Cardiology Update: Hypertension and Hyperlipidemia After Transplantation

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Objectives

Discuss	Discuss risks, contributing factors, transplant-considerations, and treatment options for hypertension and hyperlipidemia post-transplant
Analyze	Analyze treatment modalities for hyperlipidemia and their place in post-transplant care
Review	Review guideline updates and apply them to the transplant population

Hypertension

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CLINICAL PRACTICE GUIDELINE

2025 AHA/ACC/AANP/AAPA/ABC/ACCP/ACPM/AGS/AMA/ASPC/NMA/PCNA/SGIM Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults

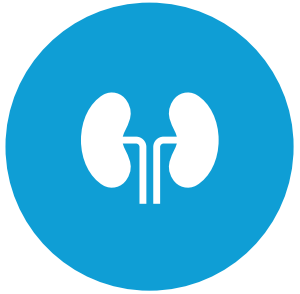
A Report of the American College of Cardiology/American Heart Association
Joint Committee on Clinical Practice Guidelines

Developed in Collaboration With and Endorsed by American Academy of Physician Associates; American Association of Nurse Practitioners; American College of Clinical Pharmacy; American College of Preventive Medicine; American Geriatrics Society; American Medical Association; American Society of Preventive Cardiology; Association of Black Cardiologists; National Medical Association; Preventive Cardiovascular Nurses Association; and the Society of General Internal Medicine.

Risks

Cardiac remodeling

Stroke



Renovascular hypertension → CKD

Non-alcoholic fatty liver disease (NAFLD)

Post-Transplant Hypertension Pathophysiology

Calcineurin Inhibitors (CNI)

- Cyclosporine > tacrolimus
- Independent of nephrotoxicity
- Potential mechanisms
  - Increase activity of vasoconstrictors
  - Stimulate renin angiotensin aldosterone system (RAAS)
  - Inhibit production of vasodilator substances

Corticosteroids

- Salt retention

Essential Hypertension

Luke R. J. Am. Soc. Nephrol. 1991

Other Contributing Factors

- Fluid / volume overload
- Pain
- Obstructive sleep apnea (OSA)
- Other medications:
  - Serotonin and norepinephrine reuptake inhibitors (SNRI)
  - Oral contraceptives

Calvi A, et al. Front Cardiovasc Med. 2021

Blood Pressure Classification

	SBP		DBP
BP Category			
Normal	<120 mm Hg	and	<80 mm Hg
Elevated	120 to 129 mm Hg	and	<80 mm Hg
Hypertension			
Stage 1	130 to 139 mm Hg	or	80 to 89 mm Hg
Stage 2	≥140 mm Hg	or	≥90 mm Hg

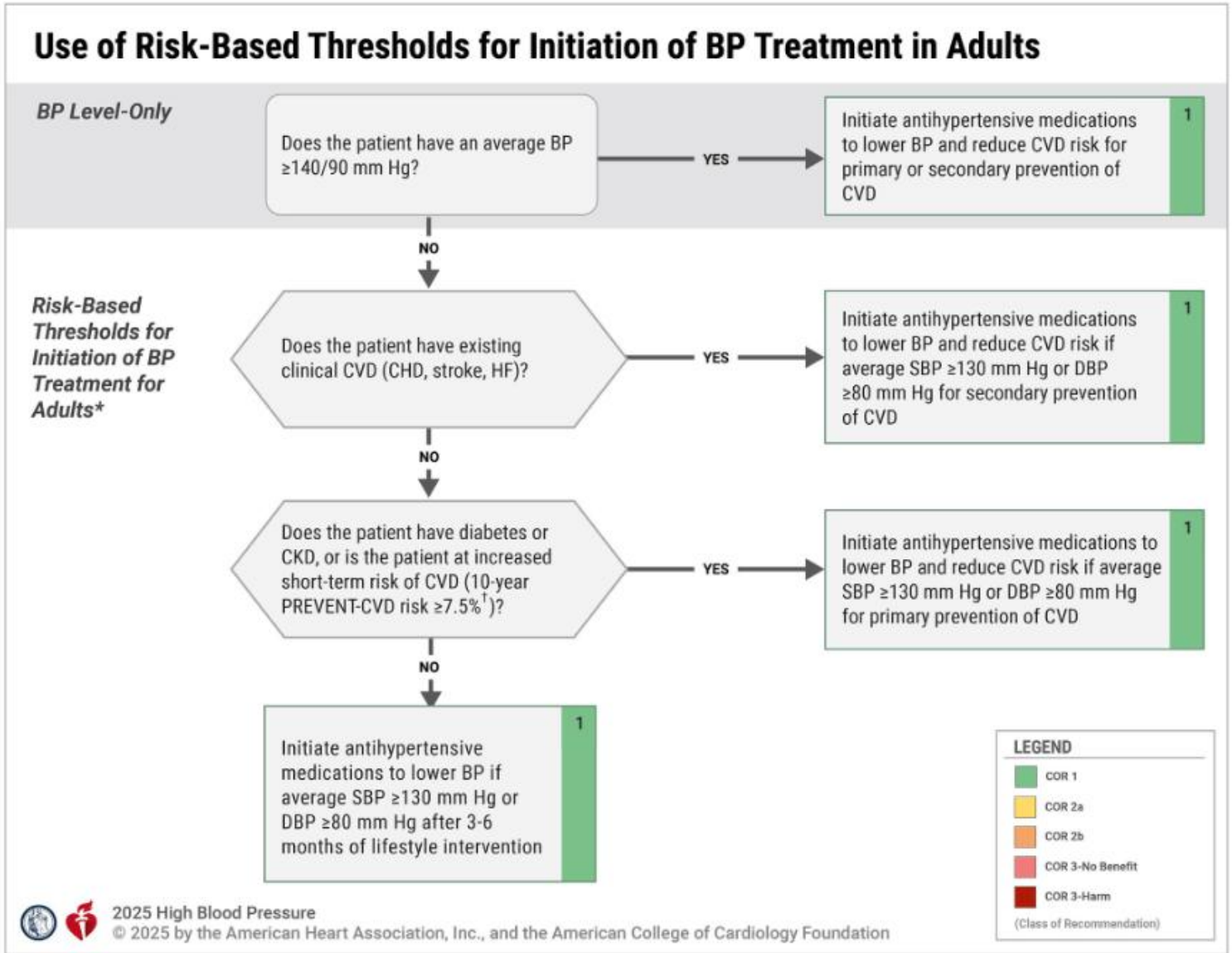
Whelton P, et al. Hypertension 2017

Blood Pressure Goals

	BP Targets	BP Categories[a]	SBP (mm Hg)	DBP (mm Hg)
JNC 7, 2003	< 140/90 mm Hg < 130/80 mm Hg for those with diabetes or chronic kidney disease	Normal	< 120	< 80
		Prehypertension	120–139	80–89
		Stage 1 hypertension	140–159	90–99
		Stage 2 hypertension	≥ 160	≥ 100
JNC 8, 2014	< 150/90 mm Hg for patients ≥ 60 < 140/90 mm Hg for patients < 60, diabetes, and chronic kidney disease	Was not a comprehensive set of recommendations, and did not discuss hypertension diagnostic thresholds		
ACP/AAFP, 2017	< 150/90 mm Hg for patients ≥ 60 < 140/90 mm Hg for patients at higher CV risk, or with a history of stroke or TIA	Was not a comprehensive set of recommendations and did not discuss hypertension diagnostic thresholds Did not address recommendations in patients < 60		
ACC/AHA, 2017	≤ 130/80 mm Hg	Normal	< 120	< 80
		Elevated	120–129	< 80
		Stage 1 hypertension[b]	130–139	80–89
		Stage 2 hypertension	≥ 140	≥ 90

