

Supplementary Table 1. Agreement rates for individual recommendations

Recommendations		Agreement rate, %
Diabetes screening in Korean adults		
1. Target population for diabetes screening		
Recommendation 1-1.	All Korean adults aged ≥ 19 years with one or more risk factors should be screened for diabetes.	100
Recommendation 1-2.	All Korean adults aged ≥ 35 years should be screened for diabetes regardless of risk factors.	100
2. Initial screening testing		
Recommendation 2-1.	Screening for diabetes may be performed using FPG, HbA1c, or a 2-hr PG during 75-g OGTT.	100
Recommendation 2-2.	For high-risk individuals or those with prior screening suggestive of prediabetes, combining two tests (e.g., FPG+HbA1c) may be considered to improve detection.	96
3. Screening interval after a normal result		
Recommendation 3.	Individuals with normal screening results, without evidence of prediabetes or diabetes, are recommended to undergo repeat screening every 1–2 years, depending on risk factors and previous test results.	96
4. Additional testing and follow-up after a prediabetes result on FPG or HbA1c		
Recommendation 4-1.	If one screening test falls within the prediabetes range, one or more additional tests should be considered to improve diagnostic accuracy.	96
Recommendation 4-2.	A 2-hr PG during 75-g OGTT should be considered in individuals with FPG 110–125 mg/dL or HbA1c 6.1%–6.4%.	100
Recommendation 4-3.	Individuals confirmed to have prediabetes after screening are recommended to undergo repeat screening annually.	96
Pharmacological management of T2DM		
1. Person-centered determinants in pharmacological treatment decision-making for T2DM		
Recommendation 1-1.	In determining whether to initiate pharmacologic therapy and which antidiabetic agents to select in individuals with T2DM, a comprehensive assessment should include: the presence of hypercatabolic symptoms; likelihood of islet failure; current glycemic status and glycemic target; body weight; comorbidities—especially cardiovascular and renal risks; the individual's dominant pathophysiologic phenotype of diabetes; life expectancy; physical and cognitive function; personal values and treatment acceptability; and social determinants of health.	92
Recommendation 1-2.	Throughout all stages of pharmacologic treatment, lifestyle modification—including medical nutrition therapy, physical activity, smoking cessation, and psychosocial management—should be consistently integrated with diabetes self-management education and support (DSMES) to optimize clinical outcomes.	96
Recommendation 1-3.	From the initiation of pharmacologic therapy through each stage of treatment, medication adherence should be regularly assessed, and treatment should be promptly intensified when individualized glycemic targets are not achieved, in order to minimize therapeutic inertia.	100
2. Hypercatabolic state or islet failure		
2-1. Initial treatment approach of hypercatabolic state		
Recommendation 2-1-1.	In individuals presenting with hypercatabolic symptoms attributable to diabetes—such as unintended weight loss, polyuria, and polydipsia—insulin therapy should be initiated promptly, including when the current blood glucose is not markedly elevated.	96
Recommendation 2-1-2.	For individuals with mild hypercatabolic features who are clinically stable and able to take oral intake, basal insulin with or without oral antihyperglycemic agents may be initiated.	88
Recommendation 2-1-3.	For individuals requiring more aggressive glycemic control—due to marked hyperglycemia or more prominent hypercatabolic features—intensive insulin regimens (basal-plus, premixed, or basal-bolus) may be initiated.	88

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Supplementary Table 1. Continued

