

POST TRANSPLANT HYPERTENSION

DR.VILESH VALSALAN

Consultant Nephrologist and Transplant physician

Academic Coordinator – ECNG

BP TARGETS - GUIDELINES

- **2017** American College of Cardiology/ American Heart Association (**ACC/AHA**) guidelines for office and ambulatory BP monitoring (ABPM) measurements were $\geq 130/80$ mmHg and $\geq 125/75$ mmHg, respectively.
- **2018** European Society of Cardiology/European Society of Hypertension (**ESC/ESH**) guidelines were office BP $\geq 140/90$ mmHg and ABPM $\geq 130/80$ mmHg.
- **2021** Kidney Disease Improving Global Outcomes [**KDIGO**] BP guidelines , hypertension was defined as office BP $\geq 130/80$ mmHg and ABPM $\geq 125/75$ mmHg

- **Office blood pressure (OBP)** :- The mean of three non-invasive BP measurements.
- **Home blood pressure monitoring (HBPM)** :- Recording at least twice the daily average of two home blood pressure readings over a minimum of 4 days.
- **24-h ambulatory blood pressure monitoring (24-h ABPM)**:- which requires wearing an electronic blood pressure measurement device to record and averages multiple readings over a 24 h period.

Risk Factors

Kidney Transplant Function Factors

- Decreased GFR
- Extracellular volume expansion
- Increased activity of RAAS
- Enhanced activity of the sympathetic nervous system
- Secondary hyperparathyroidism
 - Increase in intracellular calcium concentration
- Impaired nitric oxide synthesis and endothelium-mediated vasodilatation
- Treatment with erythropoietin
- Transplant renal artery stenosis
- Dietary potassium restriction

Donor Risk Factors

- Older donor age
- Donor hypertension
- Low donor/recipient body weight ratio

Female donor
Male recipient

Post-Kidney Transplant Hypertension

Traditional Risk Factors

- High salt intake
- Low potassium intake
- Alcohol use
- Obesity
- Physical inactivity
- Tobacco use

Immunosuppression Related Factors

- Use of calcineurin inhibitors
 - Arteriolar constriction
 - Impaired Na and H₂O excretion
 - Activation of NaK₂Cl and NaCl co-transporters
 - Decrease in GFR
 - Increase in peripheral vascular resistance
- Use of mammalian target of rapamycin (mTOR) inhibitors
 - Increased salt retention
- Use of corticosteroids
 - Excess Na and H₂O reabsorption by the renal mineralocorticoid receptor
 - Weight gain

Blood Pressure Goals and Outcomes in Kidney Transplant Recipients in an Analysis of the Collaborative Transplant Study



Methods and cohort

 Collaborative Transplant Study database

 1-year blood pressure ACC/AHA classification
209 centers
39 countries

 Kidney transplant recipients
N = 62,556

At one-year post-transplant



77% of kidney transplant recipients had hypertension



Hypertension stages 1 and 2 were associated with an 11% and 55% increased risk of death-censored graft failure

SBP values consistently lower than 140 mmHg during the first 3 years post transplantation were associated with the best 10-year graft and patient outcomes;

Lowering SBP to \leq 140 mmHg even by the 3rd year was associated with better 10-year graft and death-censored survival