Whelton P, et al. Hypertension 2017

Use of Risk-Based Thresholds for Initiation of BP Treatment in Adults



2025 High Blood Pressure

Hypertension Treatment

Lifestyle Modifications – 1st line for ALL

DIET

EXERCISE

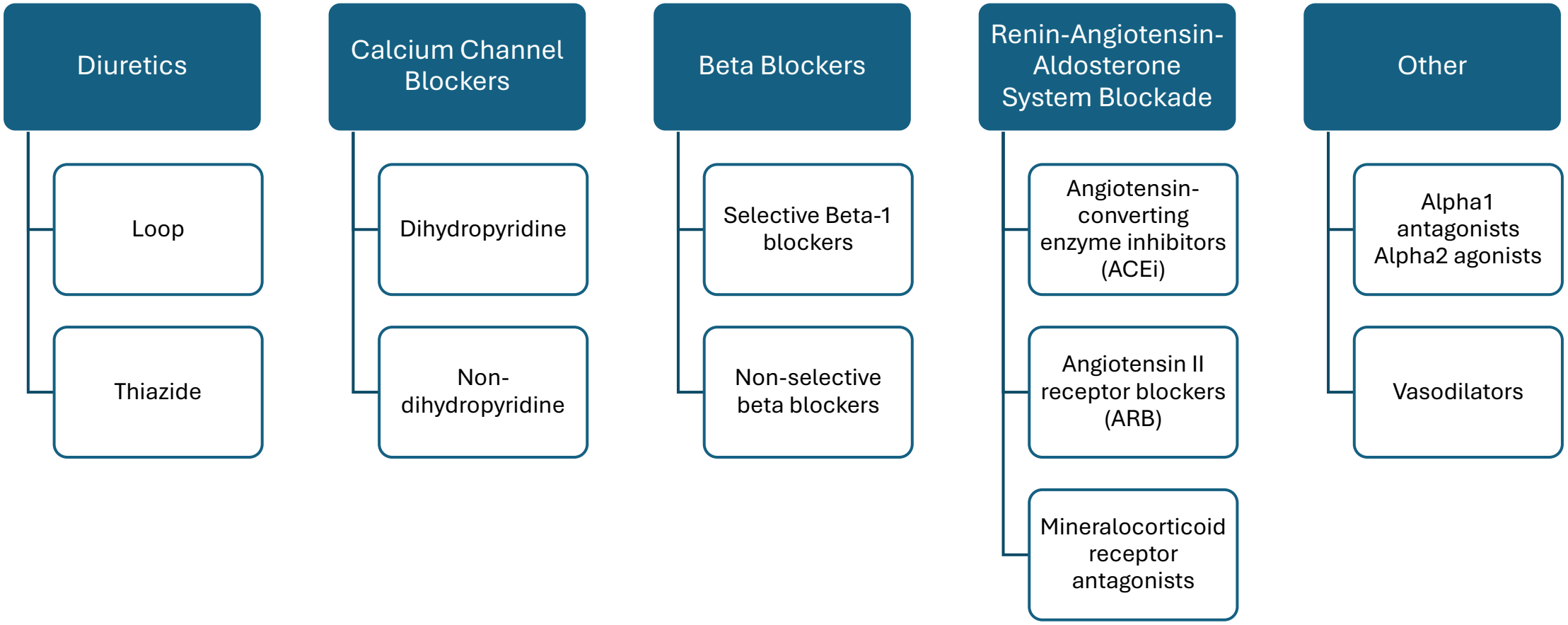
STRESS REDUCTION

Tantisatamo E et al. Front Med (Lausanne). 2020

Non-Pharmacologic Treatment

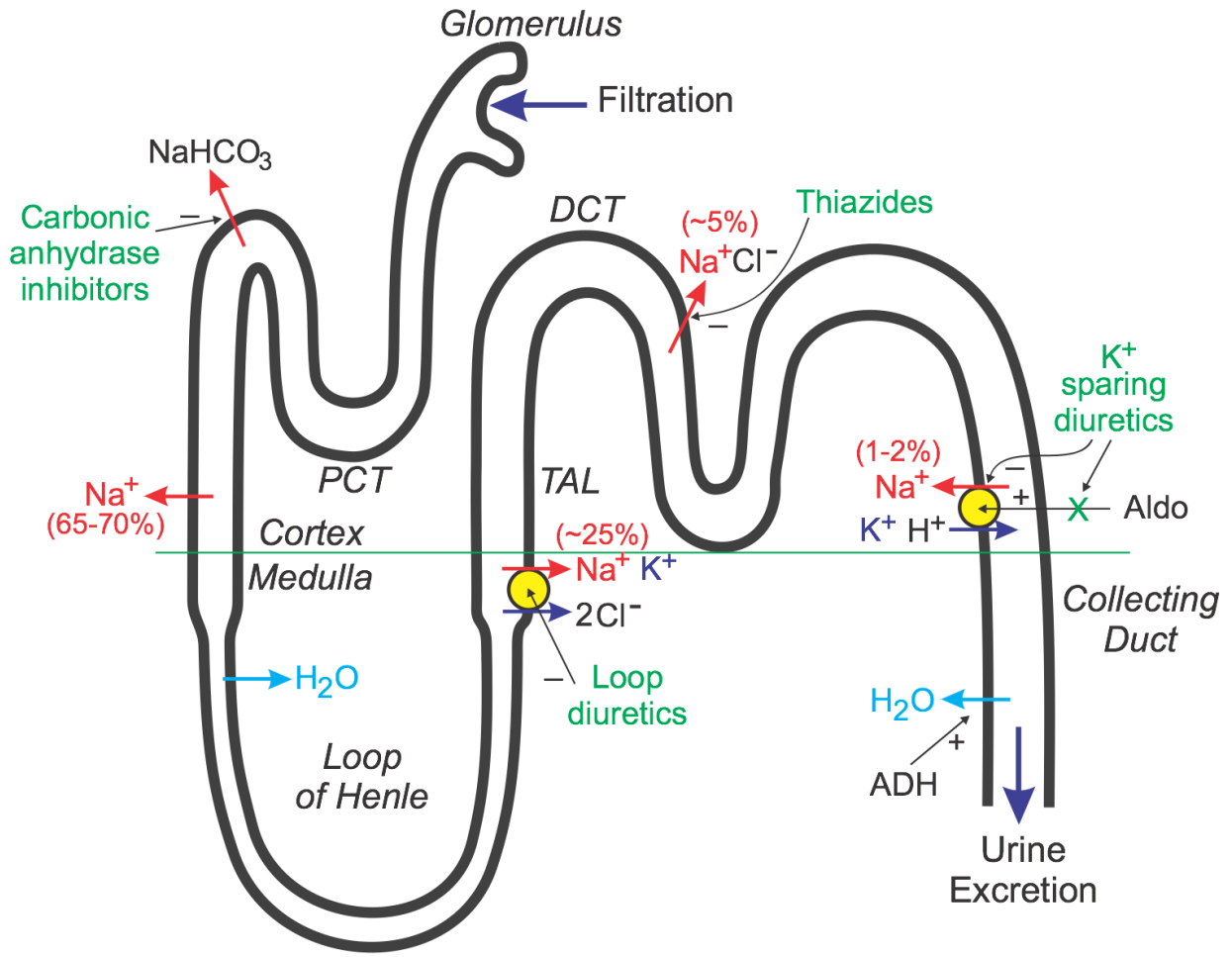
Intervention	Target/Biomarker	Evidence-Based Goals	Approximate Mean Change in SBP (mm Hg)*		References
			With Hypertension	Without Hypertension	
Weight loss	Body weight or BMI	Aim for sustained ≥5% reduction in body weight or ≥3 kg/m^2 reduction in BMI; expect about 1 mm Hg reduction for every 1-kg reduction in body weight	−6 to −8	−3 to −5	2,6,14,52
Heart-healthy diet	DASH eating pattern	Consume a diet rich in fruits, vegetables, whole grains, and low-fat dairy products, with reduced content of saturated and total fat	−5 to −8	−3 to −7	13-15,64,120
Reduced intake of sodium	Dietary sodium intake; 24-h urinary sodium	Optimal goal is <2,300 mg/d, but aim for an ideal limit of <1,500 mg/d	−6 to −8	−1 to −4	16-18,79,120,121
Use of salt substitute	Replace cooking/table salt (100% sodium chloride) with salt substitute (25%-30% potassium chloride, 65%-75% sodium chloride, and 0%-10% flavoring agents); 24-h urinary sodium and potassium	Reduce dietary sodium intake as above	−5 to −7	−5	20-22,93
Enhanced intake of potassium	Dietary potassium intake; 24-h urinary potassium	Aim for 3,500-5,000 mg/d, ideally by consumption of a diet rich in potassium; or alternative use of moderate-dose pharmacological potassium supplementation (<80 mmol)	−6	−3 to −6	25-27
Reduced alcohol intake	Alcohol consumption	Optimal goal is abstinence for all adults for best health outcomes; in patients who consume alcohol, aim for >50% reduction in daily intake to no more than 2 drinks/d in men or 1 drink/d in women	−4 to −6	−3	29
Exercise	Aerobic exercise	90-150 min/wk 65%-75% heart rate reserve	−4 to −8	−2 to −7	14,33,36,120,122
	Dynamic resistance	90-150 min/wk 50%-80% 1 rep maximum 6 exercises, 3 sets/exercise, 10 repetitions/set	−2 to −7	−2 to −5	33,36,106,107
	Isometric resistance	4 × 2 min (hand grip), 1 min rest between exercises, 30%-40% maximum voluntary contraction, 3 sessions/wk	−5 to −10	−4 to −6	14,32,33,36,109,110
Meditation	Transcendental meditation	Training by a professional, followed by 2 × 20 min sessions/d while seated comfortably with eyes closed	−5 to −7	−5	14,119
Breathing control	Slowing respiration	Device-guided session to decrease respiration to <10 breaths/min for 15 min/d	−5	−5	14

