Pharmacotherapy of Hypertension

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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.

HTN & Comorbidities

- Cerebrovascular Disease
- Valvular Heart Disease
- Aortic Disease
- Diabetes Mellitus
- Chronic Kidney Disease
- Hemodialysis Patients
- After Kidney Transplant
- Heart Failure
- Ischemic Heart Disease

Cerebrovascular Disease & HTN

Acute ischemic stroke

Acute Intracerebral Hemorrhage

Secondary Stroke Prevention

Acute ischemic stroke & HTN

بیمار آقای 68 ساله با تشخیص سکته مغزی ایسکمی در فاز حاد در بیمارستان بستری می باشد و کاندید دریافت آلتپلاز می باشد. بیمار در PMH سابقه فشار خون بالا ، سیگار ، دیس لیپیدمی و چاقی را دارد.
علائم حیاتی بیمار در حال حاضر به شرح زیر است:

BP = 230/110 mmHg, HR= 105/min , RR= 20/min T=37

اقدام درمانی برای کنترل فشار خون بیمار چیست؟

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علائم حیاتی بیمار در حال حاضر به شرح زیر است:

BP = 180/100 mmHg, HR= 105/min , RR= 20/min T=37

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Acute ischemic stroke

Treatment of hypertension in acute ischemic stroke is dependent on the following conditions:

1) Treatment with IV thrombolysis

2) Treatment with endovascular thrombectomy with successful reperfusion,

3) Patients who are not treated with thrombolytic therapy with SBP >220 or DBP>120 mmHg

4). Patients who are not treated with thrombolytic therapy with SBP <220 or DBP<120 mm Hg

5) Comorbid conditions requiring treatment (eg, concomitant acute coronary event, acute HF, aortic dissection, post fibrinolysis symptomatic ICH, or preeclampsia/eclampsia).

Treatment with IV thrombolysis & endovascular thrombectomy

Options to treat hypertension before and during reperfusion therapy for acute ischemic stroke

Patient otherwise eligible for acute reperfusion therapy except that blood pressure is >185/110 mmHg*
Labetalol 10 to 20 mg intravenously over 1 to 2 minutes, may repeat one time; **or**
Nicardipine 5 mg/hour intravenously, titrate up by 2.5 mg/hour every 5 to 15 minutes, maximum 15 mg/hour; when desired blood pressure reached, adjust to maintain proper blood pressure limits; **or**
Clevidipine 1 to 2 mg/hour intravenously, titrate by doubling the dose every 2 to 5 minutes, maximum 21 mg/hour, until desired blood pressure reached¶; **or**
Other agents (hydralazine, enalaprilat, etc) may also be considered
If blood pressure is not maintained at or below 185/110 mmHg, do **not** administer alteplase
Management to maintain blood pressure at or below 180/105 mmHg during and after acute reperfusion therapy*
Monitor blood pressure every 15 minutes for 2 hours from the start of rtPA therapy, then every 30 minutes for 6 hours, and then every hour for 16 hours
If systolic blood pressure is >180 to 230 mmHg or diastolic is >105 to 120 mmHg:
Labetalol 10 mg intravenously followed by continuous infusion 2 to 8 mg/min; **or**
Nicardipine 5 mg/hour intravenously, titrate up to desired effect by 2.5 mg/hour every 5 to 15 minutes, maximum 15 mg/hour; **or**
Clevidipine 1 to 2 mg/hour intravenously, titrate by doubling the dose every 2 to 5 minutes, maximum 21 mg/hour, until desired blood pressure reached¶
If blood pressure is not controlled or diastolic blood pressure >140 mmHg, consider intravenous sodium nitroprusside

* Different treatment options may be appropriate in patients who have comorbid conditions that may benefit from acute reductions in blood pressure, such as acute coronary event, acute heart failure, aortic dissection, or preeclampsia/eclampsia.
¶ Clevidipine has been included as part of the 2018 guidelines for the early management of patients with acute ischemic stroke[1].

Patients with SBP <220 mm Hg or DBP <120 mm Hg

For patients with ischemic stroke who are not treated with thrombolytic therapy, and do not have comorbid condition , blood pressure should not be treated acutely unless the hypertension is extreme (> 220/120 mmHg) and initiation treatment of HTN within 48 to 72 hr after acute ischemic stroke is not effective to prevent death and disability

Pathophysiology of HTN in Acute Stroke

when ICP > CVP ： CPP = MAP – ICP

There are four causes of increased ICP following ischemic stroke:

(1) brain swelling due to cytotoxic or vasogenic edema

(2) increased cerebral blood volume due to dilated cerebral arteries;

(3) obstruction of venous outflow

(4) acute hydrocephalus

The arterial blood pressure is usually elevated in patients with an acute stroke. This may be due to chronic hypertension, an acute sympathetic response, or other stroke-mediated mechanisms. In many cases, however, the acutely elevated blood pressure is necessary to maintain brain perfusion in borderline ischemic areas.

