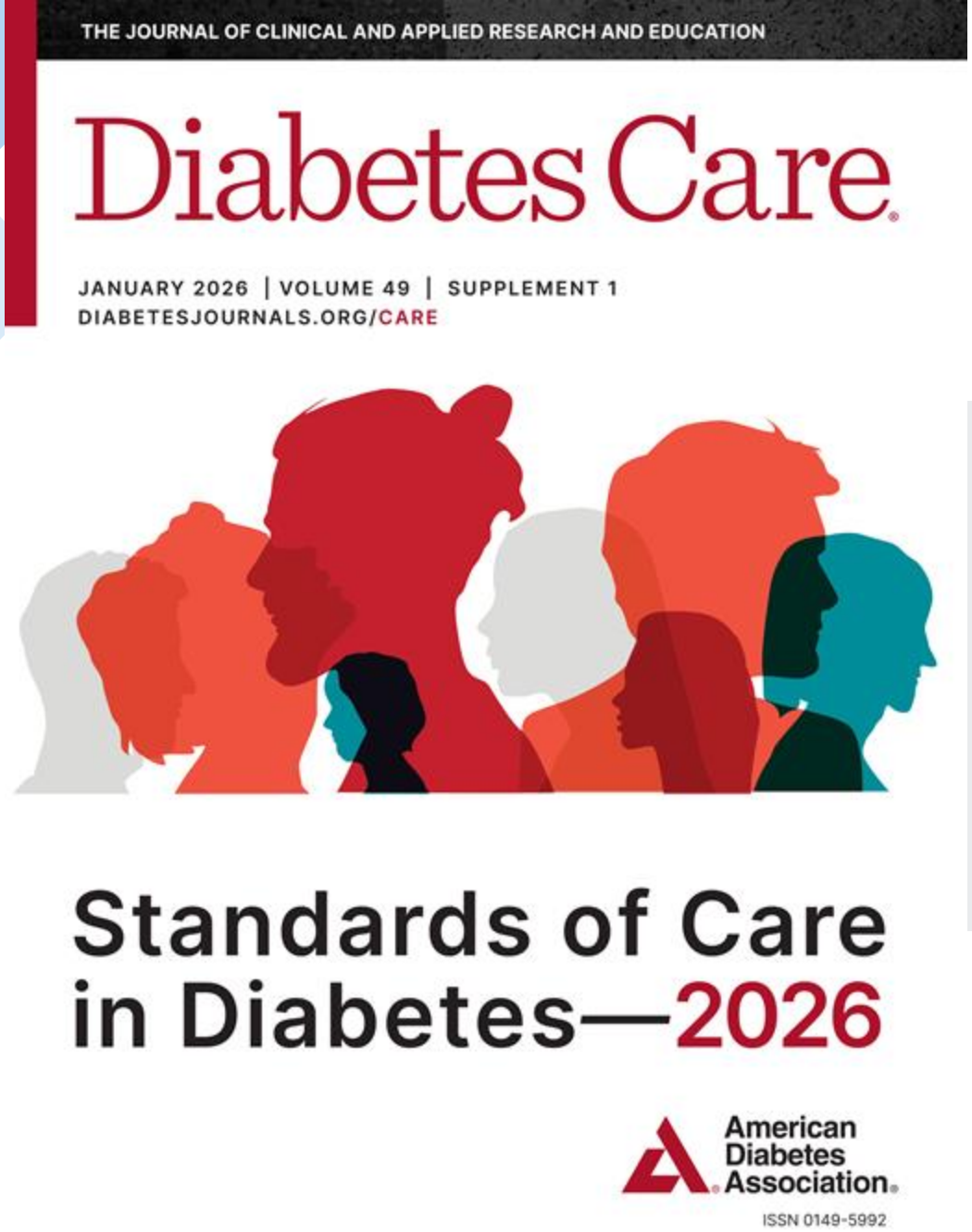
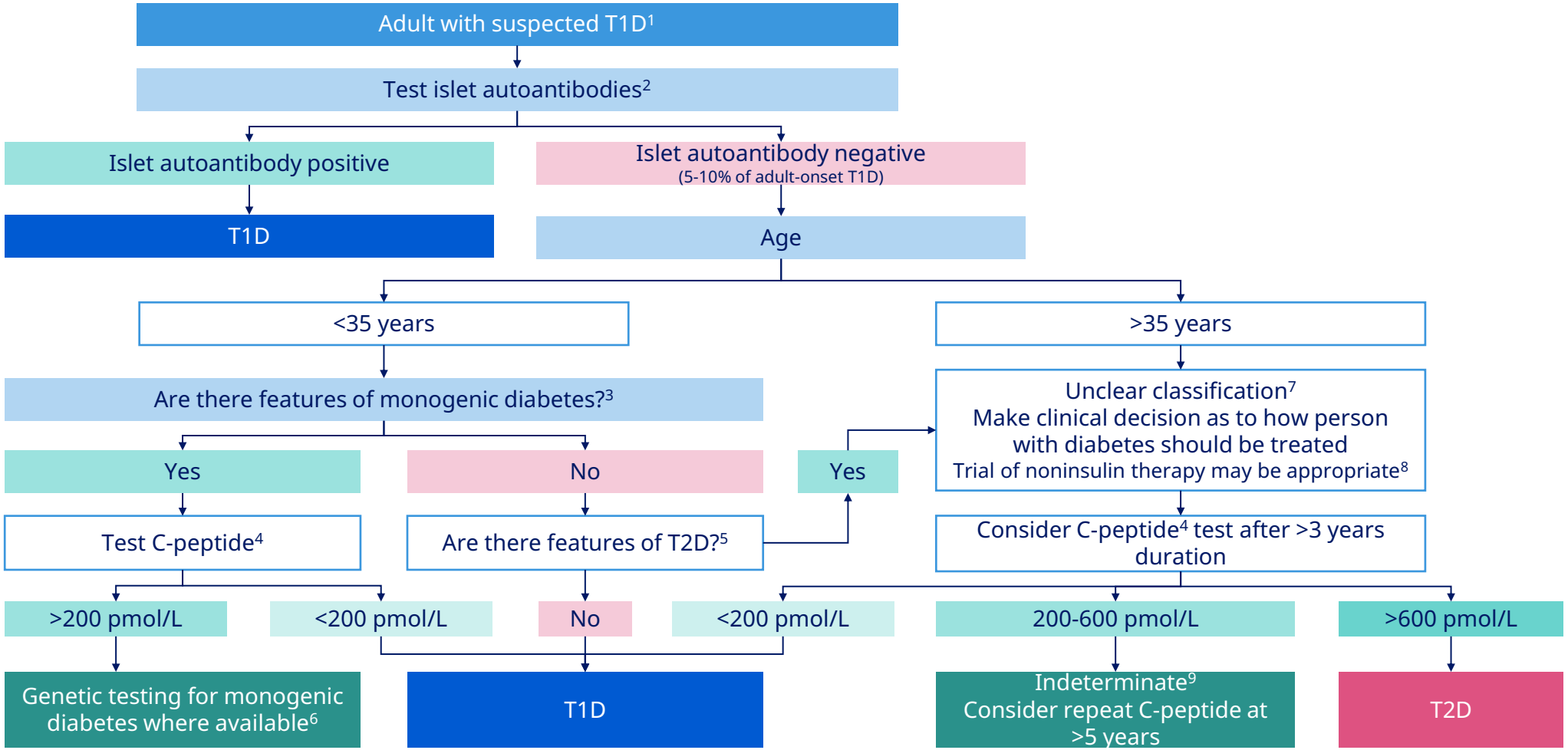