Pharmacologic Therapy



Diuretics

- Thiazide diuretics
  - Hydrochlorothiazide
  - Chlorthalidone
- Clinical Pearls:
  - Longer half-life than loop diuretics
    - furosemide, torsemide, bumetanide
  - Worsen gout
  - Lower magnesium, potassium, sodium, and calcium



Roush GC et al. *J Cardiovasc Pharmacol Ther*. 2014

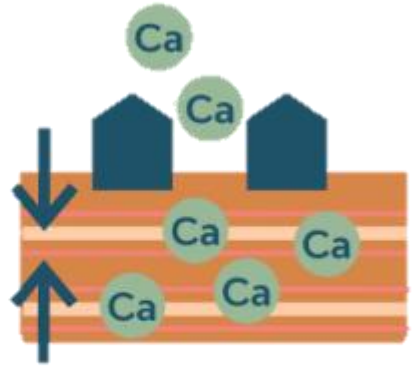
Calcium Channel Blockers

Dihydropyridine (DHP)

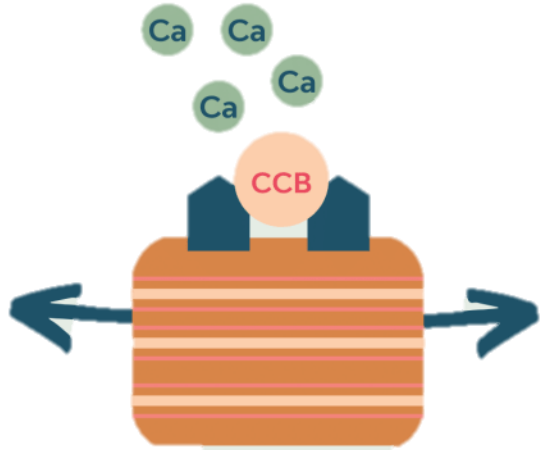
- Amlodipine, nifedipine, felodipine
  - Potential benefit with CNI toxicity
  - Peripheral edema

Non-dihydropyridine (non-DHP)

- Diltiazem, verapamil
  - Potential drug interactions due to moderate CYP3A4 inhibition
  - Bradycardia



Calcium influx into heart and vascular smooth muscles leads to vasoconstriction and contraction of these muscles.



Calcium channel blockers inhibit L type calcium channels leading to vasodilation, decreased contractility, and a reduction in heart rate.

Dominic A, J Clin Hypertens (Greenwich). 2003

Beta Blockers

Selective Beta1 blockers

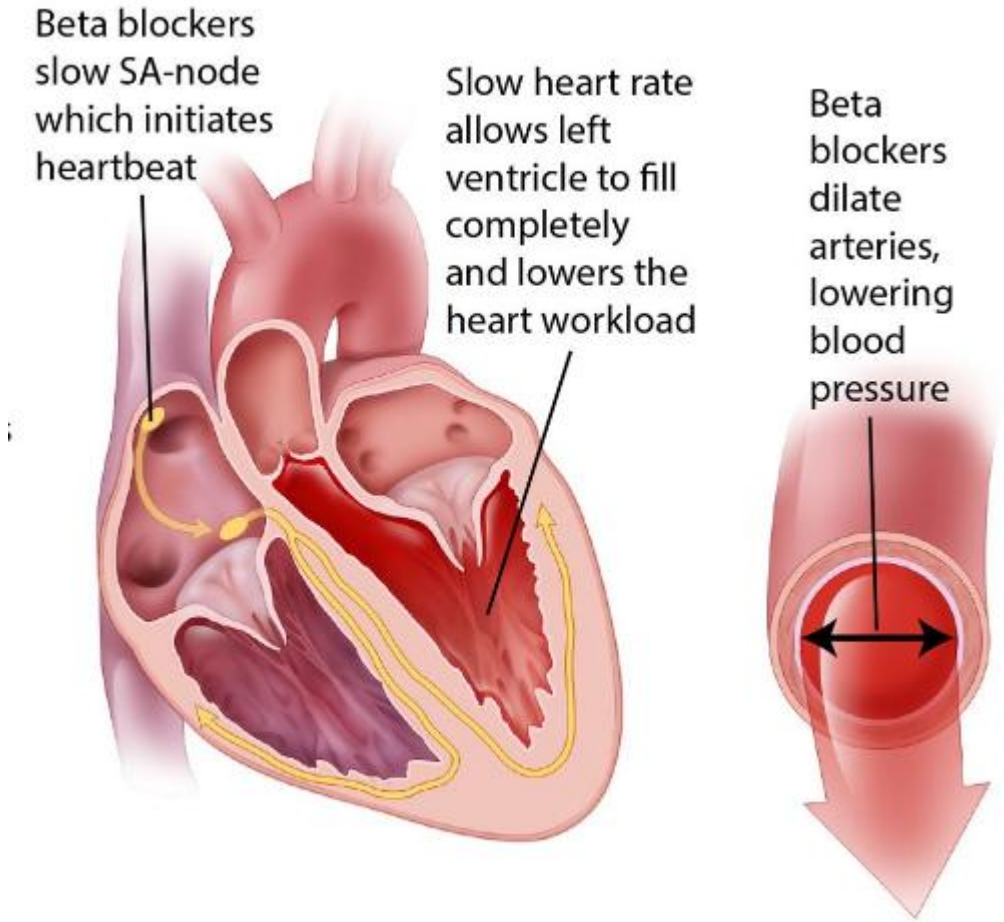
- Metoprolol, atenolol, nebivolol, esmolol

