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Cardiovascular Disease Management Consensus in Kidney Transplantation Recipients

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Background: Kidney transplantation is the definitive treatment for end-stage kidney disease; however, organ availability in Taiwan remains limited, with only 4.9% of patients on the waiting list receiving transplants in 2019. More than 86,000 individuals require regular dialysis, highlighting the need for improved transplantation rates. Although the median survival time post-transplantation is 5.7 years, nearly half of Taiwanese transplant recipients died in 2019, emphasizing cardiovascular disease as a major concern. Notably, patients who survive for more than a decade post-transplantation are increasingly prone to cardiovascular disease, diabetes, and hypertension. The aim of this study was to develop a joint consensus with the Taiwan Society of Nephrology, Taiwan Society of

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Cardiology, and Transplantation Society of Taiwan, to outline the best practices for evaluating and managing cardiovascular disease in kidney transplant recipients.

Methods: A consensus committee of 24 Taiwanese nephrologists, cardiologists, and surgeons synthesized recommendations from the literature and international guidelines, finalizing them via a formal voting process (70%/90% agreement).

Results: Clinical recommendations emphasize pre-transplant cardiac assessment, including echocardiography and perfusion imaging; vigilant post-transplant surveillance of blood pressure, lipids, and glycemic control; and the judicious use of immunosuppressants to minimize metabolic complications. Multidisciplinary collaboration between nephrologists, cardiologists, and transplant experts is crucial for delivering patient-centered care.

Conclusions: By adopting these guidelines, clinicians can promote long-term graft survival, reduce cardiovascular complications, and improve patient outcomes in the evolving transplantation landscape in Taiwan.

Key Words: Cardiovascular disease management • Kidney transplantation • Multidisciplinary collaboration

Abbreviations

ABPM	Ambulatory blood pressure monitoring
ACEIs	Angiotensin-converting enzyme inhibitors
AF	Atrial fibrillation
AMI	Acute myocardial infarction
ARBs	Angiotensin receptor blockers
ARNIs	Angiotensin receptor-neprilysin inhibitors
BP	Blood pressure
CABG	Coronary artery bypass grafting
CAD	Coronary artery disease
CCBs	Calcium channel blockers
CKD	Chronic kidney disease
CKD-MBD	Chronic kidney disease-mineral and bone disorders
CNIs	Calcineurin inhibitors
CV	Cardiovascular
CVD	Cardiovascular disease
DAPT	Dual antiplatelet therapy
DHP	Dihydropyridine
DM	Diabetes mellitus
ECG	Electrocardiography
eGFR	Estimated glomerular filtration rate
ESKD	End-stage kidney disease
HbA1c	Glycated hemoglobin
HD	Hemodialysis
HF	Heart failure
KDIGO	Kidney Disease: Improving Global Outcomes
KTRs	Kidney transplant recipients
KTx	Kidney transplantation
LVEF	Left ventricular ejection fraction
MRAs	Mineralocorticoid receptor antagonists
NOACs	Novel oral anticoagulants
NODAT	New-onset diabetes after transplantation
NSTEMI	Non-ST-segment elevation myocardial infarction
OBP	Office blood pressure
PCI	Percutaneous coronary intervention
PTDM	Post-transplantation DM
QD	Once daily
SGLT2	Sodium-glucose cotransporter-2
STEMI	ST-segment elevation myocardial infarction
TSN	Taiwan Society of Nephrology
TSOC	Taiwan Society of Cardiology
TST	Transplantation Society of Taiwan

INTRODUCTION

Hemodialysis (HD) is usually used as a management strategy in clinical practice to prolong survival and maintain the quality of life for patients with end-stage kidney disease (ESKD). Kidney transplantation (KTx) relies on the availability of a suitable organ source. Statistics from the latest Annual Report on Kidney Disease in Taiwan indicate that the number of individuals in Taiwan requiring regular HD exceeds 86,000. However, the organ transplantation rate in 2019 was only 4.9%; that is, for every 100 patients waiting for transplantation, only approximately five received a transplant.[1] Thus, the number of patients undergoing dialysis awaiting transplantation has risen steadily, but the number of individuals who undergo transplantation remains far lower than those awaiting transplantation. Substantial improvements in KTx rates are needed in Taiwan.

A systematic review published by Canadian researchers in 2011 compared the effects of HD and KTx on patient outcomes. The results indicated that patients who underwent HD had significant short-term clinical benefits, and that KTx better influenced the overall survival of patients. In addition, the kidney transplant recipients (KTRs) also had a lower incidence of subsequent cardiovascular events, such as myocardial infarction, stroke, and heart failure (HF), and were able to maintain a better quality of life.[2] Similar results were reported in a study of Korean patients published in 2016, which showed that KTRs had a significantly lower risk of cardiovascular-related death than patients undergoing HD.[3] Therefore,

timely KTx can lead to better long-term survival and outcomes than HD in patients with ESKD.

The trends of KTx in Taiwan were reported in the 2020 Annual Report on Kidney Disease, in which more than 30% of the KTRs were relatively young (20-44 years). Data on long-term follow-up of KTRs in the Annual Report revealed that the median survival time after transplantation from 2000 to 2019 was 5.7 years, with up to 49.5% of KTRs dying in 2019.

The percentage of KTRs who survived > 10 years after transplantation, especially those who lived beyond 65 years of age, increased from 2.2% in 2010 to 33.1% in 2018 ($p < 0.0001$).[4] This demonstrates the exceptional effectiveness of KTx in Taiwan. However, with prolonged survival times, the long-term survivors experienced increased rates of cardiovascular disease (CVD) ($p = 0.00071$), diabetes mellitus (DM) ($p = 0.00168$), and hypertension ($p = 0.20016$) (Table 1). Therefore, the comprehensive consideration of methods of management and care for CVD in KTRs has become a key issue for clinicians, and is the purpose of this joint consensus by the Taiwan Society of Nephrology (TSN), Taiwan Society of Cardiology (TSOC), and Transplantation Society of Taiwan (TST). A consensus best suited for CVD care in KTRs in the healthcare environment of Taiwan can be developed through the knowledge and experience of multidisciplinary experts.[4] However, such a consensus needs to be continually updated and improved. This study reports on a recent version of this consensus in Taiwan. The findings of this study may help develop strategies to improve the healthcare of KTRs with and without CVD.