ADA - Standards of Care in Diabetes 2026

ADA, American Diabetes Association
Standards of Care in Diabetes - 2026: Diabetes Care, January 2026, Vol.49, Supplement 1

This is not an all-inclusive list. Please refer to source document for full recommendations, including level of evidence rating

Investigation of suspected T1D in newly-diagnosed adults



[1]No single clinical feature confirms T1D in isolation. [2]Glutamic acid decarboxylase (GAD) should be the primary antibody measured and, if negative, should be followed by islet tyrosine phosphatase 2 (IA-2) and/or zinc transporter 8 (ZnT8) where these tests are available. In individuals who have not been treated with insulin, antibodies against insulin may also be useful. In those diagnosed at <35 years of age who have no clinical features of T2D or monogenic diabetes, a negative result does not change the diagnosis of T1D, since 5–10% of people with T1D do not have antibodies. [3]Monogenic diabetes is suggested by the presence of one or more of the following features: A1C <58 mmol/mol (<7.5%) at diagnosis, one parent with diabetes, features of a specific monogenic cause (e.g., renal cysts, partial lipodystrophy, maternally inherited deafness, and severe insulin resistance in the absence of obesity), and monogenic diabetes prediction model probability >5% (diabetesgenes.org/exeter-diabetes-app/ModyCalculator). [4]A C-peptide test is only indicated in people receiving insulin treatment. A random sample (with concurrent glucose) within 5 h of eating can replace a formal C-peptide stimulation test in the context of classification. If the result is ≥600 pmol/L (≥1.8 ng/mL), the circumstances of testing do not matter. If the result is <600 pmol/L (<1.8 ng/mL) and the concurrent glucose is <4 mmol/L (<70 mg/dL) or the person may have been fasting, consider repeating the test. Results showing very low levels (e.g., <80 pmol/L [<0.24 ng/mL]) do not need to be repeated. Where a person is insulin treated, C-peptide must be measured prior to insulin discontinuation to exclude severe insulin deficiency. Do not test C-peptide within 2 weeks of a hyperglycemic emergency. [5]Features of T2D include increased BMI (≥25 kg/m^2), absence of weight loss, absence of ketoacidosis, and less marked hyperglycemia. Less discriminatory features include non-White ethnicity, family history, longer duration and milder severity of symptoms prior to presentation, features of the metabolic syndrome, and absence of a family history of autoimmunity. [6]If genetic testing does not confirm monogenic diabetes, the classification is unclear and a clinical decision should be made about treatment. [7]T2D should be strongly considered in older individuals. In some cases, investigation for pancreatic or other types of diabetes may be appropriate. [8]A person with possible T1D who is not treated with insulin will require careful monitoring and education so that insulin can be rapidly initiated in the event of glycaemic deterioration. [9]C-peptide values 200–600 pmol/L (0.6–1.8 ng/mL) are usually consistent with T1D or maturity-onset diabetes of the young but may occur in insulin-treated T2D, particularly in people with normal or low BMI or after long duration. Reprinted and adapted from Holt et al. Diabetes Care. 2021; 44:2589-2625; Diabetes Care, January 2026, Vol.49, Supplement 1; Diabetes Care 2026;49(Supplement_1):S27–S49, https://doi.org/10.2337/dc26-S002

Decision cycle for person-centered glycemic management in T2D

Review and agree on management plan
- Review management plan
- Mutually agree on changes
- Ensure agreed modification of therapy is implemented in a timely fashion to avoid therapeutic inertia
- Decision cycle undertaken regularly (at least once/twice a year)
- Operate in an integrated system of care

Provide ongoing monitoring and support of
- Emotional well-being
- Lifestyle and health behaviors
- Tolerability of medication
- Surrogate measures of treatment including BGM/CGM, weight, step count, HbA_{1c} blood pressure, and lipids

Implement management plan
- Ensure there is regular review: more frequent contact initially is often desirable for DSMES

Agree on management plan
Specify SMART goals:
- **S**pecific
- **M**easurable
- **A**chievable
- **R**ealistic
- **T**ime limited

Assess key person characteristics
- The individual's preferences, values, and goals
- Current lifestyle and health behaviors
- Comorbidities, i.e., CVD, CKD, HF
- Clinical characteristics, i.e., age, HbA_{1c} weight
- Mental health, cognition and functional status
- Social determinants of health

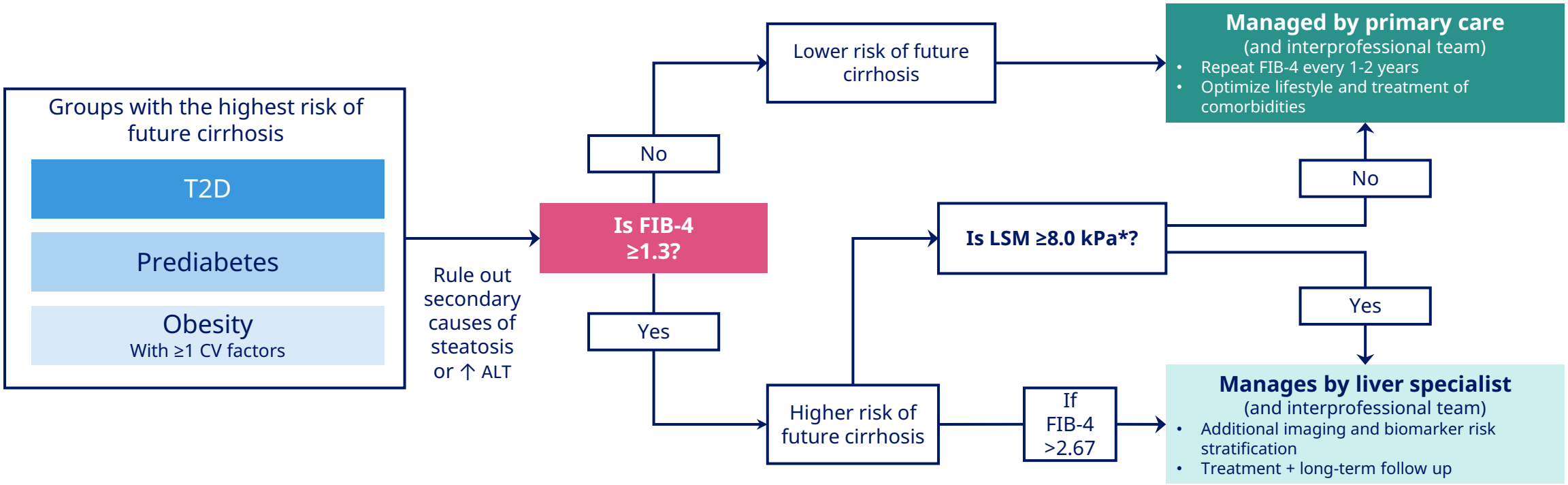
Consider specific factors that impact choice of treatment
- Individualized glycemic and weight goals
- Impact on weight, hypoglycemia and cardiovascular, kidney protection and MASLD
- Underlying physiological factors
- Side effect profiles of medications
- Complexity of treatment plan (i.e., frequency, mode of administration)
- Treatment choice to optimize medication use and reduce treatment discontinuation
- Access, cost, availability of medication(s) and lifestyle choices