	Recommendations	Agreement rate, %
Recommendation 2-1-4.	In cases with severe dehydration, altered mental status, or suspected diabetic ketoacidosis (DKA) or hyperosmolar hyperglycemic state, the individual should be promptly hospitalized. Initial treatment should include intravenous insulin infusion and fluid resuscitation, followed by transition to multiple daily doses of insulin (MDI) once stabilized and oral intake is possible.	88
2-2. Treatment evaluation and adjustment in hypercatabolic state: intensification and deintensification strategies		
Recommendation 2-2-1.	After insulin initiation at hypercatabolic state, treatment response may be evaluated based on improvement in symptoms, glucose levels, and resolution of ketonuria.	84
Recommendation 2-2-2.	If basal insulin alone (\pm OADs) is insufficient to achieve glycemic targets—based on persistent hyperglycemia, inadequate symptom resolution, or ongoing ketonuria—intensification of insulin therapy should be considered. This may involve the addition of prandial insulin (e.g., basal-plus), transition to premixed insulin, or basal-bolus regimens depending on clinical needs and meal patterns.	96
Recommendation 2-2-3.	Insulin dose reduction or discontinuation may be considered once metabolic stability is achieved, hypercatabolic symptoms have resolved, and glycemic control is maintained with declining insulin requirements.	92
2.3. Use and contraindications of OADs in hypercatabolic state		
Recommendation 2-3-1.	During insulin therapy in hypercatabolic state, the concomitant use of OADs may be considered if there are no specific contraindications.	88
Recommendation 2-3-2.	SGLT2 inhibitors should be avoided in hypercatabolic states due to safety concerns, including the risk of euglycemic DKA. If SGLT2 inhibitors are indicated for comorbid conditions, initiation may be cautiously considered only after the hypercatabolic state has resolved and metabolic stability is confirmed.	96
2.4. Management of islet failure: insulin strategies and technology-enabled care		
Recommendation 2-4-1.	In individuals with established islet failure, intensive insulin therapy using MDI or insulin pump is the preferred treatment to optimize glycemic control and reduce hypoglycemia risk.	96
Recommendation 2-4-2.	Diabetes technologies—including real-time continuous glucose monitoring, sensor-augmented pumps, or automated insulin delivery systems—should be actively considered for individuals capable of safe device use.	92
Recommendation 2-4-3.	Given the complexity of insulin management in islet failure, individuals should receive structured education from trained diabetes educators. If this is not feasible, referral to specialized diabetes centers with adequate expertise and resources is recommended.	100
3. Glycemic management		
3.1. Pathophysiology- and comorbidity-guided initial selection of antidiabetic drugs		
Recommendation 3-1.	At the initiation of pharmacological treatment, glucose-lowering agents should be selected based on the individual's underlying pathophysiology of diabetes or the presence of comorbidities.	96
Recommendation 3-1-1.	In the absence of established end-organ damage comorbidities such as atherosclerotic cardiovascular disease (ASCVD), heart failure (HF), chronic kidney disease (CKD), or ischemic stroke, drugs should be selected primarily according to the person's dominant pathophysiologic phenotype (refer to subsection 3-3).	84
Recommendation 3-1-2.	In individuals with established end-organ damage comorbidities such as ASCVD, HF, CKD, or ischemic stroke, agents with proven benefits for the relevant condition(s) should be prioritized when initiating pharmacologic therapy (refer to subsection 4).	100

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Supplementary Table 1. Continued