METHODS

To establish a consensus for CVD Management in KTRs in Taiwan, the TSN, TSOC, and TST invited 24 experienced experts, including 12 nephrologists, 7 cardiologists, and 5 transplant surgeons from various regions of Taiwan to form a consensus committee. The recommendations presented in this consensus document were synthesized based on a comprehensive review of international guidelines and published literature, specifically considering the healthcare infrastructure and clinical practice patterns unique to Taiwan. All content underwent rigorous scrutiny and received approval from all

Table 1. Analysis of comorbidities of KTRs

Disease (%)	2013	2014	2015	2016	2017	2018	2019	2020	2021	p value for trend*
Cardiovascular disease	22.8	21.3	20.3	25.3	26.1	31.4	27.2	33.2	36.1	0.00071
Ischemic heart disease	7.7	8.2	8.4	14.2	11.5	11.1	11.4	17.0	14.9	0.00708
Heart failure	18.0	15.5	14.0	14.2	18.2	25.5	19.5	23.0	25.6	0.01754
Arrhythmia	1.6	1.8	4.6	2.1	3.6	4.9	4.0	2.5	5.7	0.07659
Atrial fibrillation	1.0	0.3	1.1	1.0	1.2	1.9	0.7	0.6	3.2	0.15436
Stroke	2.3	3.3	1.8	2.4	2.1	3.4	3.2	3.6	3.5	0.06863
Hemorrhagic	0.3	0.6	0.4	0.4	0.3	0.6	0.3	0.6	0.6	0.30671
Ischemia	1.3	1.5	0.0	0.7	1.2	1.5	1.7	2.5	2.2	0.05295
Diabetes mellitus	8.4	8.5	9.8	10.7	12.7	12.3	16.1	14.0	13.3	0.00168
COPD	4.8	2.7	1.4	2.8	2.7	2.8	4.2	2.5	3.5	0.98138
Peptic ulcer	10.9	8.8	6.6	15.6	9.7	10.5	7.9	9.0	5.7	0.34136
Malignancy	1.9	6.1	2.8	3.5	6.7	2.5	4.9	3.6	2.2	0.83416
Hypertension	62.7	61.4	54.9	58.1	60.9	68.3	61.5	64.4	64.6	0.20016
Dyslipidemia	17.7	19.2	21.3	21.1	20.0	16.6	20.3	21.1	16.5	0.73355
Polycystic kidney disease	1.6	3.0	1.4	2.4	2.7	3.7	2.5	4.7	4.4	0.01164
PAD	1.0	0.9	0.7	2.1	0.9	2.8	2.5	1.1	1.9	0.17789

According to diagnostic code (ICD), which includes inpatient (once at least) and outpatient/emergency room (twice at least) status one year before renal transplantation.

* Linear regression model.

COPD, chronic obstructive pulmonary disease; ICD, International Classification of Diseases; KTRs, kidney transplant recipients; PAD, peripheral artery disease.

consensus members before inclusion in the final manuscript. This content was reviewed and endorsed by experts during consensus meetings held on October 28th, 2023 and June 22nd, 2024. The recommended statements were approved if they received agreement from more than 70% of the committee members and were strongly recommended when at least 90% agreement was reached. Table 2 lists the key consensus statements and voting results.

TOPIC DISCUSSION

Cardiovascular evaluation for recipient before renal transplantation

1. It is necessary to perform echocardiography to evaluate cardiac function before KTx (100% agreement, strongly recommended).
2. Nuclear stress myocardial perfusion imaging can be used to evaluate myocardial ischemia and left ventri-

Table 2. Key consensus statements for cardiovascular disease management for kidney transplant recipients

Consensus statement	Percent agreement
01. It is necessary to perform echocardiography to evaluate the cardiac function before kidney transplant (KTx).	100%
02. The adoption of nuclear myocardial perfusion imaging for further evaluation of cardiac function before KTx can be considered.	86%
03. Computed tomography scan assessment of the iliac arteries before KTx is recommended to ensure normal lower limb circulation in the patient postoperatively.	71%
04. Discontinuation of steroids reduces the risk of occurrence of new-onset diabetes after transplantation (NODAT), hypertension, and hyperlipidemia. However, early discontinuation may introduce the risk of transplant rejection and thus requires a case-by-case evaluation for each patient.	100%
05. Calcineurin inhibitors (CNIs) are closely associated with post-transplantation diabetes mellitus (PTDM), hypertension, and hyperlipidemia. No evidence shows that discontinuing or reducing CNI use reduces the occurrence of major cardiovascular diseases. Therefore, a case-by-case evaluation is required for each patient.	91%
06. Plasma lipid control in kidney transplant recipients (KTRs) must follow guidelines currently recommended by cardiology, atherosclerosis, and vascular societies.	93%
07. Statins are the first-line drug of choice for KTRs with hypercholesterolemia that exceeds guideline recommendations.	100%
08. The blood pressure control targets for adult KTRs are systolic blood pressure < 130 mmHg and diastolic blood pressure < 80 mmHg. Blood pressure measurements should be mainly performed by standard clinic assessments but can be substituted by 24-hour ambulatory or home monitoring.	100%
09. Dihydropyridine (DHP) calcium channel blockers (CCBs) or angiotensin receptor blockers (ARBs) are the first-choice drugs for blood pressure control in adult KTRs. However, it is still recommended that adjustments be made based on individual patient conditions, with the achievement of blood pressure targets prioritized over medication choice.	100%
10. The hemoglobin A1c (HbA1c) target of KTRs should be controlled within 6.5-7.0% to reduce the risk of cardiovascular disease.	94%
11. Sodium-glucose cotransporter-2 inhibitors (SGLT2Is) are the recommended first-line drugs for treating PTDM in KTRs.	25%
12. After KTx, changes in left ventricular ejection fraction (LVEF) should be continuously monitored, and medications for heart failure should be adjusted based on individual patient conditions.	100%
13. The principles of using angiotensin-converting enzyme inhibitors (ACEIs)/ARBs/angiotensin receptor-neprilysin inhibitors (ARNIs), mineralocorticoid receptor antagonists (MRAs), and SGLT2Is for treating first-onset heart failure in KTRs are identical to the treatment principles for heart failure in the general population.	62%
14. Screening for atrial fibrillation [such as regular outpatient electrocardiography (ECG), long-term continuous ECG recording] is recommended for patients who are going to receive KTx and those who have received a transplant.	94%
15. Regarding the combined use of novel oral anticoagulants (NOACs) with immunosuppressants in KTRs with atrial fibrillation, consultation with a cardiologist for drug selection and dose adjustment is recommended, and other stroke prevention methods should be considered.	100%
16. The combination of aspirin and clopidogrel is the first-choice regimen for KTRs who require dual antiplatelet therapies.	93%
17. For KTRs with multivessel coronary artery disease, coronary artery bypass grafting (CABG) and percutaneous coronary intervention (PCI) are good choices for revascularization.	91%

cular systolic function before KTx (86% agreement; recommended).