Use shared decision making to create a management plan
- Ensures access to DSMES
- Involves an educated and informed person (and the individual's family/caregiver)
- Explore personal preferences
- Language matters (include person-first, strengths based, empowering language)
- Includes motivational interviewing, goal setting, and shared decision making

BGM, blood glucose monitoring; CGM, continuous glucose monitoring; CKD, Chronic Kidney Disease; CVD, cardiovascular disease; DSMES, Diabetes Self-Management Education and Support; HbA_{1c}, glycated hemoglobin; HF, Heart Failure; MASLD, metabolic dysfunction-associated steatotic liver disease; T2D, type 2 diabetes;

Diabetes Care, January 2026, Vol.49, Supplement 1; Adapted from ***Figure 4.1;*** *Diabetes Care* 2026;49(Supplement_1):S61–S88, https://doi.org/10.2337/dc26-S004

Diagnostic algorithm for risk stratification in individuals with MASLD



**In the absence of LSM, consider ELF a diagnostic alternative. If ELF ≥9.8, an individual is at high risk of metabolic dysfunction–associated steatohepatitis with advanced liver fibrosis (≥F3–F4) and should be referred to a liver specialist.*
ALT, alanine aminotransferase; CV, cardiovascular; ELF, enhanced liver fibrosis test; FIB-4, fibrosis-4 index; LSM, liver stiffness measurement, as measured by vibration-controlled transient elastography; MASLD, metabolic dysfunction-associated steatotic liver disease; T2D, type 2 diabetes

Diabetes Care, January 2026, Vol.49, Supplement 1; *Diabetes Care* 2026;49(Supplement_1):S61–S88, https://doi.org/10.2337/dc26-S004; ***Figure 4.2***

Treatment algorithm in individuals with MASLD

Individualize care, targeting the following:

- Adoption of a healthy lifestyle
- Weight loss (if indicated)
- Optimal diabetes management
- Cardiovascular risk reduction
- Need for metabolic surgery (as recommended by guidelines)

MASLD with F0-F1

Obesity pharmacotheraphy	Diabetes pharmacotheraphy	MASH pharmacotheraphy
Prefer GLP-1RA, dual GIP and GLP-1RA	**Prefer GLP-1RA, dual GIP and GLP-1RA**, pioglitazone, SGLT2i	Not indicated

MASLD with F2-F3 ("at risk" MASH)

Obesity pharmacotheraphy	Diabetes pharmacotheraphy	MASH pharmacotheraphy
Prefer GLP-1RA, dual GIP and GLP-1RA	**Prefer GLP-1RA, dual GIP and GLP-1RA**, pioglitazone	Resmetirom **GLP-1RA**†

Compensated cirrhosis

Obesity pharmacotheraphy	Diabetes pharmacotheraphy	MASH pharmacotheraphy
As with F2-F3 with caution*	As with F2-F3 with caution*	⚠ Avoid

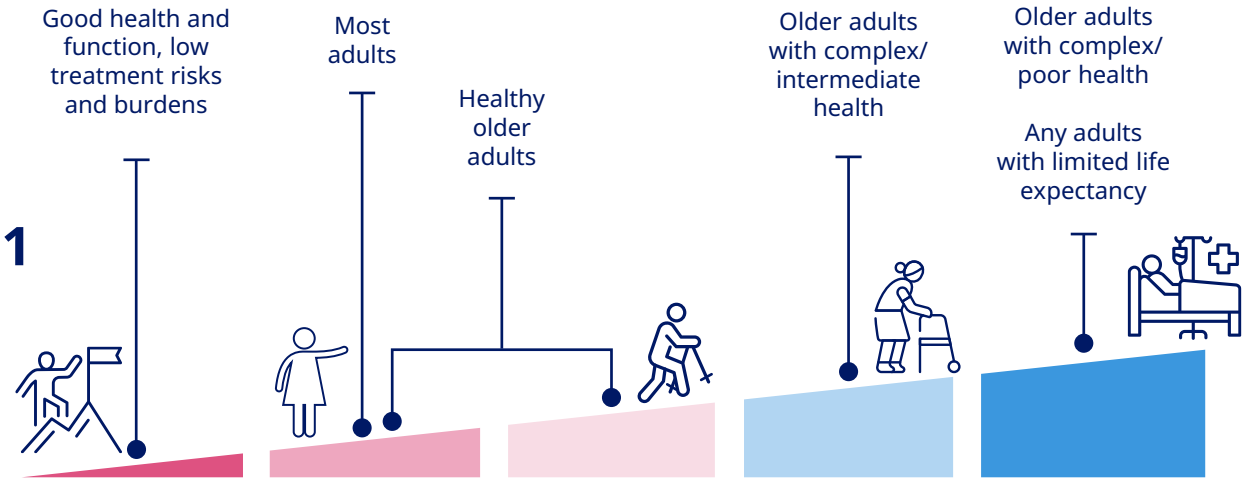
Decompensated cirrhosis

Obesity pharmacotheraphy	Diabetes pharmacotheraphy	MASH pharmacotheraphy
⚠ Avoid	Only use insulin	⚠ Avoid

**Individualized care and close monitoring needed in compensated cirrhosis given limited safety data available.*
†Only Semaglutide among GLP-1RAs has been approved by the FDA for treatment of MASH
F0-F1, no to minimal fibrosis; F2-F3, moderate fibrosis; F4, cirrhosis; GIP, glucose-dependent insulinotropic polypeptide; GLP-1RA, glucagon-like peptide-1 receptor agonist; MASH, metabolic dysfunction–associated steatohepatitis; MASLD, metabolic dysfunction-associated steatotic liver disease; SGLT2i, sodium–glucose cotransporter-2 inhibitor
Diabetes Care, January 2026, Vol.49, Supplement 1; *Diabetes Care* 2026;49(Supplement_1):S61–S88, https://doi.org/10.2337/dc26-S004;. ***Figure 4.3***