Non-selective

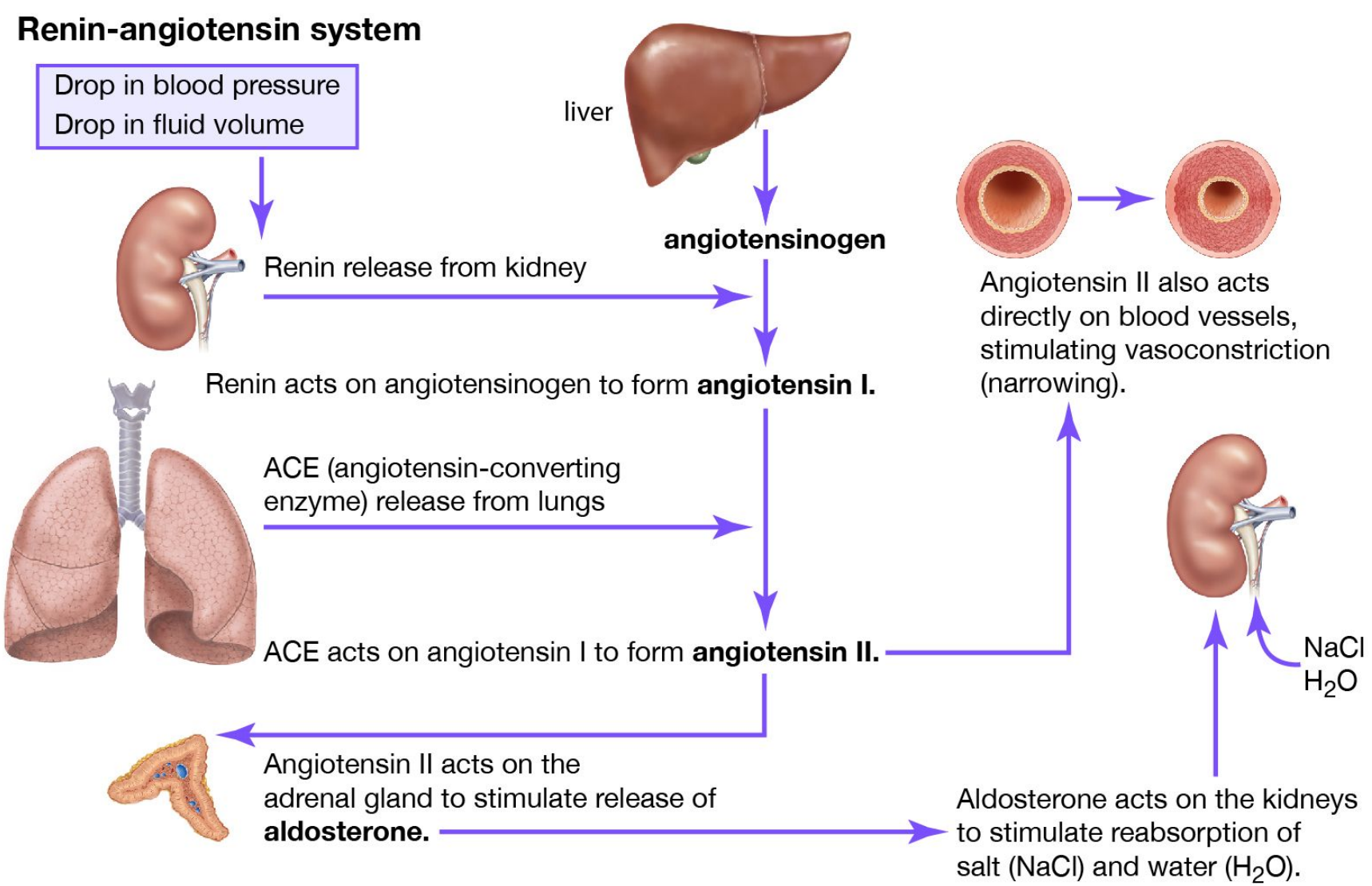
- Carvedilol, labetalol, propranolol

Clinical pearls:

- Caution in heart transplant
- Caution with airway disease (non-selective)
- Can be used for atrial fibrillation prophylaxis
- May improve tremors
- Fatigue
- ED



Masarone D, et al. Clin Transplant 2020



Fountain J, et al. Natl Lib Med. 2023

Renin-Angiotensin-Aldosterone System Blockade

Angiotensin-converting enzyme inhibitors (ACEi)

- Lisinopril, captopril, enalapril, ramipril

Angiotensin II receptor blockers (ARB)

- Losartan, candesartan, irbesartan, olmesartan, telmisartan, valsartan

Clinical Pearls:

- Improves proteinuria
- ACEi interaction with sirolimus
- Losartan reduces uric acid levels
- Acute kidney injury (AKI) -caution with CNI use
- Angioedema
- Hyperkalemia

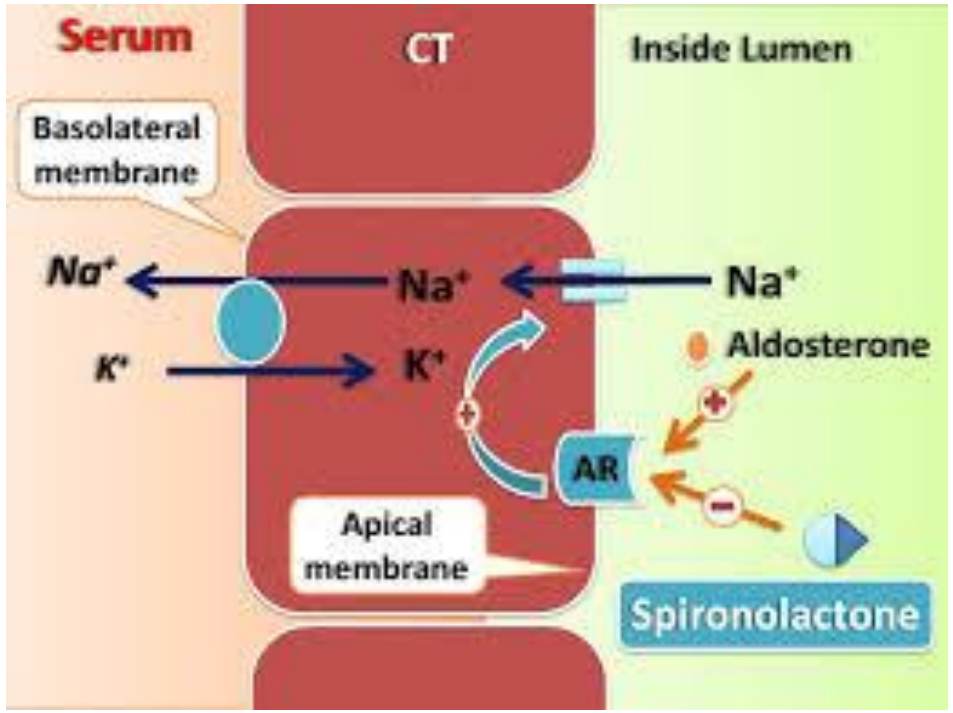
Renin-Angiotensin-Aldosterone System Blockade

