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Diabetes Mellitus
Guidelines

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Definition

Diabetes mellitus (DM) is a metabolic disease, involving inappropriately elevated blood glucose levels. DM has several categories, including type 1, type 2, maturity-onset diabetes of the young (MODY), gestational diabetes, neonatal diabetes, and secondary causes due to endocrinopathies, steroid use, etc.

The main subtypes of DM are Type 1 diabetes mellitus (T1DM) and Type 2 diabetes mellitus (T2DM), which classically result from defective insulin secretion (T1DM) and/or action (T2DM). T1DM is present in children or adolescents, while T2DM is thought to affect middle-aged and older adults who have prolonged hyperglycemia due to poor lifestyle and dietary choices. The pathogenesis for T1DM and T2DM is drastically different, and therefore each type has various etiologies, presentations, and treatments.

- **T1DM** is characterized by the destruction of beta cells in the pancreas, typically secondary to an autoimmune process. The result is the absolute destruction of beta cells, and consequentially, insulin is absent or extremely low.

- **T2DM** involves a more insidious onset where an imbalance between insulin levels and insulin sensitivity causes a functional deficit of insulin. Insulin resistance is multifactorial but commonly develops from obesity and aging.
- **MODY** is a heterogeneous disorder identified by non-insulin-dependent diabetes diagnosed at an early age (usually under 25 years). It carries an autosomal dominant transmission and does not involve autoantibodies as in T1DM. Several genes have implications in this disease, including mutations to hepatocyte nuclear factor-1-alpha (HNF1A) and the glucokinase (GCK) gene, which occurs in 52 to 65 and 15 to 32 percent of MODY cases, respectively. The genetics of this disease are still unclear as some patients have mutations but never develop the disease, and others will develop clinical symptoms of MODY but have no identifiable mutation.

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- **Gestational diabetes** is essentially diabetes that manifests during pregnancy. It is still unknown why it develops; however, some speculate that HLA antigens may play a role, specifically HLA DR2, 3, and 4. Excessive proinsulin is also thought to play a role in gestational diabetes, and some suggest that proinsulin may induce beta-cell stress. Others believe that high concentrations of hormones such as progesterone, cortisol, prolactin, human placental lactogen, and estrogen may affect beta-cell function and peripheral insulin sensitivity.
- **In addition to T1DM, T2DM, and MODY,** any disorder that

1. damage to the pancreas can result in DM including Cystic fibrosis, Hereditary hemochromatosis, Pancreatic cancer, Chronic pancreatitis.

2. Several endocrinopathies, including acromegaly, Cushing syndrome, glucagonoma, hyperthyroidism, hyperaldosteronism, and somatostatinomas, have been associated with glucose intolerance and diabetes mellitus, due to the inherent glucogenic action of the endogenous hormones excessively secreted in these conditions.

3. Drug-induced insulin resistance is also in the differential of classical diabetes. These drugs include Phenytoin, Glucocorticoids, Estrogen

Complications

Regardless of the specific type of diabetes, complications involve microvascular, macrovascular, and neuropathic issues. Microvascular and macrovascular complications vary according to the degree and the duration of poorly control diabetes and include nephropathy, retinopathy, neuropathy, and one of the most devastating consequences of DM is its effect on cardiovascular disease (ASCVD), especially if it is associated with other comorbidities like dyslipidemia and hypertension.

The most acute complication of DM is diabetic ketoacidosis (DKA), which typically presents in T1DM. This condition is usually either due to inadequate dosing, missed doses, or ongoing infection. In this condition, the lack of insulin means that tissues are unable to obtain glucose from the bloodstream. Compensation for this causes the metabolism of lipids into ketones as a substitute energy source, which causes systemic acidosis, and can be calculated as a high anion-gap metabolic acidosis. The combination of hyperglycemia and ketosis causes diuresis, acidemia, and vomiting leading to dehydration and electrolyte abnormalities, which can be life-threatening.