Individualized HbA_{1C} and CGM goals for nonpregnant adults

Figure 6.1



A1C goals	<6.5%	<7.0%	<7.5%	<8.0%	No A1C goal
CGM goals TIR:	**-**	**>70%**	**-**	**>50%**	**-**
• TBR <70 mg/dL	-	<4%	-	< 1%	<1%
• TBR <54 mg/dL	-	<1%	-	<1%	<1%
• TAR >180 mg/dL	-	<25%	-	<50%	Avoid symptomatic hyperglycemia
• TAR >250 mg/dL	-	<5%	-	<10%	

Favor more stringent goal	Favor less stringent goal
Short diabetes duration	Long diabetes duration
Low hypoglycemia risk	High hypoglycemia risk
Low treatment risks and burdens	High treatment risks and burdens
Pharmacotherapy with cardiovascular, kidney, weight, or other benefits	Pharmacotherapy without nonglycemic benefits
No cardiovascular complications	Established cardiovascular complications
Few or minor comorbidities	Severe, life-limiting comorbidities

CGM, continuous glucose monitoring; HbA_{1c}, glycated hemoglobin; TAR, time above range; TBR, time below range
Diabetes Care, January 2026, Vol.49, Supplement 1; Diabetes Care 2026;49(Supplement_1):S132–S149, https://doi.org/10.2337/dc26-S006; ***Figure 6.1***

Pharmacologic treatment of hyperglycemia in adults with T2D

USE OF GLUCOSE-LOWERING MEDICATIONS IN THE MANAGEMENT OF T2D

HEALTHY LIFESTYLE BEHAVIORS: DIABETES SELF-MANAGEMENT EDUCATION AND SUPPORT (DSMES); SOCIAL DETERMINANTS OF HEALTH (SDOH)

To avoid therapeutic inertia reassess and modify treatment regularly (3-6 months)

Goal: Cardiovascular and Kidney Risk Reduction in High-Risk individuals with T2D*

+ASCVD†

+Indicators of high CVD risk

+ASCVD/indicators of high CVD risk~

GLP-1RA# with proven CVD benefit

SGLT2i§ with proven CVD benefit

If glycemia is above goal

- For individuals on a GLP-1RA, consider adding SGLT2i with proven CVD benefit and vice versa
- Pioglitazone^

+HF

SGLT2i‡ with proven HF benefit in current or prior symptoms og HFrEF or HFpEF

SGLT2i and/or either dual GIP/GLP-1 RA or GLP-1 RA with proven symptomatic benefit in HFpEF and obesity

+CKD

eGFR < 60 mL/min/1.73 m² OR albuminuria (ACR ≥ 3.0 mg/mmol (30mg/g)). Repeat measurement is required to confirm CKD

+ CKD (on maximally tolerated dose of ACEi/ARB)

SGLT2i§ with primary evidence of reducing CKD progression

- SGLT2i can be started with eGFR ≥ 20 mL/min/1.73 m²
- Glucose-lowering efficacy is reduced with eGFR <45 mL/min/1.73m²

OR

GLP-1RA# with proven CKD benefit

If glycemia is above goal, for individuals on SGLT2i, consider incorporating a GLP-1RA or vice versa

If additional cardiovascular and kidney risk reduction, management of other metabolic comorbidities, and/or glycemic lowering is needed

+ mitigating risk of MASLD or MASH

Agents with proven or potential benefit in MASLD or MASH
GLP-1RA, dual GIP and GLP-1RA, pioglitazone, or combination of GLP-1RA with pioglitazone
Use insulin in the setting of decompensated cirrhosis

Goal: Achievement and Maintenance of Weight and glycemic Goals

+ Weight management

Efficacy for weight loss

Very High:
Semaglutide, Tirzepatide

High:
Dulaglutide, Liraglutide

Intermediate:
GLP-1RA (not listed above), SGLT2i

Neutral:
Metformin, DPP-4i

+ Achievement and maintenance of glycemic goals

Metformin or other agent (including combination therapy) that provides adequate efficacy to achieve and maintain glycemic treatment goals

Prioritize avoidance of hypoglycemia in high-risk individuals

Efficacy for glucose lowering

Very High:
Dulaglutide (high dose), semaglutide, tirzepatide, insulin
Combination oral, combination injectable (GLP-1RA and insulin)

High:
GLP-1RA (not listed above), metformin, pioglitazone, SGLT2i, sulfonylurea

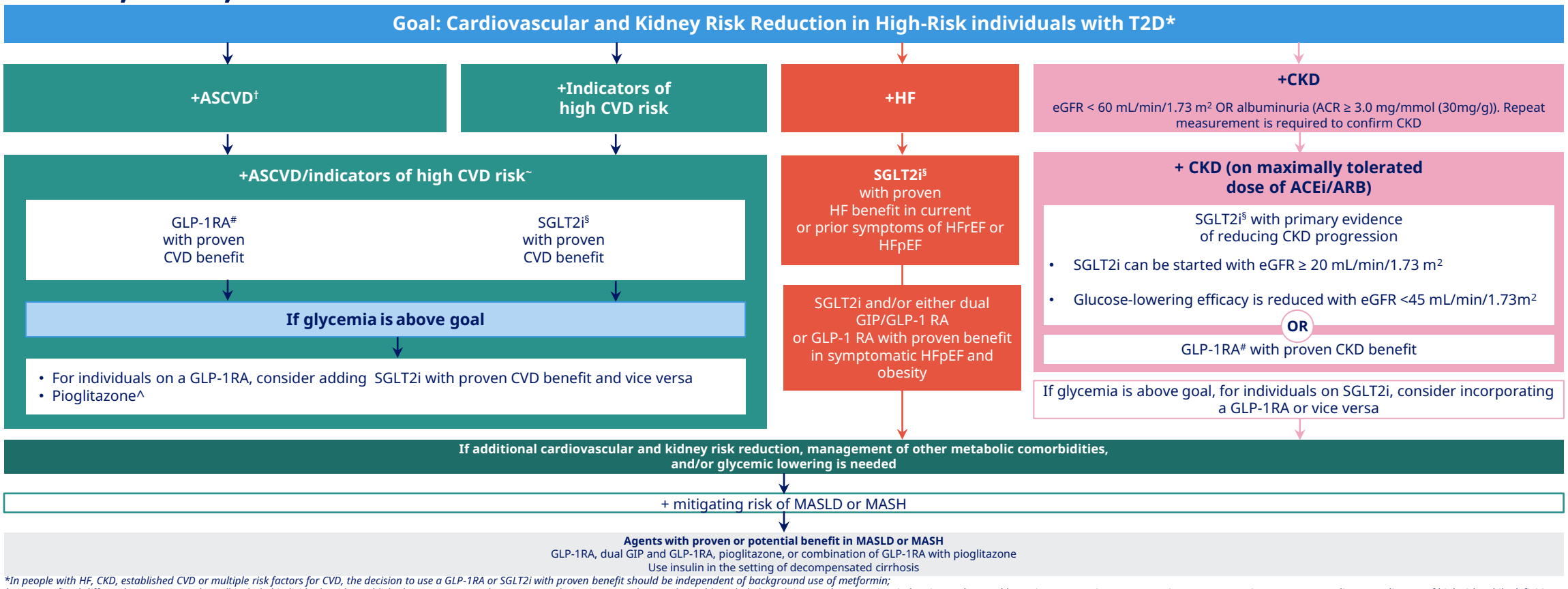
Intermediate:
DPP-4i

If glycemia is above goal or significant hypoglycemia or hyperglycemia or barriers to care are identified