Mineralocorticoid receptor antagonists

- Spironolactone
- Eplerenone

Clinical Pearls:

- Hyperkalemia
- Gynecomastia



Fountain J, et al. Natl Lib Med. 2023

Other Agents

Alpha1 antagonists

- Prazosin, doxazosin, tamsulosin, terazosin
  - Orthostatic hypotension
  - Treat benign prostatic hyperplasia (BPH), nightmares/post-traumatic stress disorder (PTSD)

Alpha2 adrenergic agonist

- Clonidine
  - Rebound hypertension!

Vasodilators

- Hydralazine
  - Can cause a lupus-like syndrome at high doses
  - Short half-life requires frequent dosing
- Minoxidil
  - Can treat alopecia
  - Should be used with diuretic and beta blockade

Fountain J, et al. Natl Lib Med. 2023

Organ-Specific Blood Pressure Considerations

Kidney

KDIGO 2009

- BP Goal <130/80 mmHg in adults
- Use any class of antihypertensive agent
- Monitor closely for adverse effects and drug-drug interactions
- When urine protein excretion is >1g/day for adults, consider an ACE-I or ARB for first-line therapy

ACC/AHA 2025

- Insufficient evidence to support specific BP targets or agents in kidney transplant recipients

KIDGO. Hypertension. 2009

Procedural or Surgical Interventions in Kidney Transplant

- Transplant renal artery angioplasty/stenting
- Treatment of obstructive sleep apnea with continuous positive airway pressure (CPAP)
- Bilateral native nephrectomy (failed native kidneys)
- Native renal denervation (sympathetic overactivity)

Tantisattamo E, et al. Front Med. 2020

Liver

American Association for the Study of Liver Diseases (AASLD) 2012 Guidelines

- Goal BP 130/80 mmHg
- Combination of lifestyle modifications and pharmacological agents
- ACEi, ARBs and direct renin inhibitors should be used as first-line therapy in liver transplant recipients with DM, CKD, and or significant proteinuria

Lucey M et al. Liver Transplantation 19(1):p 3-26, January 2013

Heart

ISHLT 2023

- BP Goal same as recommended for the general population
- Treatment should include recommendations for lifestyle modification in addition to drug therapy
- ACEi and CCB may be preferred as first-line therapy in patients with DM and as a cardiac allograft vasculopathy (CAV) prevention strategy
- HCTZ could be considered to specifically counteract CNI-induced hypertension
- CCBs should be considered the antihypertensive drug of choice when optimal blood pressure control cannot be achieved with ACEi/ARB, or when these drugs are contraindicated in HT recipients

Lung

Hyperlipidemia (HLD)

Circulation
Volume 153, Issue 17, 28 April 2026; Pages e1154-e1276
https://doi.org/10.1161/CIR.0000000000001423

CLINICAL PRACTICE GUIDELINES

2026 ACC/AHA/AACVPR/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/P Guideline on the Management of Dyslipidemia: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines

Risks

High risk for the development of post-transplant cardiovascular disease (CVD)

Atherosclerosis is accelerated after transplantation

- Can be linked to cardiovascular events

Calculating Risk

- PREVENT-ASCVD Calculator is now preferred

https://tools.acc.org/ascvd-risk-estimator-plus/#!/calculate/estimate/

The American Heart Association PREVENT™ Online Calculator

About the PREVENT Equations | Online Calculator

CVD | **ASCVD** | Heart Failure

Sex*
◉ Male ○ Female

Age (years)*
30-79

SBP (mmHg)*
90-200

Total Cholesterol (mg/dL)*
130-320

HDL Cholesterol (mg/dL)*
20-100

eGFR (mL/min/1.73m^2)*
15-140

BMI (kg/m^2)*
18.5-39.9

Diabetes
Any history of diabetes.
◉ No ○ Yes

Current Smoking
Any cigarette use within the last 30 days
◉ No ○ Yes

Lipid-lowering medication
Current use of statin medication to lower cholesterol
◉ No ○ Yes

Anti-hypertensive medication
Current use of any medication for hypertension
◉ No ○ Yes

The following three predictors are optional for further personalization of risk assessment. When they are clinically indicated or available,

If available or indicated, select "Yes" and enter the value.

UACR (mg/g)
UACR is clinically indicated for individuals with chronic kidney disease, diabetes, or hypertension
◉ No ○ Yes

HbA1C
HbA1c is clinically indicated for individuals with diabetes, prediabetes, overweight, or obesity, or those with history of gestational diabetes
◉ No ○ Yes

Zip Code
valid 5-digit zip code is needed to estimate social deprivation index [SDI]
◉ No ○ Yes

Calculate | Reset

Contributing Factors to HLD Post-transplant

- Genetic predisposition
- Age
- Excessive intake of cholesterol / saturated fats
- Obesity
- Proteinuria
- Corticosteroids
- CNIs
- Mammalian target-of-rapamycin inhibitors (mTORi)

Giles L. J Midw and Wom Hlth 2024

Lipoprotein Goals for ASCVD Risk Reduction

Patient population	LDL-C <100 mg/dL (2.6 mmol/L) Non–HDL-C <130 mg/dL (3.4 mmol/L)	LDL-C <70 mg/dL (1.8 mmol/L) Non–HDL-C <100 mg/dL (2.6 mmol/L)	LDL-C <55 mg/dL (1.4 mmol/L) Non–HDL-C <85 mg/dL (2.2 mmol/L)
Primary prevention	PREVENT-ASCVD <10% • If TG ≥150 mg/dL to 499 mg/dL, apoB goal: <90 mg/dL	PREVENT-ASCVD ≥10% • If TG ≥150 mg/dL to 499 mg/dL, apoB goal: <70 mg/dL	N/A
Severe hypercholesterolemia	**Without** FH, ASCVD risk factors, and subclinical atherosclerosis	**With** FH, ASCVD risk factors, or subclinical atherosclerosis	Severe hypercholesterolemia or HeFH with clinical ASCVD
Diabetes	**Without** ASCVD risk factors or diabetes-specific risk modifiers • apoB goal: <90 mg/dL	**With** ASCVD risk factors or diabetes-specific risk factors • apoB goal: <70 mg/dL	N/A
Subclinical atherosclerosis	CAC = 1–99 AU and <75th percentile for age, sex, and race	• CAC ≥100 to 299 AU or ≥75th percentile for age, sex, race • CAC ≥300 to 999 AU ◦ Optional goal: LDL-C <55 mg/dL, non–HDL-C <85 mg/dL and consider apoB goal <55 mg/dL	CAC ≥1000 AU
Hypertriglyceridemia	<50 y old with no additional risk enhancers	• With clinical ASCVD not at very high risk ◦ apoB goal: <70 mg/dL • Age 40–75 y with ≥1 ASCVD risk factor ◦ apoB goal: <70 mg/dL	With clinical ASCVD at very high risk • apoB goal: <55 mg/dL
Clinical ASCVD	N/A	Not at very high risk • Optional goal: LDL-C <55 mg/dL, non–HDL-C <85 mg/dL and consider apoB goal <55 mg/dL	• At very high risk ◦ apoB goal: <55 mg/dL • With CKD