In T2DM, hyperosmolar hyperglycemic syndrome (HHS) is an emergent concern. It presents similarly to DKA with excessive thirst, elevated blood glucose, dry mouth, polyuria, tachypnea, and tachycardia. However, unlike DKA, HHS typically does not present with excessive urinary ketones since insulin still gets produced by pancreatic beta cells. Treatment for DKA or HHS involves insulin administration and aggressive intravenous hydration. Careful management of electrolytes, particularly potassium, is critical in the management of these emergent conditions.

Diagnosis

According to the American Diabetes Association (ADA), a diagnosis of diabetes is through any of the following:

1. An HbA1c level of 6.5% or higher

2. A fasting plasma glucose level of 126 mg/dL (7.0 mmol/L) or higher (no caloric intake for at least 8 hours)

3. A two-hour plasma glucose level of 11.1 mmol/L or 200 mg/dL or higher during a 75-g OGTT

4. A random plasma glucose of 11.1 mmol/L or 200 mg/dL or higher in a patient with symptoms of hyperglycemia (polyuria, polydipsia, polyphagia, weight loss) or hyperglycemic crisis.

If borderline, a glucose tolerance test is an option to evaluate both fasting glucose levels and serum response to an oral glucose tolerance test (OGTT). Prediabetes, which often precedes T2DM, presents with a fasting blood glucose level of 100 to 125 mg/dL or a 2-hour post-oral glucose tolerance test (post-OGTT) glucose level of 140 to 200 mg/dL.

To test for gestational diabetes, all pregnant patients have screening between 24 to 28 weeks of gestation with a 1-hour fasting glucose challenge test. If blood glucose levels are over 140mg/dL, patients have a 3-hour fasting glucose challenge test to confirm a diagnosis. A positive 3-hours OGTT test is when there is at least one abnormal value (greater than or equal to 180, 155, and 140 mg/dL for fasting one-hour, two-hour, and 3-hour plasma glucose concentration, respectively).

Several lab tests are useful in the management of chronic DM. Home glucose testing can show trends of hyper- and hypoglycemia. The HbA1c test indicates the extent of glycation due to hyperglycemia over three months (the life of the red blood cell). Urine albumin testing can identify the early stages of diabetic nephropathy. Since patients with diabetes are also prone to cardiovascular disease, serum lipid monitoring is advisable at the time of diagnosis. Similarly, some recommend monitoring thyroid status by obtaining a blood level of thyroid-stimulating hormone annually due to a higher incidence of hypothyroidism.

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Management

A. Assessment of Glycemic Control

Glycemic control is assessed by:

1. The HbA1C measurement
2. Continuous glucose monitoring (CGM) using time in range (TIR) and/or glucose management indicator (GMI)
3. Blood glucose monitoring (BGM).

1. Glucose Assessment by HbA1C measurement

Recommendations

1. Assess glycemic status by HBA1C at least two times a year in patients who are meeting treatment goals (and who have stable glycemic control).
2. Assess glycemic status at least quarterly and as needed in patients whose therapy has recently changed and/or who are not meeting glycemic goals.

The frequency of A1C testing should depend on the clinical situation, the treatment plan, and the clinician's judgment. The use of HbA1C testing or CGM-derived TIR and GMI may provide an opportunity for more timely treatment changes during encounters between patients and health care professionals.

HbA1C Limitations

- Hemoglobin variants must be considered. Conditions that affect red blood cell turnover (hemolytic and other anemias, glucose-6-phosphate dehydrogenase deficiency, recent blood transfusion, use of drugs that stimulate erythropoiesis, end-stage kidney disease, and pregnancy) may result in discrepancies between the A1C result and the patient's true mean glycaemia.
- A1C does not provide a measure of glycemic variability or hypoglycemia. For patients prone to glycemic variability, especially people with type 1 diabetes or type 2 diabetes with severe insulin deficiency, glycemic control is best evaluated by the combination of results from BGM/CGM and A1C

2. Glucose Assessment by Continuous Glucose Monitoring

Recommendations

- Standardized, single-page glucose reports from continuous glucose monitoring (CGM) devices with visual cues, such as the ambulatory glucose profile, should be considered as a standard summary for all CGM devices.