3. Computed tomography of the iliac arteries before KTx is recommended to ensure lower limb circulation (71% agreement, recommended).

Pre-transplantation evaluations for CVD include donor and recipient evaluations. Donor evaluations can be further subdivided into deceased and living donors. It is well known that the vascular status is vital for transplantation surgery. For deceased donors, obtaining detailed premortem cardiovascular data may be considerably difficult. For living donors, evaluations primarily focus on confirming renal vessel status, including the number of renal arteries, venous drainage, and the presence of small polar vessels.[5] It is recommended that priority should be placed on treadmill or stress testing and confirmation of the history of CVD in living donors.

For recipient evaluations, both the 2020 Kidney Disease: Improving Global Outcomes (KDIGO) guidelines and the 2015 European Renal Best Practice guidelines recommend evaluating the presence or absence of certain common CVDs with high prevalence rates, including coronary artery disease (CAD), valvular disease, arrhythmia, HF, and peripheral arterial occlusion.[6-8] Noninvasive modalities such as treadmill and cardiac imaging are primarily recommended for the preoperative examination of transplant recipients. Cardiac imaging is particularly vital because the left ventricular ejection fraction (LVEF) reflects whether the recipient's cardiac output can adapt to the increased demand after transplantation. Echocardiography or stress myocardial perfusion imaging can be used to obtain objective and comprehensive data for evaluating cardiac physiology. Regarding the preoperative biochemical markers for transplant recipients, even in the absence of CAD, patients with chronic kidney disease (CKD) have higher levels of common cardiac biochemical markers such as creatinine kinase, creatine kinase-myocardial band, cardiac troponins, and B-type natriuretic peptide/N-terminal pro B-type natriuretic peptide compared with the general population.[9] Therefore, one-time cardiac biomarker measurement is not advisable before renal transplantation but may be suitable for long-term monitoring of transplant recipients. Renal vessel status is a critical preoperative evaluation parameter. A history of DM, hyperphosphatemia, smoking, or surgery-induced vascular injury increases the risk of vascular complications during transplantation.

Although pre-operative cardiovascular evaluation has become a standard procedure, limited evidence supports its use in enhancing transplantation success or post-transplantation survival rates. Therefore, noninvasive modalities rather than invasive testing are advisable.[10] Besides established CVDs, previous research has reported associations between older age, DM, hyperlipidemia, hypertension, a history of smoking, hyperphosphatemia, a history of long-term dialysis, a family history of CVD, and LVEF < 40% with a higher risk of asymptomatic CVD.[6,11]

Risk factors for CVDs in KTRs

1. Discontinuation of steroids reduces the risk of new-onset diabetes after transplantation (NODAT), hypertension, and hyperlipidemia. However, early discontinuation may introduce the risk of transplant rejection, and thus requires case-by-case evaluation for each patient (100% agreement, strongly recommended).
2. Calcineurin inhibitors (CNIs) are closely associated with post-transplantation DM (PTDM), hypertension, and hyperlipidemia. No evidence has shown that discontinuing or reducing CNI treatment decreases the occurrence of major CVDs. Therefore, case-by-case evaluation is required for each patient (91% agreement, strongly recommended).

Established CVD is a key factor in post-transplantation mortality among KTRs, regardless of whether death occurs shortly after or during long-term follow-up. It is the primary cause of mortality in KTRs within three months after KTx. The results of a long-term postoperative follow-up study suggested that CVD and infection were the most common causes of death.[12] The risk of death from CVD in patients who had progressed to ESKD or commenced dialysis was 10-15 times that of the general population. KTx considerably decreased the risk of death from CVD, although it remained higher than that of the general population.[13] Another study compared the cumulative risk for the occurrence of acute myocardial infarction (AMI) between patients on the transplantation waiting list. Overall, patients who underwent transplantation had a 17% lower adjusted risk for AMI (0.83; 95% confidence interval 0.77 to 0.90) compared to patients remaining on the waiting list. However, when examined by time period, transplant recipients had a higher AMI risk than waiting list patients during the first three months after transplantation, after

which their AMI risk decreased substantially and became lower than that of waiting list patients.[14]

The risk factors for CVD differ among patients with different degrees of kidney disease. The risk factors for early kidney disease are similar to those observed in the general population with CVD. The risk factors for middle-stage to advanced kidney disease include hypertension, proteinuria, hyperhomocysteinemia, duration of HD, anemia, lack of physical activity, and chronic kidney disease-mineral and bone disorders (CKD-MBDs). Post-transplantation risk factors include delayed graft function, acute rejection, immunosuppressants, NODAT, and post-transplantation lymphoproliferative disorders.[15] The post-transplantation cardiovascular (CV) risk factors are particularly complex. Besides the 3Hs (hypertension, hyperglycemia, and hyperlipidemia) and smoking history, such patients face CV risks caused by kidney diseases such as CKD-MBDs in the event of post-transplantation graft dysfunction.[15] Although kidney function improvement after transplantation can reduce the CV risk, it can introduce other non-traditional CV risks. Therefore, controlling post-transplantation CV risk factors and diseases is vital for KTRs.

Transplant recipients require long-term immunosuppressants, which can have metabolic effects and increase the risk of CVDs. For example, prednisolone, cyclosporine, and tacrolimus increase the risk of hypertension, hyperlipidemia, insulin resistance, and glucose intolerance, whereas mycophenolic acid and the mammalian target of rapamycin inhibitors, sirolimus and everolimus, increase the risk of hyperlipidemia. However, the inadequate use of immunosuppressants may increase the likelihood of rejection, which may cause considerable damage to various organ functions. Therefore, the optimization of immunosuppressants, use of immunosuppressants most suitable for patients (such as cyclosporine and tacrolimus), and attempts to reduce or discontinue steroids as much as possible may reduce medication-induced CV events. A previous study compared the differences in CV events and mortality between *de novo* KTRs who received everolimus combined with a reduced dose of standard calcineurin inhibitor and those who received mycophenolic acid combined with a standard dose of standard calcineurin inhibitor, and the results showed no statistically significant differences between the two groups. This demonstrates the viability of different immunosuppressant combinations in the care of KTRs.[16] A previous meta-analysis of published studies found that steroid avoidance or withdrawal in KTRs increased the risk of acute rejection compared to normal steroid use; however, differences in graft survival were not statistically significant, and the risk of subsequent CV events was significantly lower in the groups with steroid avoidance and withdrawal.[17]

Lipid control in KTRs

1. Lipid control in KTRs should follow international or national guidelines recommended by professional medical societies (93% agreement, strongly recommended).
2. Statins are the first-line drugs of choice for KTRs with hypercholesterolemia (100% agreement, strongly recommended).

