and/or deformity</td>
<td>Any of: • ulcer • spreading infections • critical limb ischaemia • gangrene • suspicion of acute Charcot arthropathy • unexplained red swollen foot</td>
</tr>
<tr>
<td>Action</td>
<td>Assess annually</td>
<td>Assess every 3–6 months</td>
<td>Assess every 1–3 months</td>
<td>Urgent referral</td>
</tr>
</tbody>
</table>

Patients with diabetes should receive counselling on avoidance of foot complications (see below).

- Patients at low risk can be assessed annually, those at moderate risk every 3–6 months, and those at high risk every 1–3 months.
- Pre-ulcerative lesions need to be treated by a trained professional by:
  - removal of callus
  - protection or draining of blisters
  - treatment of ingrown and thickened nails
  - antifungal treatment for fungal infections.
Management of active diabetic foot problems

Active foot problems require referral to a higher level of care. Best results in prevention of amputations have been achieved in settings where multidisciplinary facilities are available.

When to refer

Patients with a foot ulcer should be referred to a more specialized level for further evaluation if management by a trained professional and the necessary equipment and consumables are not available at the primary care level. Patients with gross foot deformities and/or absent peripheral pulses, and patients with suspected lower limb ischaemia will need to be referred to a higher level of care.

Urgent referral to acute services is recommended for patients with any of the following:

- infected ulcer
- spreading infection
- critical limb ischaemia
- gangrene
- suspicion of acute Charcot arthropathy
- unexplained red swollen foot.

Counselling patients on foot care

To avoid serious foot problems that could result in losing a toe, foot or leg:

- Inspect your feet daily. Check for cuts, blisters, redness, swelling, or nail problems. Use a magnifying hand mirror to look at the bottom of your feet.
- Bathe feet in lukewarm, never hot, water. Keep your feet clean by washing them daily. Use only lukewarm water – the temperature you would use on a newborn baby.
- Be gentle when bathing your feet. Wash them using a soft washcloth or sponge. Dry by blotting or patting, and dry carefully between the toes.
- Moisturize your feet but not between your toes. Use a moisturizer daily to keep dry skin from itching or cracking. But don’t moisturize between the toes – that could encourage a fungal infection.
- Cut nails carefully. Cut them straight across and file the edges. Don’t cut nails too short, as this could lead to ingrown toenails. If you have concerns about your nails, consult your doctor.
- Never treat corns or calluses yourself. No “bathroom surgery” or medicated pads. Visit your doctor for appropriate treatment.
- Shake out your shoes and feel the inside before wearing. Remember, your feet may not be able to feel a pebble or other foreign object, so always inspect your shoes before putting them on.
• Wear socks and appropriate footwear. The inside length of the shoe should be 1–2 cm longer than your foot, and should be neither too tight nor too loose.

• Keep your feet warm and dry.

• Never walk barefoot, not even at home. Always wear shoes or slippers. You could step on something and get a scratch or cut.

• Take care of your diabetes. Keep your blood glucose levels under control.

• Do not smoke. Smoking restricts blood flow in your feet.

• Get your feet examined regularly.

Resources
For further information on care of diabetic foot see the following publications:


American College of Foot and Ankle Surgeons (ACFAS). Foot health facts.


Diabetes and your feet. (https://www.cdc.gov/features/diabetesfoothealth/index.html)