Patients who are not treated with thrombolytic therapy with BP>220/120 mmHg

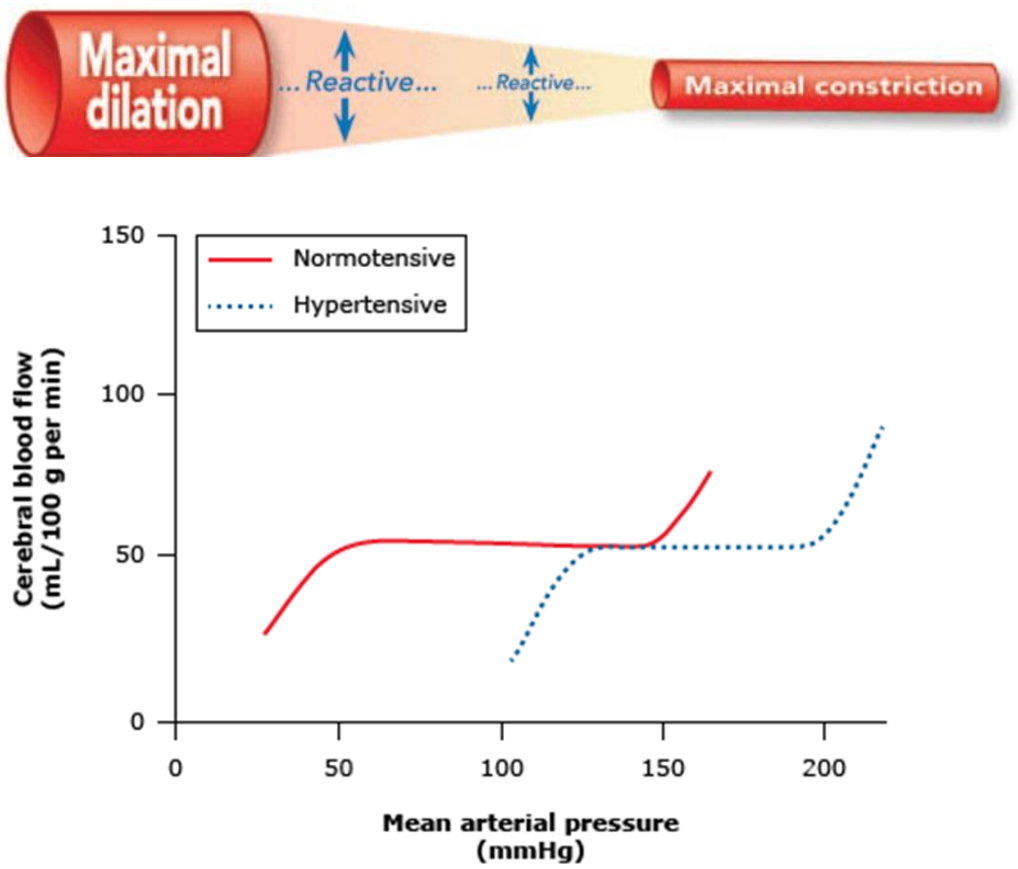
For patients with ischemic stroke who are not treated with thrombolytic therapy, blood pressure should not be treated acutely unless the hypertension is extreme (systolic blood pressure >220 mmHg or diastolic blood pressure >120 mmHg). When treatment is indicated, cautious lowering of blood pressure by approximately 15 percent during the first 24 hours after stroke onset is suggested.

When we can reach to goal BP after Acute Ischemic Stroke

When patient is neurologically stable (24 to 48 hours after stroke onset for most hospitalized) can reach to goal BP (140/90 mmHg) gradually within a few days to a week

Patients with extracranial or intracranial large artery stenoses may require a slower reduction in blood pressure (eg, over 7 to 14 days after ischemic stroke), as some degree of blood pressure elevation may be necessary to maintain cerebral blood flow to ischemic brain regions.

For this reason, we suggest not restarting antihypertensive agents until after vascular imaging is completed and a symptomatic large artery stenosis is excluded.