CV-related mortality is a major cause of post-transplantation deaths. Long-term observations have revealed that CV-related mortality is comparable to that attributed to infection, which has traditionally been of greater concern.[18] In addition, KTRs require long-term use of immunosuppressants, which can result in metabolic alterations and lead to an increased risk of CVD (Table 3).[19] The long-term risks of CVD and mortality for KTRs can be assessed using the tool developed in 2012 by researchers at Uppsala University, Sweden. The tool enables the prediction of the risk of major adverse cardiac events in patients within seven years after transplantation based on the presence or absence of DM, low-density lipoprotein level, habit or history of smoking, presence or absence of CAD, number of transplants, creatinine level, and age.[19]

According to the classification method stated in the 2013 KDIGO Clinical Practice Guidelines for Lipid Management in Chronic Kidney Disease, the targets for hyperlipidemia control in KTRs are identical to those adopted for patients with CKD. However, the most significant difference in hyperlipidemia control between transplant recipients and patients with CKD is that interactions exist between immunosuppressants and statins or fibrate-type hypolipidemic agents (Table 4).[19] The combined use of these hypolipidemic agents with cyclosporine or tacrolimus affects statin metabolism and increases the risk of myopathy. Given the drug-drug interactions of statins with cyclosporine and tacrolimus, low-potency statins such as fluvastatin 20 mg once daily (QD) or pravastatin 20 mg QD are used in patients who use the two aforementioned

Table 3. Influence of immunosuppressants on the cardiovascular risk factors of kidney transplant recipients[18]

Immunosuppressants	CV risk factors exacerbated
Corticosteroids	
Prednisone	Hyperglycemia (+++), Arterial hypertension (++), Triglycerides (++), LDL (++).
Deflazacort	Hyperglycemia (+), Arterial hypertension (++), Triglycerides (+), LDL (+).
Calcineurin inhibitors	
Cyclosporine	Hyperglycemia (+), Arterial hypertension (+++), Triglycerides (++), LDL (+++).
Tacrolimus	Hyperglycemia (+), Arterial hypertension (+++), Triglycerides (+), LDL (++).
mTOR inhibitors	
Sirolimus	Triglycerides (+++), LDL (+++).
Everolimus	Triglycerides (+++), LDL (+++).
Antiproliferative agents	
Azathioprine	No significant increase
Mycophenolate mofetil	No significant increase

CV, cardiovascular; LDL, low-density lipoprotein; mTOR, mammalian target of rapamycin.
(+), mild increase; (++), moderate increase; (+++), severe increase.
The magnitude of this effect was arbitrarily estimated on the basis of the evidence discussed in this review.

types of immunosuppressants. If the lipid goal is not achieved, a switch to medium-potency atorvastatin 10 mg QD, pravastatin 40 mg QD, or rosuvastatin 5 mg QD should be considered. For patients who do not use these two types of immunosuppressants, prioritization of the use of medium-potency atorvastatin 10 mg QD or rosuvastatin 5 mg QD can be considered; the dose can be titrated to the maximally tolerated dose if the lipid target is not attained (Figure 1).[19] The addition of a non-statin such as ezetimibe or proprotein convertase subtilisin kexin type 9 can be considered. Patients using the aforementioned hypolipidemic agents should undergo regular blood tests to monitor for myopathy-related side effects.

Blood pressure control in KTRs

1. The blood pressure (BP) control targets for adult KTRs are systolic BP < 130 mmHg and diastolic BP < 80 mmHg. BP measurements should be mainly performed using standard clinical assessments, but can be substituted by 24-hour ambulatory or home monitoring (100% agreement, strongly recommended).
2. Dihydropyridine (DHP) calcium channel blockers (CCBs) and angiotensin receptor blockers (ARBs) are the first-choice drugs for BP control in adult KTRs. However, they can be adjusted individually, with the achievement of BP targets prioritized over medication choice (100% agreement, strongly recommended).

Table 4. Drug-drug interactions between hypolipidemic agents and immunosuppressants[18]

Drugs	Dangerous interaction	Benefit
Statins		
Atorvastatin	Co-administered with CsA or Tac increase statin exposure and risk for myopathy.	LDL and Tg reduction CVD mortality reduction Plaque stabilization
Lovastatin	Co-administered with CsA or Tac increase statin exposure and risk for myopathy.	
Simvastatin	Co-administered with CsA or Tac increase statin exposure and risk for myopathy.	
Rosuvastatin	Co-administered with CsA or Tac increase statin exposure and risk for myopathy.	
Fibrates		
Gemfibrozil	Can reduce plasma levels of CsA and mTORi. Increased risk for myopathy in co-administration with statins.	LDL reduction HDL increase
Fenofibrate	Can reduce plasma levels of CsA and mTORi. Increased risk for myopathy in co-administration with statins.	
Ezetimibe	Co-administered with CsA can increase ezetimibe and CsA levels and risk for side effects.	LDL reduction
Bile acid sequestrants	Can reduce MMF, CsA, Tac and mTORi levels, their administration should be delayed by 4 h from bile acid sequestrants.	LDL reduction
Lomitapide	Can increase plasma levels of CsA, Tac and mTORi.	LDL reduction

CsA, cyclosporine; HDL, high-density lipoprotein; LDL, low-density lipoprotein; MMF, mycophenolate mofetil; mTORi, mammalian target of rapamycin inhibitors; Tac, tacrolimus; Tg, triglycerides.