Timelines and causes of HTN in post KTR patients

Immediate post-transplantation

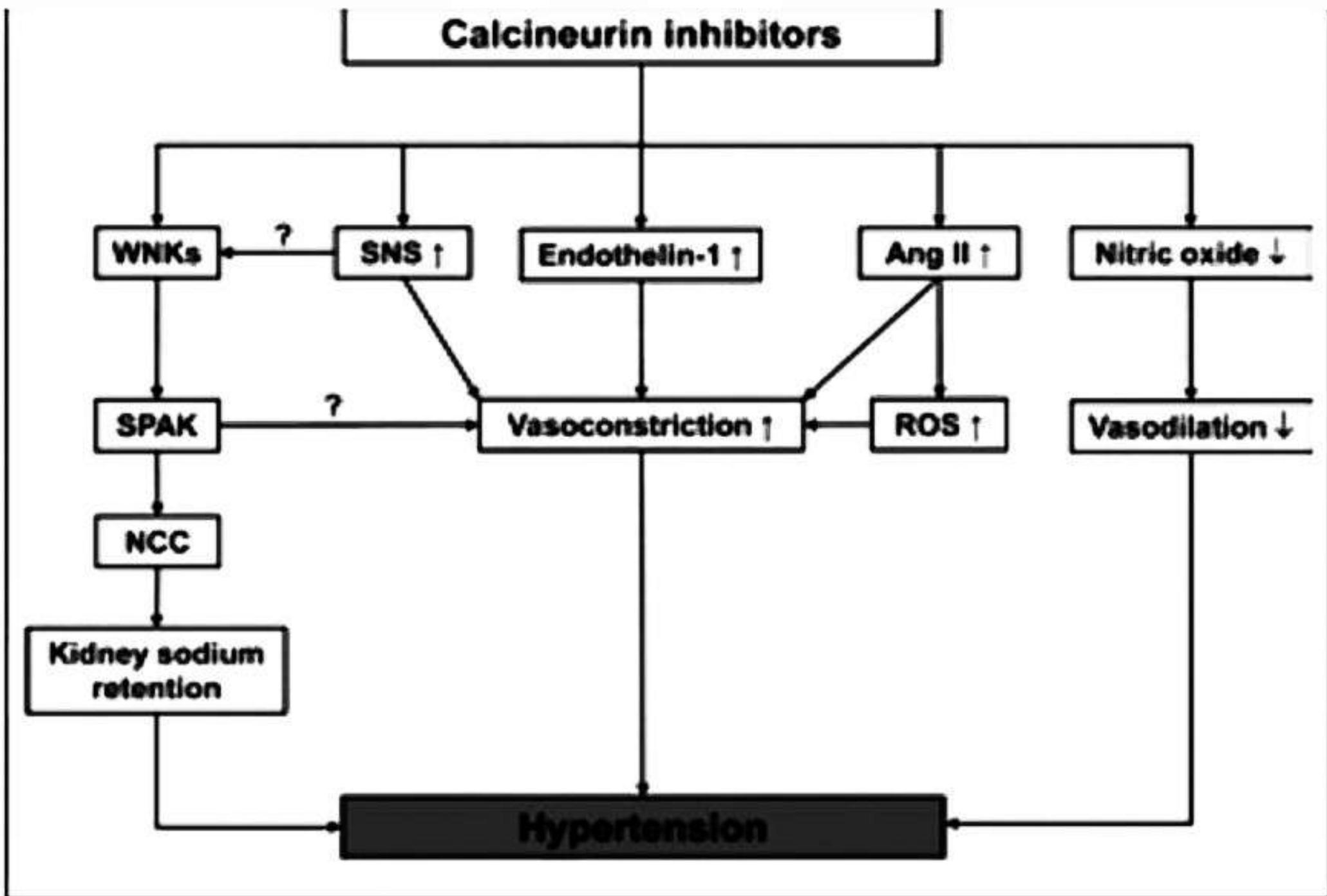
- ❖ *Peri-transplant hypervolemia*
- ❖ *Induction immunosuppressive medications*
- ❖ *Rebound hypertension*
- ❖ *Inadequate pain control*

Early post-transplantation

- ❖ *Weight gain*
- ❖ *Calcineurin inhibitors*
- ❖ *Steroids*
- ❖ *Hypertensive donor kidney*
- ❖ *Transplant renal artery stenosis*

Late post-transplantation

- ❖ *Chronic renal allograft dysfunction*
- ❖ *Fibroblast growth factor 23*
- ❖ *Obstructive sleep apnea*
- ❖ *Failed native kidneys*



Anti hypertensive medications- key points

- **Thiazides** may be considered for kidney transplant recipients with CNI-induced salt-sensitive HTN and hypomagnesemia.
- **Volume control rather than BP control** is the indication for **loop diuretics** in kidney transplant recipients during immediate and early post-transplant periods.
- **Beta-blocker** acts by mitigation of the sympathetic nervous system and decrease proinflammatory cytokines decreasing risk for atherosclerosis.
- **CCBs** counteract the systemic and renal vasoconstrictive effect of CNI via endothelin I. CCBs also prevent post-transplant acute tubular injury / DGF.
- No robust evidence for CV or survival advantages with the use of **ACEIs, ARBs, and MCRAs**, but considered in transplant recipients with **LVH, congestive heart failure, and proteinuria**.

Summary