Schematic representation of autoregulation of cerebral blood flow in normotensive and hypertensive subjects. In both groups, initial increases or decreases in mean arterial pressure are associated with maintenance of cerebral blood flow due to appropriate changes in arteriolar resistance. More marked changes in pressure are eventually associated with loss of autoregulation, leading to a reduction (with hypotension) or an elevation (with marked hypertension) in cerebral blood flow. These changes occur at higher pressures in patients with hypertension, presumably due to arteriolar thickening. Thus, aggressive antihypertensive therapy will produce cerebral ischemia at a higher mean arterial pressure in patients with underlying hypertension

Choice of antihypertensive agent

- Consensus guidelines suggest intravenous labetalol, nicardipine, and clevidipine as first-line antihypertensive agents if pharmacologic therapy is necessary in the acute phase, since they allow rapid and safe titration to the goal blood pressure.

- Intravenous nitroprusside & TNG should be considered second-line therapy since it carries added theoretical risks of increasing intracranial pressure (ICP) or affecting platelet function.

- Medications likely to cause a prolonged or precipitous decline in blood pressure (eg, rapid-acting formulations of nifedipine) should be avoided. In addition, their use is associated with an increased risk of stroke, particularly in older adult patients

Acute ischemic stroke

Treatment of hypertension in acute ischemic stroke is dependent on the following conditions:

1) Treatment with IV thrombolysis

2) Treatment with endovascular thrombectomy with successful reperfusion,

3) Patients who are not treated with thrombolytic therapy with SBP >220 or DBP>120 mmHg

4). Patients who are not treated with thrombolytic therapy with SBP <220 or DBP<120 mm Hg

5) Comorbid conditions requiring treatment (eg, concomitant acute coronary event, acute HF, aortic dissection, post fibrinolysis symptomatic ICH, or preeclampsia/eclampsia).

5.3.9.2. Acute Ischemic Stroke

Recommendations for Acute Ischemic Stroke References that support recommendations are summarized in the Evidence Table.		
COR	**LOE**	**Recommendations**
1	C-LD	1. In patients with acute ischemic stroke, hypotension and hypovolemia should be corrected to maintain systemic perfusion levels necessary to support organ function.[1–3]
1	B-NR	2. Patients who have elevated BP and are otherwise eligible for treatment with IV thrombolytics should have their BP lowered to SBP <185 mm Hg and DBP <110 mm Hg before IV thrombolytic therapy is initiated and should be maintained below 180/105 mm Hg for at least the first 24 hours after initiating thrombolytic therapy to avoid complications.[4,5]
2a	B-NR	3. In patients who undergo endovascular treatment, it is reasonable to maintain the BP at ≤180/105 mm Hg during and for 24 hours after the procedure to improve long-term functional outcomes and prevent death.[6,7]
2b	C-LD	4. In patients with BP of ≥220/120 mm Hg who did not receive IV thrombolytic or endovascular treatment and have no comorbid conditions requiring acute antihypertensive treatment, it might be reasonable to lower BP by 15% during the first 24 hours after onset of stroke to improve outcomes.[2,3]

Recommendations for Acute Ischemic Stroke (Continued)		
COR	**LOE**	**Recommendations**
3: No Benefit	**A**	5. In patients with BP <220/120 mm Hg who do not receive IV thrombolysis or endovascular treatment and do not have a comorbid condition requiring urgent antihypertensive treatment, initiating or reinitiating treatment of hypertension within the first 48 to 72 hours after an acute ischemic stroke is not effective to prevent death or disability.[8–11]
3: Harm	**A**	6. In patients undergoing successful brain reperfusion with endovascular treatment for a large vessel occlusion, lowering SBP <140 mm Hg within the first 24 to 72 hours after reperfusion can worsen long-term functional outcome.[12–14]

Acute hemorrhagic stroke & HTN

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Acute hemorrhagic stroke & HTN

- Elevated blood pressure is common in patients with acute ICH. Patients may develop elevated blood pressure due to an increase in intracranial pressure (ICP) and pain from the mass effect of the hemorrhage. Additionally, many patients with acute ICH have high blood pressure due to comorbid baseline hypertension.

- Uncontrolled elevations in blood pressure and blood pressure variability are risk factors for hemorrhagic expansion , neurological worsening, poor outcome and death.

Patients with SBP 150- 220 mm Hg

For patients with acute ICH who present with systolic blood pressure (SBP) between 150 and 220 mmHg, we suggest lowering of SBP to a target of 140 mmHg, ideally within the first one hour of presentation, provided the patient remains clinically stable. This degree of blood pressure reduction appears safe in most patients and may improve functional outcome.