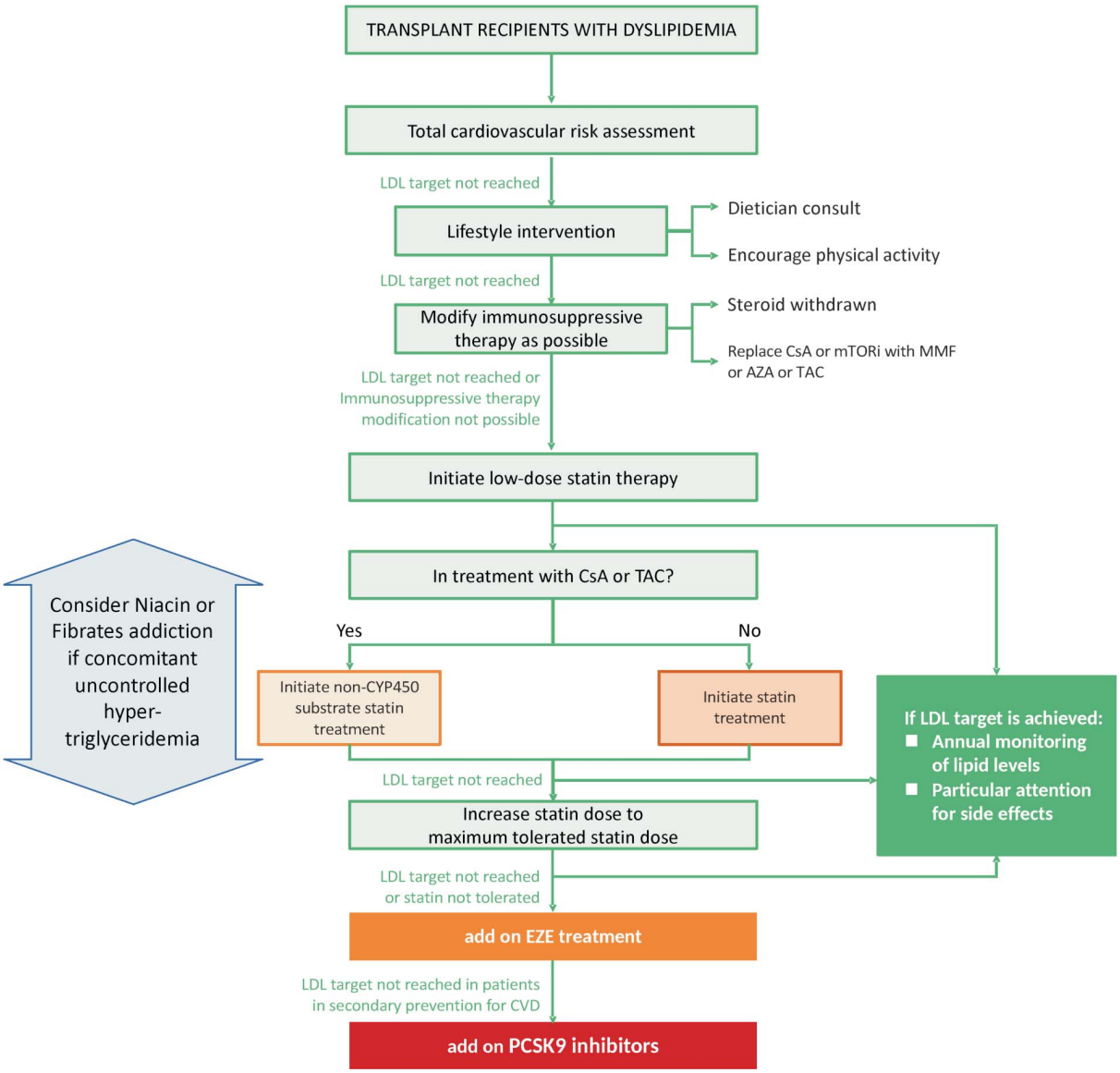

Figure 1. *Process flowchart for the selection of hypolipidemic agents for KTRs.[18] AZA, azathioprine; CsA, cyclosporine; CVD, cardiovascular disease; CYP450, cytochrome P450; EZE, ezetimibe; LDL, low-density lipoprotein; MMF, mycophenolate mofetil; mTORi, mammalian target of rapamycin inhibitors; PCSK9, proprotein convertase subtilisin/kexin type 9; TAC, tacrolimus.*

Hypertension is highly prevalent in KTRs. In addition to the traditional hypertension risk factors of age, sex, obesity, smoking, and family history, CKD and KTx are associated with additional risk factors.[20] For example, the deterioration of renal function with disease progression activates the renin-angiotensin-aldosterone system, increasing BP.[20] KTRs are also affected by the inherent risk factors of the donor (such as donor age, sex, and the presence or absence of hypertension, obesity, and family history) and graft status (such as poor allograft quality and immunosuppression in extended criteria donor KTx). Other risk factors include recipient genes, immunosuppressant use (particularly CNIs), transplant renal artery stenosis, and graft rejection.[20,21] A previous study found that the production of angiotensin II type 1 receptor-activating antibodies in KTRs led to continuous activation of the renin-angiotensin-aldosterone system, resulting in a series of rejection-related symptoms such as deterioration of renal function and hypertension, and reduced allograft survival.[22]

The literature rarely discusses BP control in KTRs. Relevant guidelines, such as the 2017 American College of Cardiology/American Heart Association hypertension guidelines and 2021 KDIGO Clinical Practice Guidelines for the Management of Blood Pressure in Chronic Kidney Disease, have provided consistent recommendation approaches.[23] First, the BP control targets are set following the recommendations of most cardiology-related treatment guidelines, that is, systolic BP < 130 mmHg and diastolic BP < 80 mmHg.[24] Previous research has found that control of systolic BP to < 130 mmHg led to better long-term survival than control to < 140 mmHg.[24] However, results of the SPRINT trial indicated that the adoption of a stricter target of 120 mmHg may instead cause a decrease in the estimated glomerular filtration rate (eGFR) and increase the risk of developing acute kidney injury.[24] Despite the limited number of studies on the strict control of BP in KTRs, the implementation or non-implementation of strict BP control did not lead to significant differences in the overall outcomes in pediatric KTRs in a small-scale trial. Instead, a tendency towards kidney function deterioration was observed.[25] Therefore, the literature does not support stricter BP control in KTRs. Conventional office blood pressure (OBP) measurements are still currently recommended in accordance with the 2021 KDIGO guidelines.[24] However, more recent BP measurement methods, such as ambulatory blood pressure monitoring (ABPM), can be used to monitor BP changes in the morning or at night. This enables better observation of BP changes throughout the day compared to traditional measurement methods.[24,26] A meta-analysis investigated the consistency in BP data between OBP measurements and ABPM, and reported differences in BP values obtained by the two methods; ABPM could detect approximately 20% of patients who had masked hypertension.[7] Another systematic review indicated that ABPM provided a better reflection of target organ damage than traditional OBP measurements, and also contributed to better CV or kidney-related clinical control effects.[26] Given its advantages, ABPM may become a tool for monitoring BP in KTRs. However, clinical studies on the use of ABPM for monitoring the BP of KTRs have not yet been reported; thus, further research is needed. Nonetheless, a meta-analysis indicated that BP could reflect the risk of end-organ damage in KTRs. The present expert consensus recommends that BP measurements should be primarily performed by standard clinical assessments but can be substituted by 24-hour ambulatory or home monitoring.