Goals – Primary Prevention

Benefit Group	First Line Therapy Based on 10-year ASCVD Risk	Goal
Baseline LDL ≥190 mg/dL	Any Risk High Intensity statin	>50% reduction and LDL<100 mg/dL or non-HDL <130 mg/dL
Age 40-75 years old with DM, baseline LDL <190mg/dL	**< 7.5%:** Moderate intensity statin	≥30-49% LDL reduction and LDL <100 or non-HDL <130 mg/dL
	>7.5%: risk enhancers, or subclinical atherosclerosis: high intensity statin	>50% reduction and LDL<100 mg/dL or non-HDL <130 mg/dL
	>20%: High intensity statin	>50% LDL reduction and LDL <70 or non-HDL <100
Age 40-75 years old without DM or ASCVD with LDL 70-189 mg/dL	**<5%:** Risk discussion	Lifestyle Modification
	5-7.4%: Consider moderate intensity statin	30-49% LDL reduction and LDL <100 mg/dL
	7.5 – 19.9%: Moderate intensity statin	30-49% LDL reduction and LDL <100 mg/dL
	≥20%: High intensity statin	>50% LDL reduction and LDL <70

Lloyd-Jones D, et al. JACC. 2022

Goals - Secondary Prevention

Benefit Group	First Line Therapy	Goal
ASCVD at high risk	High intensity statin	≥50% reduction in LDL and LDL <55 mg/dL or non–HDL <85 mg/dL
ASCVD not at very risk	High intensity statin	≥50% reduction in LDL and LDL <70 mg/dL or non–HDL <100 mg/dL
ASCVD and LDL ≥190 mg/dL	High intensity statin	≥50% reduction in LDL and LDL <70 mg/dL or non–HDL <100 mg/dL

Lloyd-Jones D, et al. JACC. 2022

Lipoprotein (a)

LDL-like particle that is structurally distinct from LDL

Concentrations are mostly genetically determined

- Population distribution varies by ancestry

Robust associations for LP(a) with ASCVD outcomes and calcific aortic valve disease

- ASCVD risk increases as LP(a) levels increase, regardless of ancestry

LP(a) >125 nmol/L (50 mg/dL) is considered elevated

- Associated with ~40% relative risk increase in ASCVD

LP(a) concentrations remain similar over time and are minimally modified by lifestyle factors

- A single measurement is generally sufficient and helps with risk stratification