- Refer to DSMES to support self-efficacy in achievement of treatment goals
- Consider technology (e.g. diagnostic or personal CGM) to identify therapeutic gaps and tailor therapy
- Identify and address SDOH that impact achievement of treatment goals

**In people with HF, CKD, established CVD or multiple risk factors for CVD, the decision to use a GLP-1RA or SGLT2i with proven benefit should be independent of background use of metformin or HbA_{1c}.*
†ASCVD: Defined differently across CVOTs but all included individuals with established CVD (e.g. MI, stroke, any revascularization procedure) and variably included conditions such as transient ischemic attack, unstable angina, amputation, symptomatic or asymptomatic coronary artery disease. Indicators of high risk: While definitions vary, most comprise ≥ 55 years of age with two or more additional risk factors (including obesity, hypertension, smoking, dyslipidemia, or albuminuria);
~ A strong recommendation is warranted for people with CVD and a weaker recommendation for those with indicators of high risk CVD. Moreover, a higher absolute risk reduction and thus lower numbers needed to treat are seen at higher levels of baseline risk and should be factored into shared decision-making process.
 # For GLP-1RA, CVOTs demonstrate their efficacy in reducing composite MACE, CV death, all-cause mortality, MI, stroke and kidney endpoints in individuals with T2D with established/high risk of CVD. One kidney outcome trial demonstrated benefit in reducing persistent eGFR reduction and CV death for a GLP-1RA in individuals with CKD and T2D;
§ For SGLT2i, CV/kidney outcomes trials demonstrate their efficacy in reducing the risk of composite MACE, CV death, all-cause mortality, MI, HHF and kidney outcomes in individuals with T2D with established/high risk of CVD;
^Low-dose pioglitazone may be better tolerated and similarly effective as higher doses.
ACEi, angiotensin-converting enzyme inhibitor; ACR, albumin/creatinine ratio; ARB, angiotensin receptor blocker; ASCVD, atherosclerotic cardiovascular disease; CGM, continuous glucose monitoring; CKD, chronic kidney disease; CV, cardiovascular; CVD, cardiovascular disease; CVOT, cardiovascular outcomes trial; DPP-4i, dipeptidyl peptidase 4 inhibitor; eGFR, estimated glomerular filtration rate; GLP-1RA, glucagon-like peptide-1 receptor agonist; HF, heart failure; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; HHF, hospitalization for heart failure; MACE, major adverse cardiovascular events; MASH, metabolic dysfunction–associated steatohepatitis; MASLD, metabolic dysfunction-associated steatotic liver disease; MI, myocardial infarction; SDOH, social determinants of health; SGLT2i, sodium-glucose cotransporter-2 inhibitor; T2D, type 2 diabetes; TZD, thiazolidinedione.
Diabetes Care, January 2026, Vol.49, Supplement 1; Diabetes Care 2026;49(Supplement_1):S183–S215, https://doi.org/10.2337/dc26-S009, ***Figure 9.4***

T2D individuals with ASCVD/indicators of high risk for CVD, HF, CKD



*In people with HF, CKD, established CVD or multiple risk factors for CVD, the decision to use a GLP-1RA or SGLT2i with proven benefit should be independent of background use of metformin;
†ASCVD: Defined differently across CVOTs but all included individuals with established CVD (e.g. MI, stroke, any revascularization procedure) and variably included conditions such as transient ischemic attack, unstable angina, amputation, symptomatic or asymptomatic coronary artery disease. Indicators of high risk: While definitions vary, most comprise ≥ 55 years of age with two or more additional risk factors (including obesity, hypertension, smoking, dyslipidemia, or albuminuria);
~ A strong recommendation is warranted for people with CVD and a weaker recommendation for those with indicators of high risk CVD. Moreover, a higher absolute risk reduction and thus lower numbers needed to treat are seen at higher levels of baseline risk and should be factored into shared decision-making process.
# For GLP-1RA, CVOTs demonstrate their efficacy in reducing composite MACE, CV death, all-cause mortality, MI, stroke and kidney endpoints in individuals with T2D with established/high risk of CVD. One kidney outcome trial demonstrated benefit in reducing persistent eGFR reduction and CV death for a GLP-1RA in individuals with CKD and T2D;
§ For SGLT2i, CV/kidney outcomes trials demonstrate their efficacy in reducing the risk of composite MACE, CV death, all-cause mortality, MI, HHF and kidney outcomes in individuals with T2D with established/high risk of CVD;
^Low-dose pioglitazone may be better tolerated and similarly effective as higher doses.
ACEi, angiotensin-converting enzyme inhibitors; ARB, Angiotensin II receptor blockers; ASCVD, atherosclerotic cardiovascular disease; CV, cardiovascular; CKD, chronic kidney disease; HbA_{1c}, glycated hemoglobin; HF, heart failure; GLP-1RAs, glucagon-like peptide-1 receptor agonists; MACE, Major adverse cardiac events; MASH, metabolic dysfunction–associated steatohepatitis; MASLD, metabolic dysfunction-associated steatotic liver disease; SGLT2i, sodium–glucose cotransporter-2 inhibitors; T2D, type 2 diabetes; TZD, thiazolidinedione. Diabetes Care, January 2026, Vol.49, Supplement 1; Diabetes Care 2026;49(Supplement_1):S183–S215, https://doi.org/10.2337/dc26-S009; **Figure 9.4**

Achievement and maintenance of weight and glycemic management goals in those without established ASCVD, CKD or HF

Goal: Achievement and Maintenance of Weight and glycemic Goals

+ Weight management

Efficacy for weight loss

Very High:
Semaglutide, Tirzepatide

High:
Dulaglutide, Liraglutide

Intermediate:
GLP-1RA (not listed above), SGLT-2i

Neutral:
Metformin, DPP-4i

+ Achievement and maintenance of glycemic goals

Metformin or other agent (including combination therapy) that provides adequate efficacy to achieve and maintain glycemic treatment goals

Prioritize avoidance of hypoglycemia in high-risk individuals

Efficacy for glucose lowering

Very High:
Dulaglutide (high dose), semaglutide, tirzepatide, insulin
Combination oral, combination injectable (GLP-1RA and insulin)

High:
GLP-1RA (not listed above), metformin, pioglitazone, SGLT2i, sulfonylurea

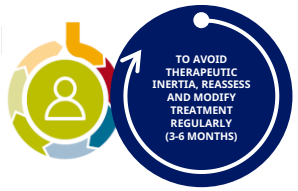
Intermediate:
DPP-4i

If HbA_{1c} is above goal or significant hypoglycemia or hyperglycemia or barriers to care are identified