Dihydropyridine CCBs and ARBs are recommended as the primary medications for BP control. A previous meta-analysis found that the relative risk of graft loss did not differ significantly between non-DHPs and a placebo.[24] In contrast, DHPs and ARBs reduced the graft loss risk by 38% and 65%, respectively. The present expert consensus recommends the use of DHP CCBs or ARBs as first-choice drugs for BP control in adult KTRs. However, KTRs often have complex conditions. It is recommended that adjustments be made based on existing risk factors and health status on a case-by-case basis, with the achievement of BP targets given top priority.[20] Furthermore, drug-drug interactions between immunosuppressants and antihypertensive drugs should be considered.

DM control in KTRs

1. The glycated hemoglobin (HbA1c) target of KTRs should be controlled within 6.5-7.0% to reduce the risk of CVD (94% agreement, strongly recommended).
2. Sodium-glucose cotransporter-2 (SGLT2) inhibitors are the recommended first-line drugs for treating PTDM in KTRs (25% agreement; consensus not reached).

The causes of PTDM in KTRs include common pathogenic mechanisms related to organs or tissues such as the pancreas, liver, and kidneys, and the influence of the use of post-transplantation immunosuppressants or viral infection (such as hepatitis C virus or cytomegalovirus infection).[27]

Considering the high risk of PTDM in KTRs, regular postoperative plasma glucose monitoring and subsequent diagnosis and treatment based on the measured plasma glucose levels are needed. Table 5 shows the diagnostic criteria for DM in KTRs.[28] However, the frequency of plasma glucose monitoring, timing of PTDM diagnosis, and choice of medications differ across different postoperative time points.[27,29] During the first five postoperative weeks, KTRs usually have unstable postoperative plasma glucose levels and are thus prone to developing transient hyperglycemia. During this period, weekly glucose tolerance testing or fasting plasma glucose measurements are recommended, and appropriate pharmacological treatments should be administered in cases of hyperglycemia. However, a diagnosis of PTDM is not re-

quired.[27,29] Regarding the choice of medication, the fact that patients may exhibit the greatest instability in plasma glucose levels during the first postoperative week should be considered. Therefore, insulin should be the first choice for plasma glucose control, with postprandial plasma glucose < 126 mg/dL being the recommended treatment target.[27,29] With the use of oral hypoglycemic agents in addition to insulin within the first postoperative year, it is recommended that glucose tolerance testing or fasting plasma glucose measurements be performed at least once every three months. Once the patient meets the criteria stated in Table 5 or manifests hyperglycemia-related symptoms, a confirmed diagnosis of PTDM is made.[27,29] Lifestyle modification should be considered the first-choice intervention for treatment. If pharmacological treatment remains necessary, oral hypoglycemic agents should be given priority over insulin, with postprandial plasma glucose < 126 mg/dL still used as the treatment target.[27,29] Beyond one year postoperatively, it is still necessary to perform regular annual follow-ups of plasma glucose-related markers. In addition to the aforementioned fasting and postprandial plasma glucose levels, HbA1c levels can be used as a diagnostic criterion for PTDM and a marker for long-term plasma glucose monitoring. A diagnosis of PTDM is made when the HbA1c level is > 6.5%, and the treatment options for this stage are identical to those of the previous stage.[27,29] The present expert consensus recommends controlling HbA1c levels between 6.5% and 7.0%. However, when setting the plasma glucose target, priority should be given to ensuring the absence of hypoglycemic symptoms. In addition, given the high tendency of drug-drug interactions between CNIs and hypoglycemic agents, particular attention should be paid to closely monitoring plasma glucose data and dose adjustment of hypoglycemics when making dose adjustments for immunosuppressants.[28]

Lastly, the principles for selecting oral hypoglycemic agents are generally similar to the process for choosing typical CKD treatment drugs; that is, metformin is the first-line drug. However, a dose reduction should be made when the eGFR is < 45 mL/min, and must be made when the eGFR is < 30 mL/min.[30] Other oral hypoglycemic agents can serve as second-line treatment options, and a decision to switch to or include other oral hypoglycemic agents can be made based on the plasma glucose status of the patient.[30] It must be noted that the most significant difference in the choice of oral hypoglycemic agents between KTRs and patients with typical kidney disease lies in the use of SGLT2 inhibitors. Currently, clinical studies on the use of SGLT2 inhibitors in KTRs remain extremely limited.[31] Although a number of observational studies have supported the use of SGLT2 inhibitors in patients with PTDM, it has also been reported that SGLT2 inhibitors may elevate the risk of post-transplantation urinary tract infection in KTRs.[30,32,33] Therefore, no consensus has been reached on whether SGLT2 inhibitors are recommended as a standard treatment for patients with PTDM.

Treatment of congestive heart failure in KTRs

1. After KTx, changes in LVEF should be continuously monitored and medications for HF should be adjusted individually (100% agreement, strongly recommended).
2. The principles of the use of angiotensin-converting enzyme inhibitors (ACEIs)/ARBs/angiotensin receptor-neprilysin inhibitors (ARNIs), mineralocorticoid receptor antagonists (MRAs), and SGLT2 inhibitors for the treatment of first-onset HF in KTRs are identical to the treatment principles for patients with HF (62% agreement, consensus not reached).

Highly complex bidirectional effects exist between

Table 5. Diagnostic criteria for diabetes mellitus in kidney transplant recipients[27]

1. Fasting plasma glucose ≥ 126 mg/dL (7.0 mmol/L). Fasting is defined as no caloric intake for at least 8 hours.

OR

2. Symptoms of hyperglycemia and a casual plasma glucose ≥ 200 mg/dL (11.1 mmol/L). Casual is defined as any time of day without regard to time since last meal. The classic symptoms of hyperglycemia include polyuria, polydipsia, and unexplained weight loss.