Patients with SBP > 220 mm Hg

For patients with acute ICH who present with SBP >220 mmHg, we suggest rapid lowering of SBP to <220 mmHg. Thereafter, the blood pressure is gradually reduced (over a period of hours) to a target range of 140 to 160 mmHg, provided the patient remains clinically stable.

Patients who deteriorate clinically during this period may require reduction of acute antihypertensive therapy. Blood pressure is monitored every five minutes and patients are monitored at least hourly to assess for neurologic deterioration.

The optimal blood pressure goal is uncertain, but an SBP of 140 to 160 mmHg is a reasonable target for patients who remain clinically stable.

Choice of antihypertensive agent

- For most patients with an initial SBP ≥160 mmHg, we prefer nicardipine for initial treatment because it is fast-acting and can be quickly titrated.

- For most patients with an initial SBP <160 mmHg, we start with labetalol for its ease of administration and long duration of effect.

- Several intravenous medications may be used to control blood pressure in this setting
- including nicardipine, labetalol, clevidipine, esmolol, and enalaprilat .

- Nitroprusside and nitroglycerin are typically avoided because they may increase ICP

5.3.9.1. Acute Intracerebral Hemorrhage

Recommendations for Acute Intracerebral Hemorrhage		
COR	**LOE**	**Recommendations**
2a	**A**	1. For adult patients with acute spontaneous intracerebral hemorrhage (ICH) who present with SBP between 150 and 220 mm Hg, it can be beneficial to immediately lower SBP to 130 to <140 mm Hg for at least 7 days after ICH to improve functional outcomes but stop antihypertensive medications if SBP <130 mm Hg.[1–3]
2a	**B-NR**	2. In adults with acute spontaneous ICH requiring acute BP lowering, careful titration to ensure smooth, nonlabile, and sustained control of BP, avoiding peaks and large variability in SBP, can be beneficial for improving functional outcomes.[3,4]
3: Harm	**B-NR**	3. For adult patients with acute spontaneous ICH who present with SBP >220 mm Hg, SBP should not be lowered below 130 mm Hg to reduce adverse events.[5–7]

Drug	Dose range	Onset of action (minutes)	Duration of action (minutes)	Adverse effects[¶]	Role[Δ]
Vasodilators					
Clevidipine	Initially 1 to 2 mg/hour as IV infusion with rapid titration. Most patients respond to 4 to 6 mg/hour and are treated with maximum doses of 21 mg/hour or less. NOTE: Delivered in lipid emulsion. 1000 mL maximum per 24 hours (equivalent to 21 mg/hour) due to lipid load.	2 to 4	5 to 15	Atrial fibrillation, nausea, lipid formulation contains potential allergens (eg, soy, egg)	Hypertensive emergencies including postoperative hypertension.
Enalaprilat	1.25 to 5 mg every 6 hours IV	15 to 30	Approximately 6 to >12 hours	Precipitous fall in pressure in high-renin states; variable response, headache, dizziness	Acute left ventricular failure. Due to slow onset and long duration of effect, rarely used. Avoid use in AMI, kidney function impairment, or pregnancy.
Fenoldopam	Initially 0.1 mcg/kg per minute[◇] as IV infusion titrated to a maximum of 1.6 mcg/kg per minute	5 to 10	30 to 60	Tachycardia, headache, nausea, flushing	Most hypertensive emergencies. Use caution or avoid with glaucoma or increased intracranial pressure.
Hydralazine	10 to 20 mg IV	10 to 20 IV	1 to ≥4 hours IV	Sudden precipitous drop in blood pressure, tachycardia, flushing, headache, vomiting, aggravation of angina	In general, hydralazine should be avoided due to its prolonged and unpredictable hypotensive effect. Labetalol and nicardipine are generally preferred choices for treatment of eclampsia.
	10 to 20 mg IM (40 mg maximum per labeling)	20 to 30 IM	4 to 6 hours IM		
Nicardipine	5 to 15 mg/hour as IV infusion. Some patients may require up to 30 mg/hour.	5 to 15	approximately 1.5 to ≥4 hours	Tachycardia, headache, dizziness, nausea, flushing, local phlebitis, edema	Most hypertensive emergencies, including pregnancy induced. Avoid use in acute heart failure. Caution with coronary ischemia.
Nitroglycerin (glyceryl trinitrate)	5 to 100 mcg/minute as IV infusion	2 to 5	5 to 10	Hypoxemia, tachycardia (reflex sympathetic activation), headache, vomiting, flushing, methemoglobinemia, tolerance with prolonged use	Potential adjunct to other IV antihypertensive therapy in patients with coronary ischemia (ACS) or acute pulmonary edema.
Nitroprusside	0.25 to 10 mcg/kg per minute as IV infusion. To minimize risk of cyanide toxicity, infusion duration should be as short as possible and generally not exceed 2 mcg/kg per minute. Use of maximal dose (ie, 8 to 10 mcg/kg per minute) should not exceed 10 minutes. Patients who receive higher doses (ie, >500 mcg/kg at a rate exceeding 2 mcg/kg per minute) should receive sodium thiosulfate infusion to avoid cyanide toxicity.	0.5 to 1	1 to 10	Elevated intracranial pressure, decreased cerebral blood flow, reduced coronary blood flow in CAD, cyanide and thiocyanate toxicity, nausea, vomiting, muscle spasm, flushing, sweating	Due to its toxicity, nitroprusside should generally be avoided if preferred agents are available. Nitroprusside should be avoided in patients with AMI, CAD, CVA, elevated intracranial pressure, kidney function impairment, or hepatic impairment.