Yadalam et al. J. Am. Coll. Cardiol., 2024

Hyperlipidemia Treatment

Lifestyle Modification

DIET

EXERCISE

SMOKING CESSATION

ALCOHOL REDUCTION / CESSATION

Giles L. J Midw and Wom Hlth 2024

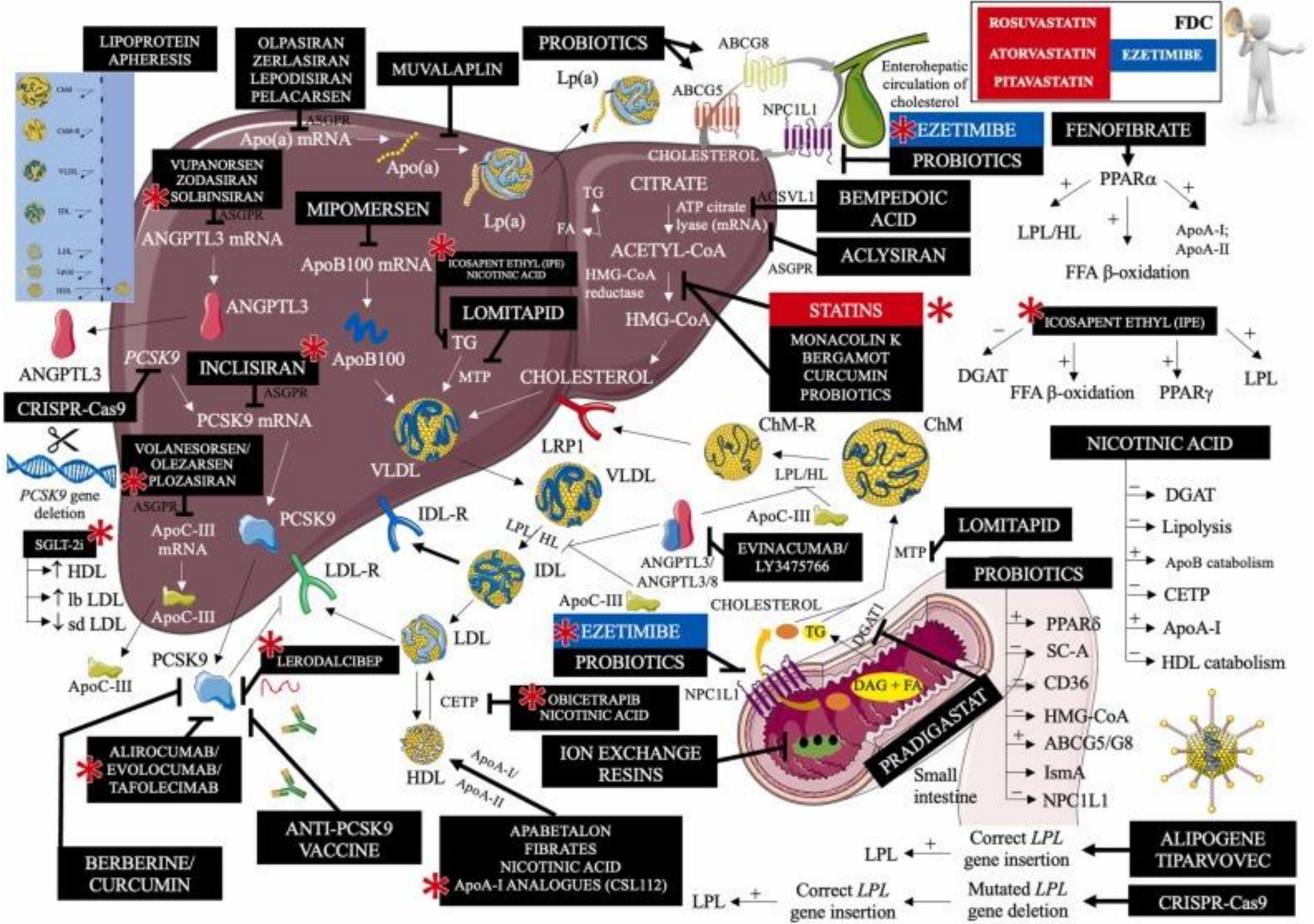
Pharmacologic Therapy

First-line

- Hydroxymethylglutaryl-CoA (HMG-CoA) reductase inhibitors

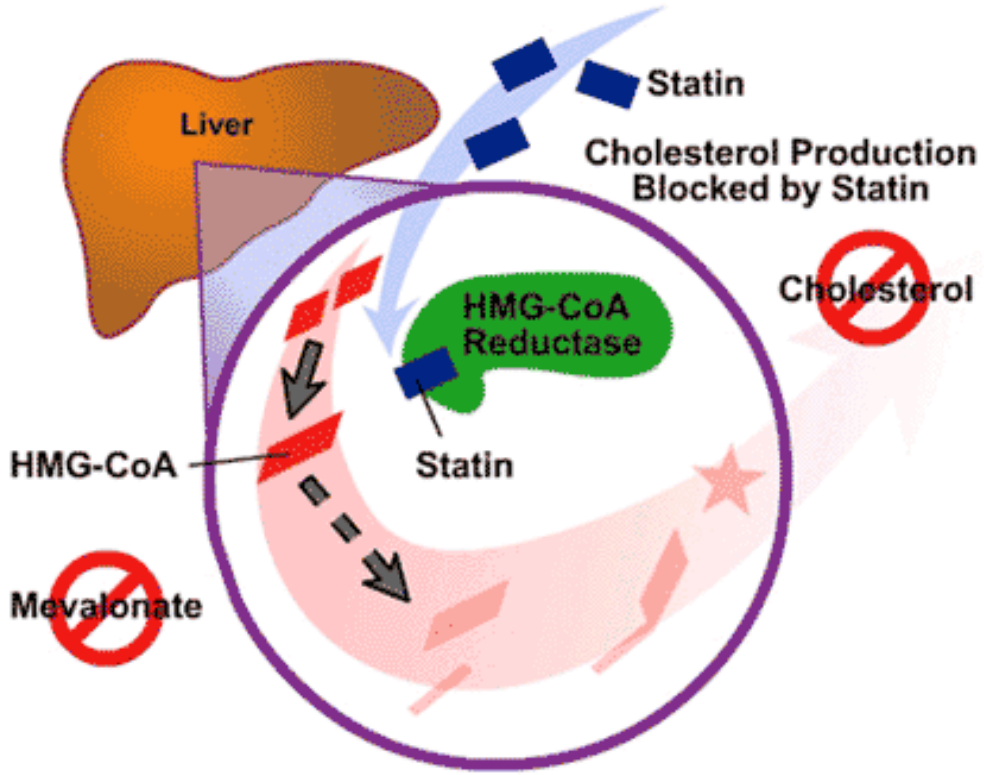
If Goals not met:

- Ezetimibe
- Bile acid sequestrants
- Niacin
- Fibrates
- Proprotein convertase subtilisin-kexin type 9 (PCSK9) inhibitors
- Microsomal triglyceride transfer protein (MTP) inhibitors
- Angiopoietin-like protein 3 (ANGPTL3)
- Antilipemic Small Interfering Ribonucleic Acid (siRNA) Agent


LIPOPROTEIN APHERESIS
OLPASIRAN ZERLASIRAN LEPODISIRAN PELACARSEN
MUVALAPLIN
PROBIOTICS
ABCG8
ABCG5
NPC1L1
Enterohepatic circulation of cholesterol
ROSUVASTATIN
ATORVASTATIN
PITAVASTATIN
FDC
EZETIMIBE
EZETIMIBE
PROBIOTICS
FENOFIBRATE
PPARα
LPL/HL
ApoA-I; ApoA-II
FFA β-oxidation
Lp(a)
Apo(a) mRNA
Apo(a)
ASGPR
VUPANORSEN ZODASIRAN SOLBINSIRAN
ANGPTL3 mRNA
MIPOMERSEN
ApoB100 mRNA
ICOSAPENT ETHYL (IPE) NICOTINIC ACID
LOMITAPID
CHOLESTEROL
CITRATE
ATP citrate lyase (mRNA)
ACSVL1
BEMPEDOIC ACID
ACLYSIRAN
TG
FA
ACETYL-CoA
HMG-CoA reductase
HMG-CoA
STATINS
MONACOLIN K
BERGAMOT
CURCUMIN
PROBIOTICS
ICOSAPENT ETHYL (IPE)
DGAT
FFA β-oxidation
PPARγ
LPL
ANGPTL3
PCSK9
INCLISIRAN
ApoB100
MTP
CRISPR-Cas9
PCSK9 mRNA
PCSK9 gene deletion
VOLANESORSEN/ OLEZARSEN PLOZASIRAN
ApoC-III mRNA
VLDL
LRP1
ChM-R
ChM
NICOTINIC ACID
SGLT-2i
↑ HDL
↑ lb LDL
↓ sd LDL
IDL-R
LDL-R
IDL
LPL/HL
ApoC-III
EVINACUMAB/ LY3475766
ANGPTL3/ ANGPTL3/8
LOMITAPID
MTP
PROBIOTICS
DGAT
Lipolysis
ApoB catabolism
CETP
ApoA-I
HDL catabolism
LDL
EZETIMIBE
PROBIOTICS
CHOLESTEROL
TG
DGAT1
DAG + FA
NPC1L1
PRADIGASTAT
PPARδ
SC-A
CD36
HMG-CoA
ABCG5/G8
IsmA
NPC1L1
Small intestine
LERODALCIBEP
CETP
OBICETRAPIB NICOTINIC ACID
ION EXCHANGE RESINS
HDL
ApoA-I/ ApoA-II
ALIROCUMAB/ EVOLOCUMAB/ TAFOLECIMAB
ANTI-PCSK9 VACCINE
APABETALON FIBRATES NICOTINIC ACID ApoA-I ANALOGUES (CSL112)
BERBERINE/ CURCUMIN
LPL
Correct LPL gene insertion
Mutated LPL gene deletion
ALIPOGENE TIPARVOVEC
CRISPR-Cas9

HMG CoA Reductase Inhibitors

- Lovastatin, simvastatin, and atorvastatin are metabolized via CYP3A4
- All statins are substrates of OATP1B1 transporter
  - Using them with CNIs increases statin concentrations
  - Increased risk of myalgias / rhabdomyolysis
- Cholesterol synthesis occurs at night, so statins should be administered in the evening



Bansal A, et al. *HMG CoA Reductase Inhibitors*. Natl Lib Med. 2023

HMG CoA Reductase Inhibitors

Drug	Low Intensity 20-25% ↓	Moderate Intensity 30-49% ↓	High Intensity ≥50% ↓	Clinical Pearls
Lovastatin	10-20 mg	40-80 mg		Metabolized via CYP3A4 Short half-life
Pravastatin	10-20 mg	40-80 mg		
Simvastatin	10 mg	20-40 mg		Metabolized via CYP3A4 Can cause kidney injury Short half-life
Fluvastatin	20-40 mg	80 mg		Short half-life
Pitavastatin		1-4 mg		
Atorvastatin	5 mg	10-20 mg	40-80 mg	Metabolized via CYP3A4 Long half life
Rosuvastatin		5-10 mg	20-40 mg	Can cause kidney injury Long half life

Bansal A, et al. *HMG CoA Reductase Inhibitors*. Natl Lib Med. 2023

Non-statin therapies

Drug Class	Mechanism of Action	LDL-C Lowering	Side Effects
Ezetimibe	- Reduces cholesterol absorption in the small intestine - Raise LDL receptor activity	15-25%	GI
Bempedoic acid	- Decreases cholesterol production upstream of HMG-Co reductase - Increases LDL receptor expression on hepatocytes	15-25%	Hyperuricemia Tendon rupture
PCSK9 inhibitors: (alirocumab, evolocumab)	- Inhibits PCSK9 to increase the number of LDL receptors available to clear circulating LDL-C	45%-64%	Headache, injection-site reaction
Inclisiran	- Antilipemic Small Interfering Ribonucleic Acid (siRNA) Agent - Inhibits PCSK9 production in the liver	48% - 52%	Injection site reactions
Bile acid sequestrants	- Impairs absorption of cholesterol - Raise LDL receptor activity	15-25% depending on dose	Constipation GI Increases TG *Contraindicated with MMF *Decreases statin concentration