Time in range (TIR) is associated with the risk of microvascular complications and can be used for assessment of glycemic control. Additionally, time below range (TBR) and time above range (TAR) are useful parameters for the evaluation of the treatment plan.

Standardized CGM report for clinical care

1. Number of days CGM device is worn (recommend 14 days)	
2. Percentage of time CGM device is active (recommend 70% of data from 14 days)	
3. Mean glucose	
4. Glucose management indicator	
5. Glycemic variability (%CV) target ≤36%*	
6. TAR: % of readings and time >250 mg/dL (>13.9 mmol/L)	Level 2 hypoglycemia
7. TAR: % of readings and time 181–250 mg/dL (10.1–13.9 mmol/L)	Level 1 hypoglycemia
8. TIR: % of readings and time 70–180 mg/dL (3.9–10.0 mmol/L)	In Range
9. TBR: % of readings and time 54–69 mg/dL (3.0–3.8 mmol/L)	Level 1 hypoglycemia
10. TBR: % of readings and time <54 mg/dL (<3.0 mmol/L)	Level 2 hypoglycemia

CGM is rapidly improving diabetes management. As stated in the recommendations, time in range (TIR) is a useful metric of glycemic control and glucose patterns, and it correlates well with HbA1C in most studies. Additionally, time below range (<70 and <54 mg/dL [3.9 and 3.0 mmol/L]) and time above range (>180 mg/dL [10.0 mmol/L]) are useful parameters for insulin dose adjustments and reevaluation of the treatment plan.

For many people with diabetes, glucose monitoring is key for achieving glycemic targets. CGM has become a standard method for glucose monitoring for most adults with type 1 diabetes. Both approaches to glucose monitoring allow patients to evaluate individual responses to therapy and assess whether glycemic targets are being safely achieved.

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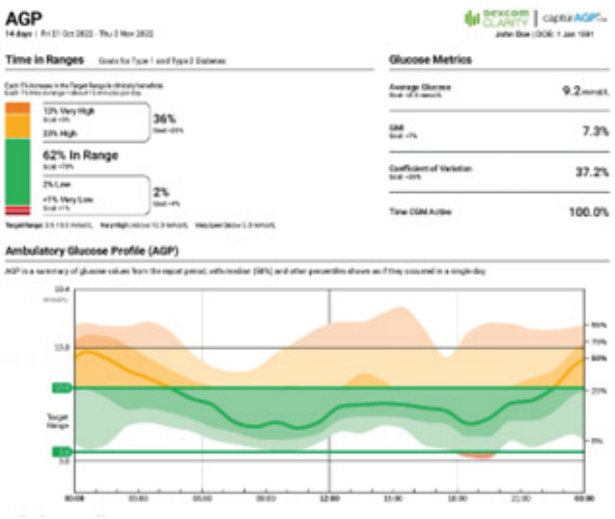
To make these metrics more actionable, standardized reports with visual cues, such as the ambulatory glucose profile (Fig 1), are recommended and may help the patient and the health care professional better interpret the data to guide treatment decisions. BGM and CGM can be useful to guide medical nutrition therapy and physical activity, prevent hypoglycemia, and aid medication management.

While A1C is currently the primary measure to guide glucose management and a valuable risk marker for developing diabetes complications, the CGM and GMI provide the insights for a more personalized diabetes management plan.

Figure 1



With the advent of recent technology, CGM has evolved rapidly in both accuracy and affordability. As such, many patients have these data available to assist with self-management and their health care professionals' assessment of glycemic status. Reports can be generated from CGM that will allow the health care professional and person with diabetes to determine TIR, calculate GMI, and assess hypoglycemia, hyperglycemia, and glycemic variability.

B. Glycemic Targets

An A1C goal for many nonpregnant adults of <7% (53 mmol/mol) without significant hypoglycemia is appropriate.

If using ambulatory glucose profile/glucose management indicator to assess glycemia, a parallel goal for many nonpregnant adults is time in range of >70% with time below range <4% and time above <54 mg/dL <1%. For those with frailty or at considerable risk of hypoglycemia, a target of >50% time in range with <1% time below range is recommended.