- Refer to DSMES to support self-efficacy in achievement of treatment goals
- Consider technology (e.g. diagnostic or personal CGM) to identify therapeutic gaps and tailor therapy
- Identify and address SDOH that impact achievement of treatment goals

ASCVD, atherosclerotic cardiovascular disease; CV, cardiovascular; CKD, chronic kidney disease; DPP4i, Dipeptidyl peptidase-4 inhibitor; HbA1c, glycated hemoglobin; HF, heart failure; GLP-1RAs, glucagon-like peptide-1 receptor agonists; SGLT2i, sodium–glucose cotransporter-2 inhibitors; T2D, type 2 diabetes; TZD, thiazolidinedione. *Diabetes Care, January 2026, Vol.49, Supplement 1; Diabetes Care 2026;49(Supplement_1):S183–S215, https://doi.org/10.2337/dc26-S009;* ***Figure 9.4***

Algorithm for intensifying to injectable therapies (1/2)



Use Principles in **Figure 9.4** including reinforcement of behavioral interventions (weight management and physical activity) and provision of DSMES, to meet individualized treatment goals

If injectable therapy is needed to attain glycemic goals[1]

Consider GLP-1RA or dual GIP and GLP-1RA in most individuals prior to insulin[2]
INITIATION: Initiate appropriate starting dose for agent selected (varies within class)
TITRATION: Titration to maintenance dose (varies within class)

If already on GLP-1RA or dual GIP/GLP-1RA or if these are not appropriate, OR if insulin is preferred

If glycaemia is above goal

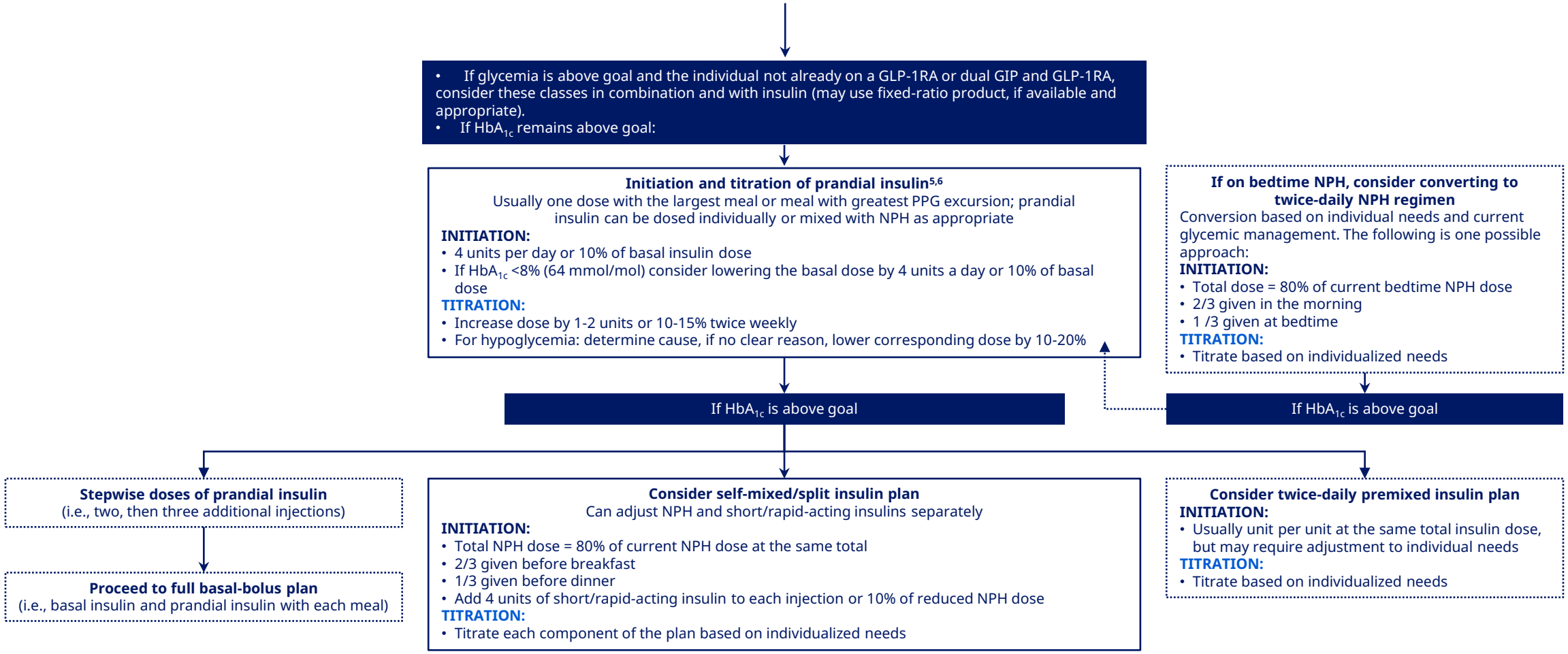
Considerations for adding basal insulin[3]
Choice of basal insulin should be based on person-specific considerations, including cost. Refer to **Table 9.4** for insulin cost information. Consider prescription of glucagon for emergent hypoglycemia.

Initiation and titration of basal analog or bedtime NPH insulin[4]
INITIATION: Start 10 units per day **OR** 0.1-0.2 units/kg per day
TITRATION:
- Set FPG goal (see Section 6: "glycemic Goals and hypoglycemia")
- Choose evidence-based titration algorithm, e.g., increase 2 units every 3 days to reach FPG goal without hypoglycemia
- For hypoglycemia: determine cause, if no clear reason, lower dose by 10-20%

Assess adequacy of insulin dose at every visit
Consider clinical signals to evaluate for overbasalization and need to consider adjunctive therapies (e.g., elevated bedtime-to-morning and/or postprandial-to-preprandial differential, hypoglycemia [aware or unaware], high glucose variability)