Adrenergic inhibitors					
Esmolol	500 mcg/kg is typical loading dose given over 1 minute; then initiate IV infusion at 25 to 50 mcg/kg per minute; titrate incrementally up to maximum of 300 mcg/kg per minute. May consider repeating a loading dose (eg, ≤500 mcg/kg) prior to each up-titration step.	1 to 2	10 to 30	Nausea, flushing, bronchospasm, first-degree heart block, infusion-site pain; half-life prolonged in setting of anemia	Perioperative hypertension. Avoid use in acute decompensated heart failure.
Labetalol	Initial bolus of 20 mg IV followed by 20 to 80 mg IV bolus every 10 minutes (maximum 300 mg) **or** 0.5 to 2 mg/minute as IV loading infusion following an initial 20 mg IV bolus (maximum 300 mg)	5 to 10	2 to 4 hours	Nausea/vomiting, paresthesias (eg, scalp tingling), bronchospasm, dizziness, nausea, heart block	Most hypertensive emergencies including myocardial ischemia, hypertensive encephalopathy, pregnancy, and postoperative hypertension. Avoid use in acute decompensated heart failure. Use cautiously in obstructive or reactive airway.
Metoprolol	Initially 1.25 to 5 mg IV followed by 2.5 to 15 mg IV every 3 to 6 hours	20	5 to 8 hours	Refer to labetalol	Myocardial ischemia, perioperative hypertension. Avoid use in acute decompensated heart failure.
Phentolamine	5 to 15 mg IV bolus every 5 to 15 minutes	1 to 2	10 to 30	Tachycardia, flushing, headache, nausea/vomiting	Alternative option for catecholamine excess (eg, adrenergic crisis secondary to pheochromocytoma or cocaine overdose).

ACS: acute coronary syndrome; AMI: acute myocardial infarction; CAD: coronary artery disease; CVA: cerebrovascular accident; IM: intramuscular injection; IV: intravenous injection.

* The treatment of acute aortic syndromes (eg, acute aortic dissection, aortic intramural hematoma) requires specific medication management to minimize disease extension and is presented separately. Refer to UpToDate topics discussing acute aortic syndromes and acute aortic dissection.

¶ Hypotension may occur with all agents.

Δ IV short-acting agents for treatment of hypertensive emergency should be administered immediately by clinicians who are trained and experienced in their titration using continuous noninvasive electronic monitoring of blood pressure, heart rate, and cardiac rhythm. Patients should be admitted to an intensive care unit as rapidly as possible. A combination of IV agents is often selected depending upon the acute indication. Refer to appropriate UpToDate clinical topic for suggested combinations.

◇ Initial fenoldopam doses in range of 0.01 to 0.3 mcg/kg per minute have been described.

Secondary Stroke Prevention

بیمار خانم 62 ساله با سابقه دیس لیپیدمی ، فشار خون بالا ، دیابت از 8 سال پیش می باشد. چهار ماه پیش بدلیل سکته مغزی ایسکمیک حاد در آی سی یو بستری شده و خوشبختانه با اقدامات حمایتی لازم و پایداری نورولوژیک از بیمارستان مرخص شده است. جهت کنترل فشار خون بیمار چه رژیم دارویی ارجح است؟