C. Pharmacologic Therapy for Type 1 Diabetes

- Intensive insulin replacement with multiple daily injections of insulin or continuous subcutaneous administration through an insulin pump, providing the best combination of effectiveness and safety for people with type 1 diabetes.
- Most individuals with type 1 diabetes should use rapid-acting insulin analogs to reduce hypoglycemia risk.
- Individuals with type 1 diabetes should receive education on how to match mealtime insulin doses to carbohydrate intake, fat, and protein content, and anticipated physical activity.

1. Nsulin Therapy

Because the hallmark of type 1 diabetes is absent or near-absent β-cell function, insulin treatment is essential for individuals with type 1 diabetes. Human Insulin and Insulin Analogs are available for insulin replacement therapy.

Insulins also are classified by the timing of their action in the body – specifically, how quickly they start to act (ONSET), when they have a maximal effect (PEAK)and how long they act (DURATION OF ACTION).

A variety of insulins (basal and prandial) and modes of administration (syringe, pen, prefilled pen, and pump) are available. Achieving optimal glycemic control while avoiding hypoglycemia and other adverse effects (such as excessive weight gain and lipodystrophy) requires individualized insulin therapy supplemented by glucose monitoring (either intermittent self-monitoring of blood glucose with a glucose meter or CGM) and proper therapeutic education (carbohydrate counting and insulin dose adjustments according to carbohydrate intake, activity levels, and blood glucose values, and proper injection technique)

Insulin replacement regimens typically consist of

1. Basal-bolus insulin regimens
2. multiple daily injections (MDI) of basal and prandial (mealtime) insulin
3. continuous subcutaneous insulin infusion (CSII).

Management

In general, individuals with type 1 diabetes require 50% of their daily insulin as basal and 50% as prandial, but this is dependent on a number of factors, including whether the individual consumes lower or higher carbohydrate meals. Total daily insulin requirements can be estimated based on weight, with typical doses ranging from 0.4 to 1.0 units/kg/day.

Higher amounts are required during puberty, pregnancy, and medical illness. The American Diabetes Association notes 0.5 units/kg/day as a typical starting dose in individuals with type 1 diabetes who are metabolically stable, with half administered as prandial insulin given to control blood glucose after meals and the other half as basal insulin to control glycaemia in the periods between meal absorption.

2. Noninsulin Treatments for Type 1 Diabetes

Injectable and oral glucose-lowering drugs have been studied for their efficacy as adjuncts to insulin treatment of type 1 diabetes.

- **Pramlintide** is based on the naturally occurring β-cell peptide amylin and is approved for use in adults with type 1 diabetes. Results from randomized controlled studies show variable reductions of A1C (0–0.3%) and body weight (1–2 kg) with addition of pramlintide to insulin.

- The addition of metformin to adults with type 1 diabetes caused small reductions in body weight and lipid levels but did not improve A1C.

- The addition of the glucagon-like peptide 1 (GLP-1) receptor agonists (RAs) liraglutide and exenatide to insulin therapy caused small (0.2%) reductions in A1C compared with insulin alone in people with type 1 diabetes and also reduced body weight by ~ 3 kg.

- The addition of a sodium–glucose cotransporter 2 (SGLT2) inhibitor to insulin therapy has been associated with improvements in A1C and body weight when compared with insulin alone; however, SGLT2 inhibitor use in type 1 diabetes is associated with a two- to fourfold increase in ketoacidosis.

- **The risks and benefits of adjunctive agents continue to be evaluated, but only pramlintide is approved for treatment of type 1 diabetes.**

D. Pancreas and Islet Transplantation

Successful pancreas and islet transplantation can normalize glucose levels and mitigate microvascular complications of type 1 diabetes. However, patients receiving these treatments require life-long immunosuppression to prevent graft rejection and/or recurrence of autoimmune islet destruction. Given the potential adverse effects of immunosuppressive therapy, pancreas transplantation should be reserved for patients with type 1 diabetes undergoing simultaneous renal transplantation, following renal transplantation, or for those with recurrent ketoacidosis or severe hypoglycemia despite intensive glycemic management. With the advent of improved continuous glucose monitors, closed-loop pump-sensor systems, and devices that offer alternative approaches for patients with hypoglycemia unawareness, the role of pancreas transplantation alone, as well as islet transplant, will need to be reconsidered.