Recommendations		Agreement rate, %
3.2. Glycemic target-based initial selection of treatment intensity and combination therapy		
Recommendation 3-2.	At the initiation of pharmacologic therapy, medications with sufficient glucose-lowering efficacy should be selected to achieve glycemic targets, considering the individual's current glycemic status and target goals.	96
Recommendation 3-2-1.	If monotherapy is unlikely to achieve the glycemic target, initial combination therapy with two or three OADs with complementary mechanisms of action should be considered.	100
Recommendation 3-2-2.	If the glycemic target is unlikely to be achieved with oral combination therapy, initiating injectable therapy—such as a GLP-1/GIP dual agonist, a GLP-1RA, or insulin—should be considered.	96
3.3. Pathophysiology-based initial selection of antidiabetic drugs for Korean patients with T2DM		
3.3.1. Predominant insulin resistance		
Recommendation 3-3-1a.	In adults with T2DM who are obese and considered to have an insulin resistance–dominant phenotype, GLP-1/GIP dual agonists, GLP-1RAs, SGLT2 inhibitors, and metformin may be preferred options for both their insulin-sensitizing and weight-reducing effects.	84
Recommendation 3-3-1b.	Although TZDs may cause modest weight gain, they effectively improve insulin resistance and can be beneficial for insulin resistance–dominant individuals. To minimize weight gain, combining TZDs with weight-reducing agents may be an effective strategy.	88
3.3.2. Predominant insulin insufficiency		
Recommendation 3-3-2a.	In adults with T2DM who are not obese and considered to have an insulin insufficiency–dominant phenotype, DPP-4 inhibitors or sulfonylureas can be considered as potential first-line oral agents.	80
Recommendation 3-3-2b.	In older adults, frail individuals, or those with lower body weight, DPP-4 inhibitors are preferred due to weight neutrality and a low risk of hypoglycemia.	84
3.4. Intensification strategies in individuals already receiving glucose-lowering medications		
3.4.1. Early intensification and combination therapy with oral agents		
Recommendation 3-4-1a.	Treatment should be promptly intensified to effectively achieve the glycemic target at an early stage, by uptitrating current medications to their maximum tolerated doses and/or by adding agents with different mechanisms of action, based on the individual's glycemic status and existing treatment regimen.	100
Recommendation 3-4-1b.	If glycemic targets are not achieved despite triple OADs, and injectable treatment is not feasible, an up-to quadruple OADs regimen may be considered in limited situations—specifically when the individual does not exhibit symptoms of hypercatabolic states, has moderately elevated hyperglycemia (e.g., HbA1c < 8.0%), and is not suspected to have significant islet failure.	100
3.4.2. Early intensification of injectable therapy, prioritizing incretin-based agents		
Recommendation 3-4-2a.	If glycemic targets are not achieved despite appropriate oral combination therapy, injectable therapies should be initiated promptly.	100
Recommendation 3-4-2b.	When selecting an injectable agent, GLP-1RAs (or GLP-1/GIP dual agonist) are preferred over basal insulin in individuals without symptoms of hypercatabolic states and with low likelihood of islet failure.	92
Recommendation 3-4-2c.	If glycemic targets are not achieved with either GLP-1RA (or GLP-1/GIP dual agonist) or basal insulin, combination therapy using both agents should be considered.	100
Recommendation 3-4-2d.	When combining GLP-1RA and basal insulin, fixed-ratio combination therapy may be considered to reduce injection frequency and improve adherence.	96

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Supplementary Table 1. Continued

Recommendations		Agreement rate, %
3.4.3. Avoiding overbasalization and transitioning to intensive insulin regimens		
Recommendation 3-4-3a.	In individuals receiving basal insulin therapy (\pm OADs) who experience frequent hypoglycemia and marked glycemic fluctuation, overbasalization should be suspected, and treatment should be intensified by reducing basal insulin and adding postprandial glucose-lowering strategies such as GLP-1RA (or GLP-1/GIP dual agonist) or insulin intensification.	92
Recommendation 3-4-3b.	If glycemic targets are not achieved despite combination therapy with GLP-1RA (or GLP-1/GIP dual agonist) and basal insulin, intensive insulin therapy—such as basal-plus, premixed, or basal-bolus regimens—should be implemented.	100
3.4.4. Optimizing combination therapy with injectables and oral agents		
Recommendation 3-4-4a.	In individuals on intensive insulin therapy, GLP-1RA (or GLP-1/GIP dual agonist) may be added to provide additional clinical benefits, including weight loss, reduced insulin requirements, and improved management of cardiovascular, renal, and metabolic dysfunction-associated steatotic liver disease (MASLD)-related risks.	100
Recommendation 3-4-4b.	In all people with T2DM receiving injectable therapy (GLP-1RA, basal insulin, or intensive insulin therapy), combination with OADs may be added to achieve additional clinical benefits, such as reduced insulin requirements, and cardiovascular and renal risk management. However, DPP-4 inhibitors should not be combined with GLP-1RA.	100
Recommendation 3-4-4c.	In individuals receiving insulin therapy, any addition, withdrawal, or dose adjustment of GLP-1RA or oral agents should prompt reassessment of insulin dosing to ensure appropriateness.	96
3.5. Deintensification strategies after optimizing glycemic control		
Recommendation 3-5-1.	In individuals who have achieved stable glycemic control within target ranges through sustained lifestyle management and continued DSMES, stepwise deintensification of pharmacologic therapy may be considered—particularly in older adults and those with renal or cognitive impairment or polypharmacy—to reduce hypoglycemia, adverse drug reactions, and treatment burden.	96
Recommendation 3-5-2.	Deintensification should be implemented gradually under close clinical monitoring, with the capacity to promptly re-intensify therapy if glycemic deterioration occurs.	96
4. Comorbidity management		
4.1. Management of cardiovascular and renal risk with end-organ damage		
4.1.1. Heart failure		
Recommendation 4-1-1a.	In adults with T2DM and HF, SGLT2 inhibitors with proven HF benefits are preferentially recommended regardless of HbA1c levels, and therapy should be continued unless contraindications or adverse effects are present.	100
Recommendation 4-1-1b.	If SGLT2 inhibitors are contraindicated or cannot be used, certain GLP-1RA (e.g., semaglutide) with demonstrated benefits in HF with preserved ejection fraction with obesity may be considered as an alternative.	92
4.1.2. Chronic kidney disease		
Recommendation 4-1-2a.	In cases of albuminuria or decreased eGFR, prioritize the use of SGLT2 inhibitors with proven renal benefits, regardless of HbA1c levels, and maintain therapy unless there are contraindications or side effects.	100
Recommendation 4-1-2b.	If SGLT2 inhibitors are contraindicated or not tolerated, GLP-1RAs with renal benefit may be considered as an alternative.	100