5.3.9.3. Secondary Stroke Prevention

Recommendations for Secondary Stroke Prevention References that support recommendations are summarized in the Evidence Table.		
COR	**LOE**	**Recommendations**
1	**A**	1. In patients with hypertension who have experienced an ischemic stroke, transient ischemic attack (TIA), or ICH, treatment with a thiazide-type diuretic, ACEi, or ARB is recommended for lowering BP and reducing recurrent stroke and ICH risk.[1–3]
1	**B-R**	2. In patients with hypertension who have experienced an ischemic stroke, TIA, or ICH, an office SBP/DBP goal of <130/80 mm Hg is recommended to reduce the risk of recurrent stroke, ICH, and other vascular events.[1,3–5]
2a	**B-R**	3. In patients with no history of hypertension who have experienced an ischemic stroke, TIA, or ICH and have an average office SBP/DBP of ≥130/80 mm Hg, antihypertensive medication treatment can be beneficial to reduce the risk of recurrent stroke, ICH, and other vascular events.[5–7]

HTN & CKD

بیمار آقای 60 ساله با سابقه 20 ساله هایپرتنشن، دیابت و سابقه 8 ساله CKD می باشد. در حال حاضر GFR

بیمار 50 ml/min بوده و میزان پروتئینوری بیمار شدید می باشد. داروهای مصرفی بیمار در حال حاضر

شامل آتورواستاتین 20 میلی گرم روزانه ، مت فورمین1 گرم روزانه ، لیناگلیپتین 5 میلی گرم روزانه ، گلی

کلازید 80میلی گرم روزانه ، تلمیزارتان 80 میلی گرم روزانه و نفروویت روزی یک عدد می باشد. با دریافت

رژیم دارویی مذکور پروفایل قند خون بیمار در حد قابل قبول می باشد ولی متوسط فشار خون بیمار 145/90

میلیمتر جیوه است. اقدام درمانی مناسب جهت کنترل فشار خون بالای بیمار چیست؟

HTN and CKD

- Prevalence of HTN among CKD patients: 67-92%.
- CKD is an important risk factor for CVD.
- Coexistence of HTN and CKD further increases the risk of CVD events.

2025 AHA high blood pressure guideline. Circulation 2025.

- Increased activity of both the renin-angiotensin aldosterone system (RAAS) and sympathetic nervous system due to decreased GFR leads to sodium avidity and subsequent volume expansion.

- It is important to recognize and treat hypertension early in these patients because studies have shown increasing difficulty in achieving BP targets in later stages of CKD.

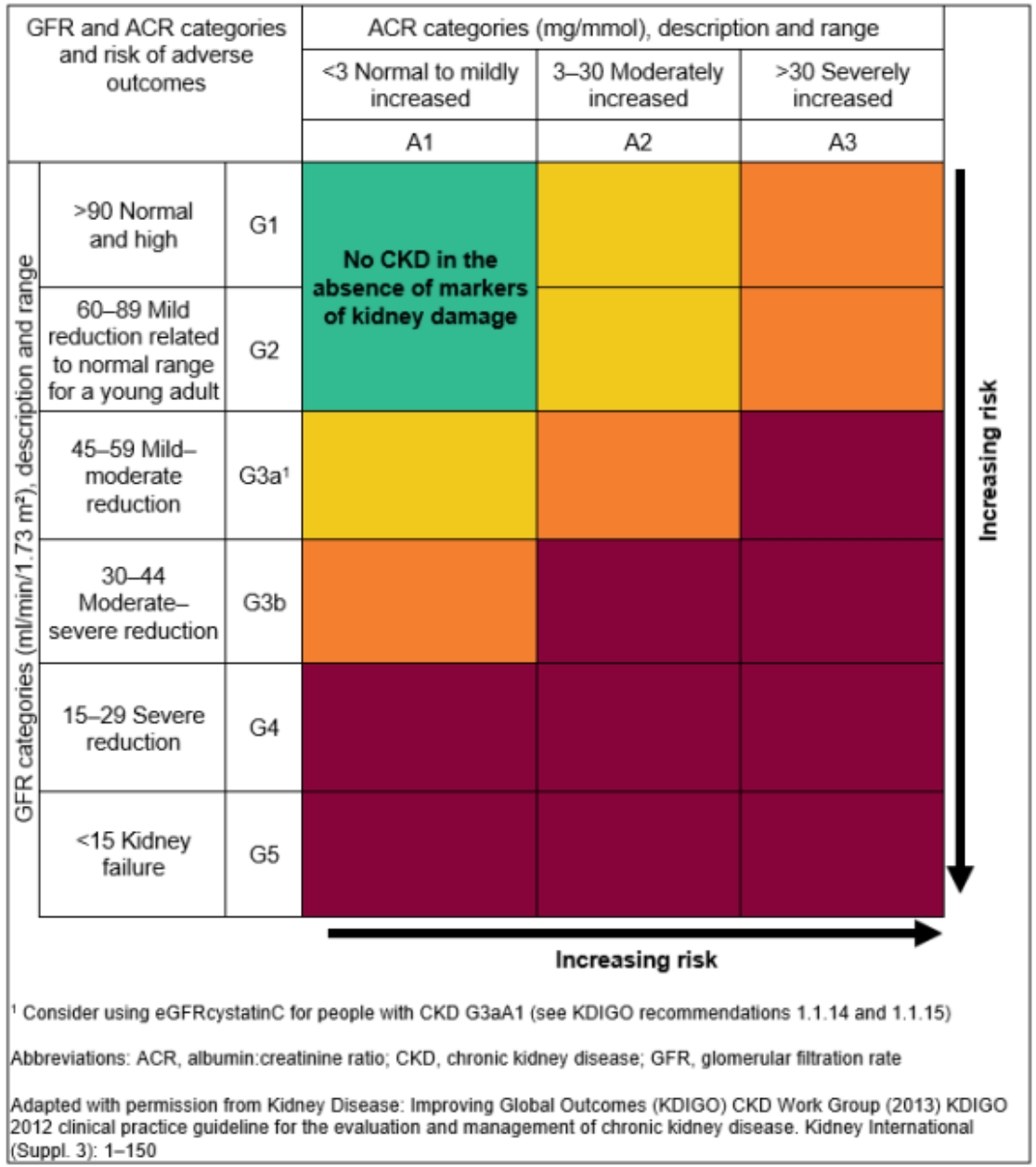
Am J Kidney Dis. 2024; 84(3):374-387.