E. Pharmacologic Therapy for Type 2 Diabetes

Metformin is the preferred initial pharmacologic agent for the treatment of type 2 diabetes.
The American Diabetes Association/European Association for the Study of Diabetes consensus report "Management of Hyperglycemia in Type 2 Diabetes, 2018" and the 2019 update recommend a patient-centered approach to choosing appropriate pharmacologic treatment of blood glucose (Fig.2). This includes consideration of efficacy and key patient factors:

1. important comorbidities such as atherosclerotic cardiovascular disease (ASCVD) and indicators of high ASCVD risk, chronic kidney disease (CKD), and heart failure (HF)
2. hypoglycemia risk
3. effects on body weight
4. side effects
5. cost
6. patient preferences.

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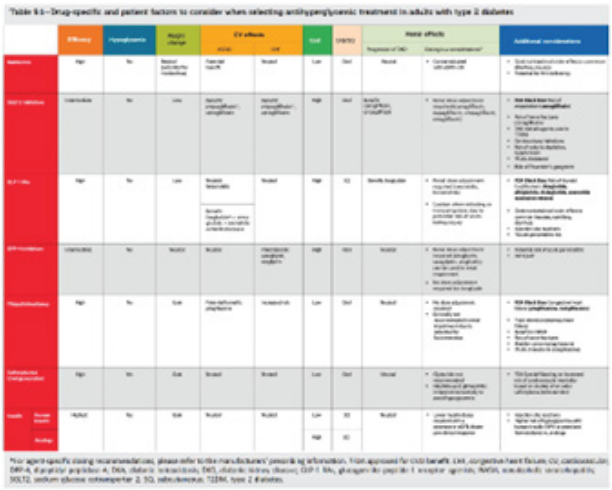
Figure 1

Drug-specific and patient factors to consider when selecting antihyperglycemic treatment in adults with type 2 diabetes.



Lifestyle modifications must be combined with oral pharmacologic agents for optimal glycemic control, particularly as type 2 diabetes mellitus progresses with continued loss of pancreatic beta-cell function and insulin production.

- **Classification of Oral Hypoglycemic Medications**

Sulfonylureas bind to adenosine triphosphate-sensitive potassium channels (K-ATP channels) in the beta cells of the pancreas; this leads to the inhibition of those channels and alters the resting membrane potential of the cell, causing an influx of calcium and the stimulation of insulin secretion.

Meglitinides exert their effects via different pancreatic beta-cell receptors, but they act similarly to sulfonylureas by regulating adenosine triphosphate-sensitive potassium channels in pancreatic beta cells, thereby causing an increase in insulin secretion.

Metformin increases hepatic adenosine monophosphate-activated protein kinase activity, thus reducing hepatic gluconeogenesis and lipogenesis and increasing insulin-mediated uptake of glucose in muscles.

Thiazolidinediones activate peroxisome proliferator-activated receptor gamma (PPAR-γ), a nuclear receptor, which increases insulin sensitivity and resultant peripheral uptake of glucose and increases the level of adiponectin, a fat tissue-secreted cytokine, which increases not only the number of insulin-sensitive adipocytes but also stimulates fatty acid oxidation.

Alpha-glucosidase inhibitors competitively inhibit alpha-glucosidase enzymes in the intestinal brush border cells that digest the dietary starch, thus inhibiting the polysaccharide reabsorption and the metabolism of sucrose to glucose and fructose.

DPP-4 inhibitors inhibit the enzyme dipeptidyl peptidase 4 (DPP- 4). These deactivate glucose-dependent insulinotropic polypeptide (GIP) and glucagon-like peptide 1 (GLP-1), among others. Therefore, these influence glucose control through multiple effects, such as decreasing glucagon release and increasing glucose-dependent insulin release, decreasing gastric emptying, and increasing satiety.