OR

3. Two-hour plasma glucose > 200 mg/dL (11.1 mmol/L) during an oral glucose tolerance test. The test should be performed as described by the WHO, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.

WHO, World Health Organization.

the pathological mechanisms of HF and CKD. The deterioration of kidney function leads to issues such as body fluid retention and BP elevation. This increases the cardiac workload and the risk of HF. The occurrence of HF increases the tendency for inadequate kidney perfusion, which causes further CKD deterioration. New-onset HF occurs in up to 17-21% of patients with CKD, and approximately 55% of patients with HF have CKD. In clinical practice, these two diseases often occur concurrently and influence each other.[34,35] During the treatment of patients with HF complicated by CKD, consideration is usually given to the side effects associated with HF medications used in guideline-directed medical therapy, such as hypotension, deterioration of kidney function, and hyperkalemia.

Controlling HF is vital for KTRs. A previous study reported that kidney transplant candidates with an LVEF < 30% measured before transplantation had a median survival of approximately three years. A 1% improvement in LVEF led to a 25% reduction in mortality risk.[36] Even among KTRs in whom no CVDs were found within one year after transplantation, approximately 3.6% developed HF within five years, and up to 21.6% developed HF within 20 years, with the risk of HF being 2-5 times higher than that of normal individuals. Compared with patients with normal LVEF, those with abnormal LVEF also had a higher risk of CV-related death. Therefore, LVEF is crucial for overall survival before and after KTx.[34,37] Patients who develop HF after transplantation are also at a higher risk of death and graft failure.[35,38,39] Evidence is lacking to support the clinical benefits of regular post-transplantation echocardiographic examinations in KTRs. However, for patients with symptoms and signs of suspected HF or hemodynamic instability during dialysis, echocardiography should be considered.[34] The criteria for patients with HF are identical to those adopted for non-KTRs.[40,41] Most clinical studies have excluded transplant recipients, leading to a lack of clinical trial data on the use of HF-related medications in KTRs. In addition, the limited existing research is complicated by small sample sizes. In principle, medication is still based on general HF guidelines and in accordance with strategies for patients with CKD.[34,40,41] Although a decrease in eGFR may occur during the early stages of ACEI/ARB, ARNI, and SGLT2 inhibitor use, the decrease is usually transient and reversible, and they can still provide significant long-term benefits to clinical outcomes. Therefore, discontinuation of HF medication following a temporary decline in kidney function is not advisable. Besides eGFR, the patient's individual condition and other clinical data should be assessed to determine whether kidney injury has occurred before considering any dose adjustments for HF medications.[34] For patients with manageable hyperkalemia, the use of ACEIs/ARBs, ARNIs, and MRAs can still be considered. However, anti-hyperkalemia medication and/or discontinuation of these medications must be considered due to the occurrence of severe hyperkalemia. Previous clinical studies have demonstrated that the use of SGLT2 inhibitors in patients with CKD, DM, and eGFR > 20-25 mL/min/1.73 m^2 effectively reduced the occurrence of HF-related hospitalization events and the risk of CV death. Clinical studies on the treatment of HF in KTRs are scarce and lack a consensus. Among the various SGLT2 inhibitors, empagliflozin is currently the most widely used for treating KTRs.[42] However, as mentioned previously, the risk of infection in patients must be considered, and regular monitoring and caution are required when using these medications.

Treatment of arrhythmia in KTRs

1. Screening for atrial fibrillation (AF) (such as regular outpatient electrocardiography [ECG] and long-term continuous ECG recording) is recommended in patients who have undergone KTx and those who have received a transplant (94% agreement, strongly recommended).
2. Regarding the combined use of novel oral anticoagulants (NOACs) and immunosuppressants in KTRs with AF, consultation with a cardiologist for drug selection and dose adjustment is recommended, and the use of other stroke prevention methods should be considered (100% agreement, strongly recommended).

Although KTRs have a lower risk of CV events after transplantation than before transplantation, they still have a higher risk of CV events than the general population.[43] For example, the annual incidence of post-transplantation new-onset AF in KTRs has been reported to be approximately 3.6%, which was higher than that of the general population. Risk factors associated with post-transplantation AF include older age, male sex, white race, kidney failure from hypertension, and CAD.[43] The occurrence of new-onset AF significantly increases the

risk of post-transplantation mortality and death-censored graft loss. Similarly, patients with AF before transplantation have a higher risk of mortality and death-censored graft loss than those without AF before transplantation.[43-45] Therefore, active treatment and control of AF are necessary for KTRs. Besides the risks posed by AF, the occurrence of post-transplantation AF increases the risk of ischemic stroke in patients.[45]

Considering the risks introduced by AF in KTRs, screening for AF (such as regular outpatient ECG and long-term continuous ECG recording) is recommended for patients who have undergone KTx and those who have received a transplant.[46,47] In patients in whom AF has been detected through screening, the use of NOACs is necessary to reduce the risk of future ischemic stroke events. However, the greatest difference in NOAC use between KTRs and other patients is due to the fact that most immunosuppressants used by KTRs have potential drug-drug interactions with NOACs, with CNIs exerting the most significant influence.[48] Therefore, particular care must be taken when adjusting the dose and monitoring NOAC use in KTRs. Among the four currently available NOACs, dabigatran has the most severe drug-drug interactions with CNIs and should be avoided.[48] Given that significant drug-drug interactions exist between tacrolimus and all types of NOACs, warfarin can be considered in such patients. Previous studies comparing the differences in therapeutic effects and safety between NOACs and warfarin in KTRs found that the therapeutic effects did not differ significantly. Nevertheless, the patients who received warfarin had a higher tendency of graft failure.[49,50] Therefore, the three NOACs other than dabigatran may serve as the main options for stroke prevention. Although significant interactions exist between NOACs and immunosuppressants, regularly monitoring the plasma concentration of NOACs is not advisable and should only be considered under the following circumstances: occurrence of stroke after the use of NOACs, patient weight > 120 kg, observation of significant drug-drug interactions, and consideration of using reversal agents in patients requiring emergency surgery.[47] In summary, during the combined use of NOACs with immunosuppressants in KTRs with AF, it is recommended that cardiologists and doctors responsible for kidney transplant care engage in joint discussion, selection, and dose adjustments of medications and consider the use of alternative methods for stroke prevention.