BP Treatment Threshold in CKD

Recommendations for BP Treatment Threshold and the Use of CVD Risk Estimation to Guide Drug Treatment of Hypertension (Continued)

COR	LOE	Recommendations
1	A	5. In adults with hypertension without clinical CVD but with diabetes or CKD or at increased short-term CVD risk (ie, estimated 10-year CVD risk ≥7.5% based on PREVENT*), initiation of medications to lower BP is recommended when average SBP is ≥130 mm Hg to reduce the risk of CVD events and total mortality.[5–10]
1	C-LD	6. In adults with hypertension without clinical CVD but with diabetes or CKD or at increased 10-year CVD risk (ie, ≥7.5% based on PREVENT*), initiation of medications to lower BP is recommended when average DBP is ≥80 mm Hg to reduce the risk of CVD events and total mortality.[5–10]

2025 AHA high blood pressure guideline. Circulation 2025.



[1] Consider using eGFRcystatinC for people with CKD G3aA1 (see KDIGO recommendations 1.1.14 and 1.1.15)

Abbreviations: ACR, albumin:creatinine ratio; CKD, chronic kidney disease; GFR, glomerular filtration rate

Adapted with permission from Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group (2013) KDIGO 2012 clinical practice guideline for the evaluation and management of chronic kidney disease. Kidney International (Suppl. 3): 1–150

Interventions that delay CKD Progression

RAAS blockers

SGLT2Is

Non Steroidal Mineral Receptor Antagonists

RAAS Inhibitors (What's New?)

Recommendation 3.6.1:	**We recommend starting renin-angiotensin-system inhibitors (RASi) (angiotensin-converting enzyme inhibitor [ACEi] or angiotensin II receptor blocker [ARB]) for people with CKD and severely increased albuminuria (G1–G4, A3) without diabetes *(1B)*.**
Recommendation 3.6.2:	**We suggest starting RASi (ACEi or ARB) for people with CKD and moderately increased albuminuria (G1–G4, A2) without diabetes *(2C)*.**
Recommendation 3.6.3:	**We recommend starting RASi (ACEi or ARB) for people with CKD and moderately-to-severely increased albuminuria (G1–G4, A2 and A3) with diabetes *(1B)*.**
Recommendation 3.6.4:	**We recommend avoiding any combination of ACEi, ARB, and direct renin inhibitor (DRI) therapy in people with CKD, with or without diabetes *(1B)*.**

KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. KI. 2024

Practice Point 3.6.1: RASi (ACEi or ARB) should be administered using the highest approved dose that is tolerated to achieve the benefits described because the proven benefits were achieved in trials using these doses.

Practice Point 3.6.2: Changes in BP, serum creatinine, and serum potassium should be checked within 2–4 weeks of initiation or increase in the dose of a RASi, depending on the current GFR and serum potassium.

Practice Point 3.6.3: Hyperkalemia associated with use of RASi can often be managed by measures to reduce the serum potassium levels rather than decreasing the dose or stopping RASi.