GLP-1 and Dual GLP-1/GIP Receptor Agonists

These medications have similar effects to the GLP-1 and GIP produced in the body but are resistant to being broken down by the DPP-4 enzyme. These medications can result in large benefits in lowering blood glucose and body weight. Some agents in this class have also been shown to prevent heart disease. Most of these medications are injected, with the exception of one that is taken by mouth once daily, called semaglutide (Rybelsus).

Injectable GLP-1 receptor agonists currently on the market include:

Dulaglutide (Trulicity), Exenatide (Byetta), Exenatide extended release (Bydureon), Liraglutide (Victoza), Lixisenatide (Adlyxin), Injectable semaglutide (Ozempic) One dual GLP-1/GIP receptor agonist is currently on the market called tirzepatide (Mounjaro).

SGLT2 inhibitors inhibit sodium-glucose co-transporter 2 (SGLT-2) in proximal tubules of renal glomeruli, causing inhibition of 90% glucose reabsorption and resulting in glycosuria in people with diabetes which in turn lowers the plasma glucose levels.

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Cycloset, a sympatholytic dopamine D2 receptor agonist, resets the hypothalamic circadian rhythm, which might have been altered by obesity. This action results in the reversal of insulin resistance and a decrease in glucose production.

- **Adverse Effects**

The following are adverse effects of various hypoglycemic drugs:

Sulfonylureas: Syncope (less than 3%), dizziness (2% to 7%), nervousness (4%), anxiety (less than 3%), depression (<3%), hypoesthesia (less than 3%), insomnia (<3%), pain (<3%), paresthesia (less than 3%), drowsiness (2%), headache (2%), diaphoresis (less than 3%), pruritus (1% to less than 3%), hypoglycemia (less than 3%), increased lactate dehydrogenase, diarrhea (1% to 5%), flatulence (3%), dyspepsia (less than 3%), and vomiting (less than 3%).

Repaglinide: Hypoglycemia (16% to 31%), weight gain, headache (9% to 11%), upper respiratory tract infection (10% to 16%), and cardiovascular ischemia (4%).

Metformin: Gastrointestinal upset such as diarrhea (12% to 53%), nausea and vomiting (7% to 26%), flatulence (4% to 12%), chest discomfort, flushing, palpitation, headache (5% to 6%), chills, dizziness, taste disorder, diaphoresis, nail disease, skin rash, vitamin B12 deficiency. Also, in less than 1% of patients, it causes lactic acidosis, which can be life-threatening, and is precipitated by conditions predisposing to hypoperfusion and hypoxemia, such as severe renal failure (eGFR less than 30 ml/min/1.73 m2).

Thiazolidinediones: Edema (less than or equal to 27%), hypoglycemia (less than or equal to 27%), cardiac failure (less than or equal to 8%), headache, bone fracture (less than or equal to 5%), myalgia (5%), sinusitis (6%), and pharyngitis.

Alpha-glucosidase inhibitors: Adverse effects include flatulence (74%) that tends to decrease with time, diarrhea (31%), abdominal pain (19%), and increased serum transaminases (less than or equal to 4%).

DPP4 inhibitors:
Sitagliptin: Hypoglycemia (1%), nasopharyngitis (5%), increased serum creatinine, acute pancreatitis (including hemorrhagic or necrotizing forms), and acute renal failure.

Saxagliptin: Peripheral edema (4%), headache (7%), hypoglycemia (6%), urinary tract infection (7%), lymphocytopenia (2%), and acute pancreatitis.
Linagliptin: Hypoglycemia (7%), increased uric acid (3%), increased serum lipase (8%; more than three times upper limit of normal), nasopharyngitis (7%), and acute pancreatitis.

SGLT-2 inhibitors: Dyslipidemia (3%), hyperphosphatemia (2%), hypovolemia (1%), nausea, fungal vaginosis (7% to 8%), urinary tract infection (6%), increased urine output (3% to 4%), dysuria (2%), influenza (2% to 3%), bone fracture (8%), and renal impairment.