Grundy S et al. *Guidelines for the Management of High Blood Cholesterol.* Natl Lib Med 2022

Triglyceride-Lowering therapies

Drug Class	Mechanism of Action	TG Lowering	Side Effects
Omega-3 fatty acids	- Reduces secretion of VLDL - Enhances TG secretion	15-61%	
Fibrates (fenofibrate, gemfibrozil)	- Reduces secretion of VLDL - Enhances degradation of VLDL	30-50%	Myopathy (in combination with statins)
Niacin	- Reduces hepatic TG esterification - Decreases release of free fatty acids from adipose tissue	10-50%	Flushing Hepatotoxicity Increased insulin resistance

PCSK9 Inhibitors

Agent	Dosing	Adverse Effects	Other Considerations
Evolocumab (Repatha®)	140mg SQ every 2 weeks	Injection site reactions Angioedema (rare) Potential for immunogenicity Flu-like illness	Avoid with latex allergy
Alirocumab (Praluent®)	75mg SQ every 2 weeks		May cause LFT elevation
Lerodalcibep	300mg SQ monthly		Third generation

PCSK9i in SOT

Warden B, et al. J Am Coll Cardiol Case Rep 2020

CASE REPORT: CLINICAL CASE SERIES

Use of PCSK9 Inhibitors in Solid Organ Transplantation Recipients

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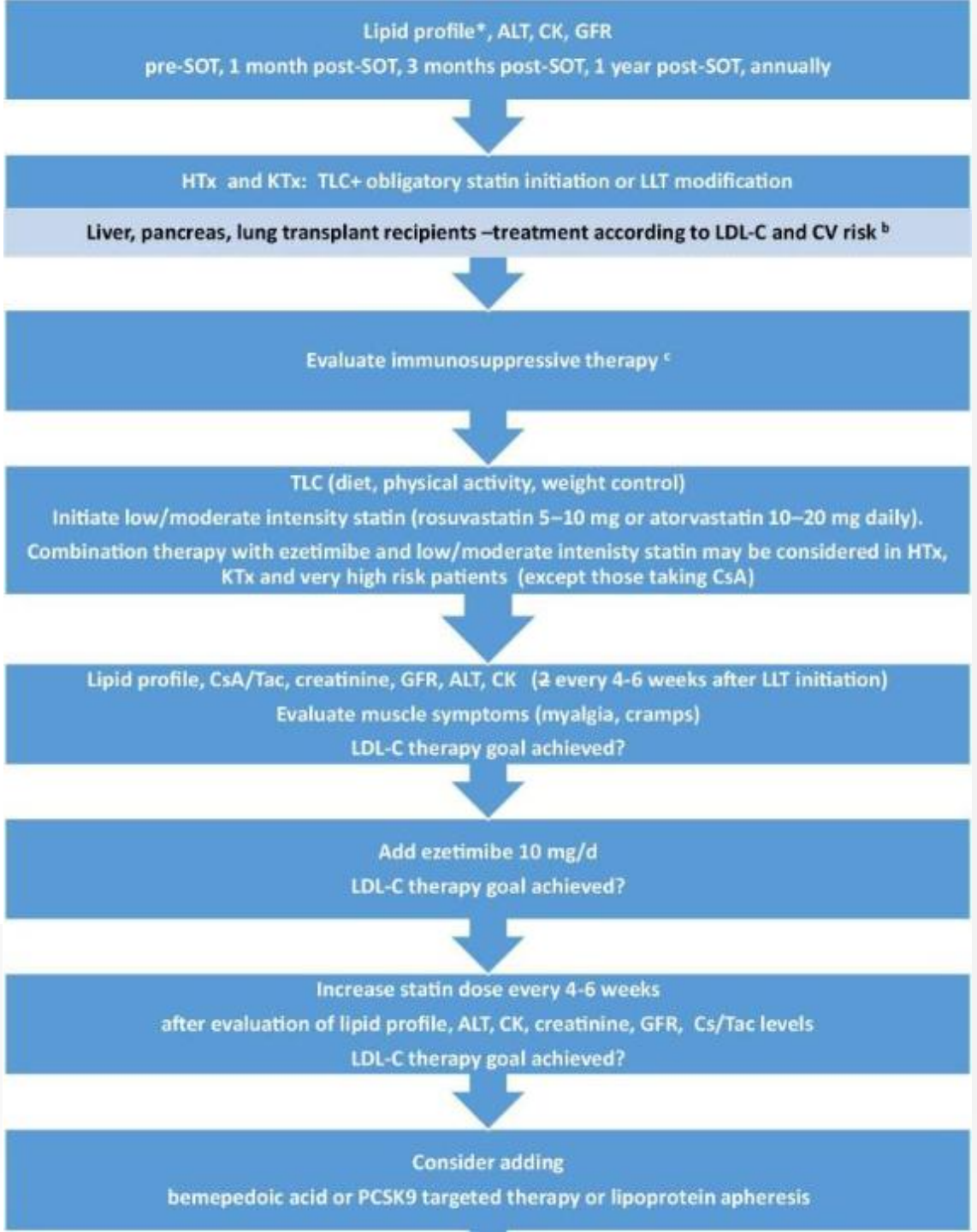
Results

- Case series of 12 SOT recipients with inadequately treated HLD with standard lipid lowering therapy
  - 75% heart transplant
  - Kidney, liver, and lung also represented
- Indications were familial HLD (50%), ASCVD risk (75%) and CAV (18%)
- Median LDL-C levels decreased 60%
- Median CSA levels decreased (37%), tacrolimus increased (6%)
  - Proportion of time within therapeutic range did not change
- 25% of patients experienced mild, self-limiting adverse reactions
  - Rhinorrhea, injection site reactions, nausea
  - No discontinuation

Warden B, et al. J Am Coll Cardiol Case Rep 2020

Lipid Management in SOT



Mickiewicz et al. Nephrol Dial Transplant. 2025.

Organ Specific Considerations

Kidney

- KDIGO 2013
  - Statin treatment for all adult kidney transplant recipients (KTRs), except those aged <30 years of age and without prior cardiovascular risk factors (CVRF)

KIDGO. Hypertension. 2013

Heart

- ISHLT 2023
  - In adults, the use of statins after HT is recommended regardless of cholesterol levels. Due to pharmacologic interactions with CNI and risk for toxicity, statin doses should generally be lower than those recommended for hyperlipidemia.
  - Although there is no evidence for a target LDL concentration in these patients, it is reasonable to aim for level below 100 mg/dL (or 2.5 mmol/L) for most patients, with more aggressive targets reserved for those with evidence of CAV
  - PCSK9 inhibitors are reasonable adjuncts to statins in adult heart transplant patients with uncontrolled hyperlipidemia or as alternative agents in the setting of statin intolerance

Velleca A, et al. JHLT. 2023

Summary

- Refer to organ specific guidance (where available) for hypertension and lipid management
- Use medication clinical pearls to help guide difficult clinical decision making / agent selection
- Employ newer agents in cases where tolerability or efficacy is unattainable with first-line agents