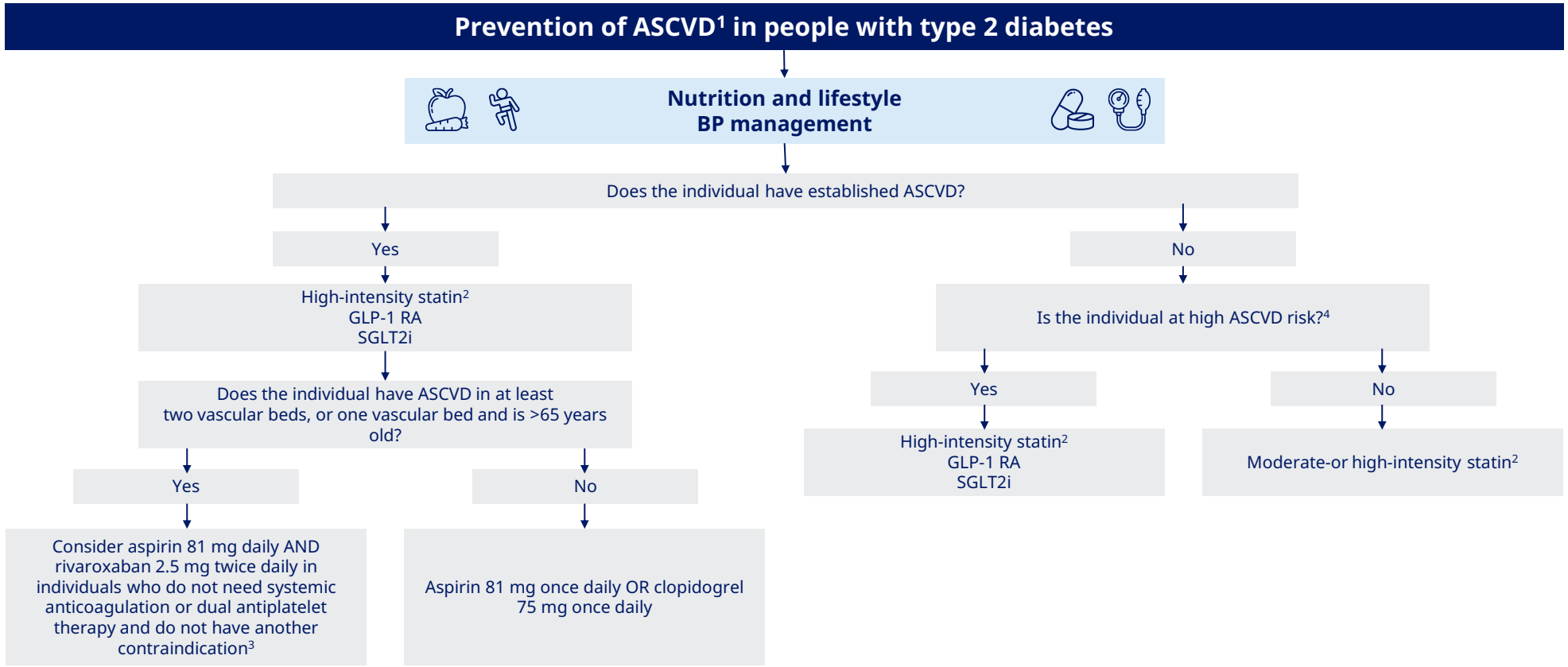
1. Consider insulin as the first injectable if symptoms of hyperglycemia are present, when HbA_{1c} levels (>10% [86 mmol/mol]) or blood glucose levels (≥300 mg/dL [≥16.7 mmol/L]) are very high, or a diagnosis of T1D is a possibility.
2. When selecting GLP-1 RAs, consider individual preference, glycemic lowering, weight-lowering effect, and frequency of injection. If CVD is present, consider GLP-1 RA with proven CVD benefit; oral or injectable GLP-1 RAs are appropriate
3. For people on GLP-1RA and basal insulin combination, consider use of a fixed-ratio combination product (iDegLira or iGlarLixi).
4. Consider switching from evening NPH to a basal analog if the patient develops hypoglycemia and/or frequently forgets to administer NPH in the evening and would be better managed with an AM dose of a long-acting basal insulin.
CVD, cardiovascular disease; DSMES, diabetes self-management education and support; FPG, fasting plasma glucose; GIP, glucose-dependent insulinotropic polypeptide; GLP-1RA, glucagon-like peptide-1 receptor agonist; HbA_{1c}, glycated hemoglobin; NPH, Neutral Protamine Hagedorn; T1D, type 1 diabetes
Diabetes Care, January 2026, Vol.49, Supplement 1; Diabetes Care 2026;49(Supplement_1):S183–S215, https://doi.org/10.2337/dc26-S009; ***Figure 9.5***

Algorithm for intensifying to injectable therapies (2/2)



5. Prandial insulin options include injectable rapid- and ultra-rapid-acting analog insulins, injectable short-acting human insulin, or inhaled human insulin.
6. If adding prandial insulin to NPH, consider initiation of a self-mixed or premixed insulin plan to decrease the number of injections required.
FPG, fasting plasma glucose; GLP-1RA, glucagon-like peptide-1 receptor agonist; GIP, glucose-dependent insulinotropic polypeptide; NPH, Neutral Protamine Hagedorn; PPG, postprandial glucose
Diabetes Care, January 2026, Vol.49, Supplement 1; Diabetes Care 2026;49(Supplement_1):S183–S215, https://doi.org/10.2337/dc26-S009; ***Figure 9.5***

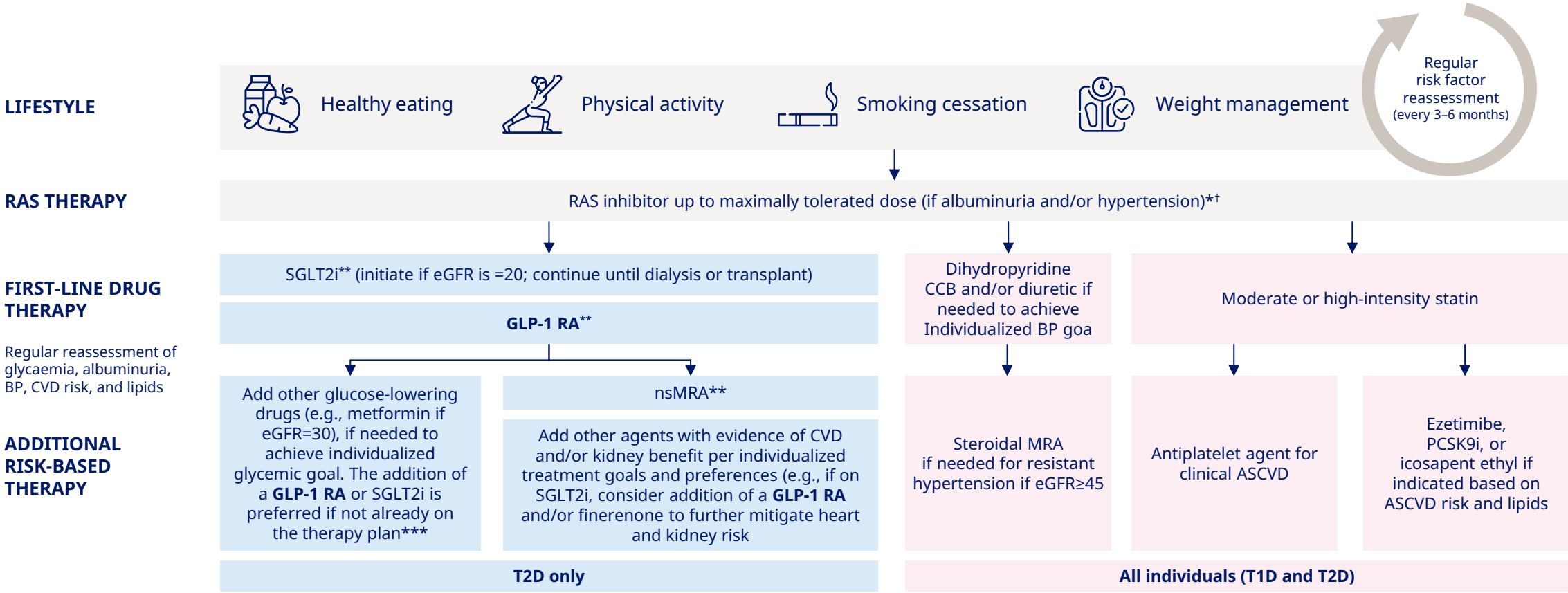
Approach to prevent ASCVD in people with type 2 diabetes