GLP-1 and Dual GLP-1/GIP Receptor Agonists

The most frequently exhibited side effects from GLP-1 agonists include nausea, vomiting, and diarrhea that could lead to an acute kidney injury due to volume contraction. Dizziness, mild tachycardia, infections, headaches, and dyspepsia may also occur. Injection-site pruritus and erythema are also common, most notably with the longer-acting medications in this class.

There is a minimal risk of minor episodes of hypoglycemia; however, research has not described any major hypoglycemic episodes at this time. Patients can form antibodies to particular GLP-1 analogs that could affect the efficacy of these medications, particularly with exenatide. This immunogenicity could lead to injection site reactions and even potential anaphylaxis. Anti-drug antibodies were more common, and titers were higher with the weekly dosed formulation of exenatide than with the twice-daily formulation of exenatide.

Combination therapy with GLP-1 agonists and dipeptidyl peptidase-4 inhibitors is not a current recommendation due to statistically insignificant glycemic improvement and enhanced hypoglycemic effects. The interactions between GLP-1 agonists and other oral anti-diabetic medications remain unclear.
Cycloset: Dizziness, fatigue, headache, constipation, rhinitis, nausea, and weakness.

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- **Contraindications**

The following are contraindications for different classes of oral hypoglycemic drugs.
Metformin: Hypersensitivity to the drug, severe renal dysfunction (eGFR less than 30 mL/minute/1.73 m2), and metabolic acidosis, including diabetic ketoacidosis.
Sulfonylureas: Hypersensitivity to the drug or sulfonamide derivatives, type 1 diabetes mellitus, and diabetic ketoacidosis.
Pioglitazone: Hypersensitivity to the drug, New York Heart Association Class III or IV heart failure, serious hepatic impairment, bladder cancer, history of macroscopic hematuria, and pregnancy.
Alpha-glucosidase inhibitors: Hypersensitivity to acarbose, diabetic ketoacidosis, cirrhosis, inflammatory bowel disease, ulcers of the intestine, partial intestinal obstruction, digestive and absorptive issues

SGLT 2 inhibitors: History of serious hypersensitivity to the drug, end-stage renal disease (ESRD), and patients on dialysis.
DPP-4 inhibitors: Dose adjustment of saxagliptin is needed for eGFR less than 45 mL/min/1.73 m^2 with the dose of 2.5 mg once daily. For sitagliptin, a low dose of 25 mg daily is given in patients with a creatinine clearance of less than 30 ml/min/1.73 m^2 and is contraindicated in patients on hemodialysis or peritoneal dialysis. Linagliptin does not need any dose adjustment.
Cycloset: Allergy to the drug, breastfeeding, and syncopal migraine.

Coding

CPT code	Description
E08.0	Diabetes mellitus due to underlying condition with hyperosmolarity
E08.1	Diabetes mellitus due to underlying condition with ketoacidosis
E08.2	Diabetes mellitus due to underlying condition with kidney complications
E08.3	Diabetes mellitus due to underlying condition with ophthalmic complications
E08.4	Diabetes mellitus due to underlying condition with neurological complications
E08.5	Diabetes mellitus due to underlying condition with circulatory complications
E08.6	Diabetes mellitus due to underlying condition with other specified complications
E10.1	Type 1 diabetes mellitus with ketoacidosis
E10.2	Type 1 diabetes mellitus with kidney complications
E10.3	Type 1 diabetes mellitus with ophthalmic complications
E10.4	Type 1 diabetes mellitus with neurological complications
E10.5	Type 1 diabetes mellitus with circulatory complications
E10.6	Type 1 diabetes mellitus with other specified complications
E11.0	Type 2 diabetes mellitus with hyperosmolarity
E11.1	Type 2 diabetes mellitus with ketoacidosis
E11.2	Type 2 diabetes mellitus with kidney complications
E11.3	Type 2 diabetes mellitus with ophthalmic complications
E11.4	Type 2 diabetes mellitus with neurological complications
E11.5	Type 2 diabetes mellitus with circulatory complications
E11.6	Type 2 diabetes mellitus with other specified complications

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