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Supplementary Table 1. Continued

Recommendations		Agreement rate, %
4.1.3. Atherosclerotic cardiovascular disease		
Recommendation 4-1-3.	In cases of ASCVD, prioritize the use of GLP-1RAs or SGLT2 inhibitors with proven cardiovascular benefits.	96
4.1.4. Ischemic stroke		
Recommendation 4-1-4.	In adults with T2DM and a history of ischemic stroke or transient ischemic attack, GLP-1RA or TZDs may be considered to reduce the risk of recurrent stroke.	96
4.3. Management of metabolic comorbidity without end-organ damage		
4.3.1. Morbid obesity		
Recommendation 4-2-1.	In adults with T2DM and comorbid obesity, anti-obesity pharmacotherapy—such as GLP-1/GIP dual agonist (e.g., tirzepatide) or GLP-1RAs (e.g., semaglutide and liraglutide)—should be considered, regardless of glycemic status, when lifestyle intervention alone is insufficient.	96
4.3.2. Metabolic dysfunction-associated steatotic liver disease		
Recommendation 4-2-2a.	In adults with T2DM and MASLD, GLP-1/GIP dual agonist (e.g., tirzepatide) or GLP-1RAs (e.g., semaglutide and liraglutide) may be considered to improve hepatic steatosis, and cardiometabolic risk factors, regardless of glycemic status.	84
Recommendation 4-2-2b.	In cases where GLP-1/GIP dual agonist or GLP-1RA are not tolerated, contraindicated, or unavailable, TZDs may be considered as an alternative option for improving insulin sensitivity and hepatic steatosis.	92
Severe diabetes mellitus, not yet established but needs to be clarified		
Recommendation 1.	In determining whether to initiate pharmacologic therapy and which antidiabetic agents to select in individuals with severe T2DM, a comprehensive assessment should be made of the following: a high complication stage, indicating substantial target organ damage; a high metabolic grade, reflecting severe insulin deficiency, marked insulin resistance, and uncontrolled or highly variable glycemia; or both.	80

FPG, fasting plasma glucose; HbA1c, glycosylated hemoglobin; 2-hr PG, 2-hour plasma glucose; OGTT, oral glucose tolerance test; T2DM, type 2 diabetes mellitus; OAD, oral antihyperglycemic drug; SGLT2, sodium-glucose cotransporter 2; GLP-1, glucagon-like peptide-1; GIP, glucose-dependent insulinotropic polypeptide; GLP-1RA, glucagon-like peptide-1 receptor agonist; TZD, thiazolidinedione; DPP-4, dipeptidyl peptidase-4; eGFR, estimated glomerular filtration rate.