ASCVD, atherosclerotic cardiovascular disease; BP, blood pressure; CKD, chronic kidney disease; CV, cardiovascular; GLP-1 RA, glucagon-like peptide 1 receptor agonist; LVEF, left ventricle ejection fraction; SGLT2i, sodium–glucose cotransporter 2 inhibitor

Diabetes Care, January 2026, Vol.49, Supplement 1; Diabetes Care 2026;49(Supplement_1):S216–S245, https://doi.org/10.2337/dc26-S010

Holistic approach for improving outcomes in patients with diabetes and CKD



The majority of participants in SGLT2i, GLP-1 RA and nsMRA kidney outcome trials were receiving background optimised RAS inhibitor therapy. With demonstrated benefit in this population***Glucose-lowering efficacy of GLP-1 RAs is preserved at low eGFR; glucose-lowering efficacy of SGLT2i is diminished at lower eGFR. BP cuff, BP lowering; glucose meter, glucose lowering; heart, cardioprotection; kidney, kidney protection; scale, weight management. eGFR is presented in units of mL/min/1.73 m^2. †ACEi or ARB (at maximal tolerated doses) should be first-line therapy for hypertension when albuminuria is present. Otherwise, dihydropyridine calcium channel blocker or diuretic can also be considered; all three classes are often needed to attain BP targets. †Finerenone is currently the only nsMRA with proven clinical kidney and cardiovascular benefits. ACEi, angiotensin-converting enzyme inhibitor; ACR, albumin-to-creatinine ratio; ARB, angiotensin receptor blocker; ASCVD, atherosclerotic cardiovascular disease; BP, blood pressure; CCB, calcium channel blocker; CVD, cardiovascular disease; eGFR, estimated glomerular filtration rate; GLP-1 RA, glucagon-like peptide 1 receptor agonist; HTN, hypertension; MRA, mineralocorticoid receptor antagonist; nsMRA, nonsteroidal mineralocorticoid receptor antagonist; PCSK9i, proprotein convertase subtilisin/kexin type 9 inhibitor; RAS, renin-angiotensin system; SGLT2i, sodium–glucose cotransporter 2 inhibitor; T1D, type 1 diabetes; T2D, type 2 diabetes*

Diabetes Care, January 2026, Vol.49, Supplement 1; Diabetes Care 2026;49(Supplement_1):S246–S260, https://doi.org/10.2337/dc26-S011; ***Figure 11.2***

T2D individuals with ASCVD/indicators of high risk for CVD, HF, CKD

2025[1]

Goal: Cardiovascular and Kidney Risk Reduction in High-Risk individuals with T2D

+CKD

eGFR < 60 mL/min/1.73 m^2 OR albuminuria (ACR ≥ 3.0 mg/mmol (30mg/g)).
Repeat measurement is required to confirm CKD

+ CKD (on maximally tolerated dose of ACEi/ARB)

SGLT2i with primary evidence of reducing CKD progression

- SGLT2i can be started with eGFR ≥ 20 mL/min/1.73 m^2
- Continue until initiation of dialysis or transplantation
- Glucose-lowering efficacy is reduced with eGFR <45 mL/min/1.73m^2

OR

GLP-1RA# with proven CKD benefit

If HbA_{1c} is above goal, for individuals on SGLT2i, consider incorporating a GLP-1RA or vice versa

 # For GLP-1RA, CVOTs demonstrate their efficacy in reducing composite MACE, CV death, all-cause mortality, MI, stroke and kidney endpoints in individuals with T2D with established/high risk of CVD. One kidney outcome trial demonstrated benefit in reducing persistent eGFR reduction and CV death for a GLP-1RA in individuals with CKD and T2D

In the ADA SoC 2026 algorithm update, a. the decision to use a GLP-1 RA or SGLT2i with proven benefit should be made irrespective of attainment of glycemic goal and b. the suggestion to continue SGLT2i use in those with CKD on already on maximal doses of ACE/ARB till initiation of dialysis has been removed .

2026[2]

Goal: Cardiovascular and Kidney Risk Reduction*

+CKD

eGFR < 60 mL/min/1.73 m^2 OR albuminuria (ACR ≥ 3.0 mg/mmol (30mg/g)).
Repeat measurement is required to confirm CKD

+ CKD (on maximally tolerated dose of ACEi or ARB)

SGLT2i† with primary evidence of reducing CKD progression

- SGLT2i can be started with eGFR ≥ 20 mL/min/1.73 m^2
- Glucose-lowering efficacy is reduced with eGFR <45 mL/min/1.73m^2

OR

GLP-1RA# with proven CKD benefit

If glycemia is above goal, for individuals on SGLT2i, consider incorporating a GLP-1RA or vice versa

 # For GLP-1RA, CVOTs demonstrate their efficacy in reducing composite MACE, CV death, all-cause mortality, MI, stroke and kidney endpoints in individuals with T2D with established/high risk of CVD. One kidney outcome trial demonstrated benefit in reducing persistent eGFR reduction and CV death for a GLP-1RA in individuals with CKD and T2D

** In people with HF, CKD, established CVD, or multiple risk factors for CVD, the decision to use a GLP-1 RA or SGLT2i with proven benefit should be made irrespective of attainment of glycemic goal. † ASCVD: Defined differently across CVOTs but all included individuals with established CVD (e.g., MI, stroke, and arterial revascularization procedure) and variably included conditions such as transient ischemic attack, unstable angina, amputation, and symptomatic or asymptomatic coronary artery disease. Indicators of high risk: While definitions vary, most comprise ≥55 years of age with two or more additional risk factors (including obesity, hypertension, smoking, dyslipidemia, or albuminuria).*

ACEi, angiotensin-converting enzyme inhibitor; ACR, albumin/creatinine ratio; ARB, angiotensin receptor blocker; ASCVD, atherosclerotic cardiovascular disease; CKD, chronic kidney disease; CV, cardiovascular; CVD, cardiovascular disease; CVOT, cardiovascular outcomes trial; eGFR, estimated glomerular filtration rate; GLP-1RA, glucagon-like peptide-1 receptor agonist; HF, heart failure; MACE, major adverse cardiovascular events; MI, myocardial infarction; SGLT2i, sodium-glucose cotransporter-2 inhibitor; T2D, type 2 diabetes; TZD, thiazolidinedione . Diabetes Care, January 2026, Vol.49, Supplement 1; Diabetes Care 2026;49(Supplement_1):S246–S260, https://doi.org/10.2337/dc26-S011; ***Figure 9.4***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