Besides the issue of drug-drug interactions during the use of NOACs in KTRs who develop AF, approximately 30% of KTRs experience AF. Risk factors associated with its occurrence include male sex, length of dialysis therapy, and the presence of coronary artery calcification.[51] However, the occurrence of AF is mainly related to CAD or previous myocardial infarction. Therefore, the risk of AF can be effectively reduced through good control of CAD.[51] For patients who require the use of antiarrhythmic agents for controlling arrhythmias, attention should be paid to the interactions of these medications with immunosuppressants. For example, the combined use of antiarrhythmic agents such as propafenone, flecainide, and amiodarone, with tacrolimus significantly prolongs QT duration. Therefore, caution should be exercised when using these medications.

Treatment of CAD in KTRs

1. The combination of aspirin and clopidogrel is the first-choice regimen for KTRs who require dual antiplatelet therapy (DAPT) (93% agreement, strongly recommended).
2. For KTRs with multivessel CAD, coronary artery bypass grafting (CABG) and percutaneous coronary intervention (PCI) are good options for revascularization (91% agreement; strongly recommended).

Cardiovascular events are the most common cause of death among KTRs in short- and long-term post-transplantation follow-up.[52] In particular, CAD is a major cause of death or an indirect cause of graft loss in KTRs. Compared to the general population, KTRs have a higher risk of AMI, with an incidence of 3.5% at three months post-operatively, and subsequent mortality is as high as 50%.[52] Therefore, the detection and treatment of CAD is vital for KTRs.

As with other cardiovascular comorbidities, guidelines and expert consensus are lacking on the treatment of CAD in KTRs. Recommendations are mainly based on strategies similar to those used for patients with CKD, with adjustments performed according to the status of the KTRs. The presence of hemodynamic stress, a proinflammatory state, plaque destabilization, and endothelial dysfunction in patients after transplantation leads to a higher risk of post-transplantation AMI. In clinical practice, older age of the kidney transplant recipient or do-

nor, or significant elevation of biomarkers such as troponin T and troponin I during pre- or post-transplantation monitoring indicates that the recipient is at high risk for AMI. Therefore, postoperative care must be provided with caution in such patients.[53]

Clinical studies on the use of medications for the primary prevention of CAD in KTRs have not been reported. Therefore, the use of any medication for the primary prevention of CAD in CVD-free KTRs is currently not advisable. In patients with a history of related diseases, the use of aspirin for primary prevention can be considered, whereas clopidogrel can be used as an alternative in patients allergic to aspirin. Patients who have previously experienced AMI or have undergone coronary artery stent placement require DAPT for secondary prevention. However, the presence of platelet aggregation issues in most KTRs and drug-drug interactions between platelet aggregation inhibitors and immunosuppression therapy increases the risk of bleeding side effects.[54] For example, combination therapy with prednisolone and tacrolimus impairs the gastrointestinal epithelium and causes ulcerated mucosal damage, increasing the risk of bleeding.[54] Therefore, care must be taken when using DAPT for secondary prevention in KTRs.

The choice of platelet aggregation inhibitor is usually based on the type of CAD. In principle, aspirin use is recommended for patients with stable angina, ST-segment elevation myocardial infarction (STEMI), or non-STEMI (NSTEMI). Patients with STEMI or NSTEMI will require combination therapy with a second platelet aggregation inhibitor such as clopidogrel or ticagrelor.[55] For patients with STEMI who have undergone PCI, clopidogrel, ticagrelor, or prasugrel can be selected for combination therapy with aspirin.[55] However, considering the lack of clinical trial data on the use of DAPT in KTRs and the greater availability of clinical experience in using clopidogrel, the preferential use of clopidogrel in patients who require DAPT can be considered. However, appropriate choices should be made after incorporating other clinical factors.

Although previous studies have reported the prevalence of post-transplantation coronary artery calcification among KTRs, the severity of CAD in such patients remains relatively lower than that observed in patients on long-term dialysis.[56] In the event of multiple coronary artery occlusions in KTRs, a choice must be made between PCI and CABG for revascularization. A recent meta-analysis comparing the two techniques reported that although the short-term survival rate of the PCI group was higher than that of the CABG group, both procedures led to similar long-term survival rates.[57] Therefore, it is recommended that either PCI or CABG be selected for treating KTRs with multiple coronary artery occlusions. To reach a treatment decision, other factors, such as patient condition, anticipated short- or long-term outcomes, and a shared decision-making approach with the patient should be adopted.

CONCLUSION

KTx is the optimal treatment for patients with advanced kidney disease, with younger patients undergoing transplantation achieving greater long-term survival benefits. Data published by the National Health Insurance Administration of the Ministry of Health and Welfare of Taiwan indicate that the 10-year survival rate of patients with KTx is 79%, which is more than three times the survival rate of 22.9% in patients undergoing kidney dialysis. The remarkable effectiveness of KTx can be attributed to the comprehensive care system in Taiwan for transplantation surgery, which encompasses interdisciplinary and cross-specialty care. However, CVD induced by kidney disease is the leading cause of mortality in KTRs. Therefore, improving cardiovascular risk factors is necessary to enhance long-term outcomes and reduce the occurrence of CVD.

With the prolongation of long-term survival after transplantation, the maintenance of cardiovascular health is essential. This includes pre-transplantation evaluations; meticulous care before and after surgery; and provision of interdisciplinary care in the pre-, peri-, and post-transplantation stages through cross-specialty collaborations and holistic, integrated care approaches. Patients must also adopt a mindset of self-health management, maintain an appropriate diet and exercise routine, adhere to medication prescriptions and long-term follow-ups, and cultivate good lifestyle habits to prevent other chronic diseases or complications.

New knowledge gained

This multidisciplinary consensus integrating nephro-

logy, cardiology, and transplant surgery perspectives affirms the need to adapt international guidelines to the specific Taiwanese healthcare context. While strong consensus was achieved for foundational management, such as pre-transplant cardiac assessment, blood pressure targets, and first-line statin use, critical areas of clinical uncertainty were also identified. Notably, consensus was not reached on the first-line use of SGLT2 inhibitors for post-transplantation diabetes or the standardized application of general heart failure medication principles, highlighting the urgent need for localized clinical evidence.

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DECLARATION OF CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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