Practice Point 3.6.4: Continue ACEi or ARB therapy unless serum creatinine rises by more than 30% within 4 weeks following initiation of treatment or an increase in dose.

Practice Point 3.6.5: Consider reducing the dose or discontinuing ACEi or ARB in the setting of either symptomatic hypotension or uncontrolled hyperkalemia despite medical treatment, or to reduce uremic symptoms while treating kidney failure (estimated glomerular filtration rate [eGFR] <15 ml/min per 1.73 m^2).

Practice Point 3.6.6: Consider starting people with CKD with normal to mildly increased albuminuria (A1) on RASi (ACEi or ARB) for specific indications (e.g., to treat hypertension or heart failure with low ejection fraction).

Practice Point 3.6.7: Continue ACEi or ARB in people with CKD even when the eGFR falls below 30 ml/min per 1.73 m^2.

AHA 2025 provides the same recommendations

KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. KI. 2024

Non Steroidal Mineral Receptor Antagonists

Recommendation 3.8.1: We suggest a nonsteroidal mineralocorticoid receptor antagonist with proven kidney or cardiovascular benefit for adults with T2D, an eGFR >25 ml/min per 1.73 m^2, normal serum potassium concentration, and albuminuria (>30 mg/g [>3 mg/mmol]) despite maximum tolerated dose of RAS inhibitor (RASi) *(2A)*.

Practice Point 3.8.1: Nonsteroidal MRA are most appropriate for adults with T2D who are at high risk of CKD progression and cardiovascular events, as demonstrated by persistent albuminuria despite other standard-of-care therapies.

Practice Point 3.8.2: A nonsteroidal MRA may be added to a RASi and an SGLT2i for treatment of T2D and CKD in adults.

Practice Point 3.8.3: To mitigate risk of hyperkalemia, select people with consistently normal serum potassium concentration and monitor serum potassium regularly after initiation of a nonsteroidal MRA (Figure 26).

K^+ ≤4.8 mmol/l

- Initiate finerenone
  - 10 mg daily if eGFR 25–59 ml/min/1.73 m^2
  - 20 mg daily if eGFR ≥60 ml/min/1.73 m^2
- Monitor K^+ at 1 month after initiation and then every 4 months
- Increase dose to 20 mg daily, if on 10 mg daily
- Restart 10 mg daily if previously held for hyperkalemia and K^+ now ≤5.0 mmol/l

K^+ 4.9–5.5 mmol/l

- Continue finerenone 10 mg or 20 mg
- Monitor K^+ every 4 months

K^+ >5.5 mmol/l

- Hold finerenone
- Consider adjustments to diet or concomitant medications to mitigate hyperkalemia
- Recheck K^+
- Consider reinitiation if/when K^+ ≤5.0 mmol/l

KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. KI. 2024

Practice Point 3.8.4: The choice of a nonsteroidal MRA should prioritize agents with documented kidney or cardiovascular benefits.

Practice Point 3.8.5: A steroidal MRA may be used for treatment of heart failure, hyperaldosteronism, or refractory hypertension, but may cause hyperkalemia or a reversible decline in glomerular filtration, particularly among people with a low GFR.

Low evidence for HTN control with Non Steroidal Mineral Receptor Antagonists
Spironolacton / Eplerenon is preffered agnts for resistant HTN
Avoid concureent use of Sprironolacton/Eplerenon with NSMRA

KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. KI. 2024

SGLT2 Inhibitors

(3) Interventions to Delay CKD Progression and Manage Complications

- Prescribe sodium–glucose cotransporter-2 (SGLT-2) inhibitors in adults with CKD and either of the following:
  - Type 2 diabetes and eGFR ≥20 mL/minute/1.73 m^2
  - No diabetes and eGFR ≥20 mL/minute/1.73 m^2 and ACR ≥200 mg/g; ***OR*** eGFR 20–45 mL/minute/1.73 m^2 and ACR <200 mg/g; ***OR*** heart failure (with or without proteinuria)
- Prescribe statin or statin-plus-ezetimibe combination therapy to patients who are not receiving dialysis and are 50 or older; ***OR*** are 18 to 49 with concurrent diabetes, heart disease, prior stroke, or 10-year risk for major adverse coronary events >10%.

COMMENT

The most relevant updates to this latest iteration of KDIGO's CKD guidelines are to use cystatin C routinely (when available) plus creatinine for estimating GFR and prescribing SGLT-2 inhibitors and statins quite broadly for patients with CKD. These interventions are likely to lead to more-accurate identification of and improved outcomes for patients with CKD.

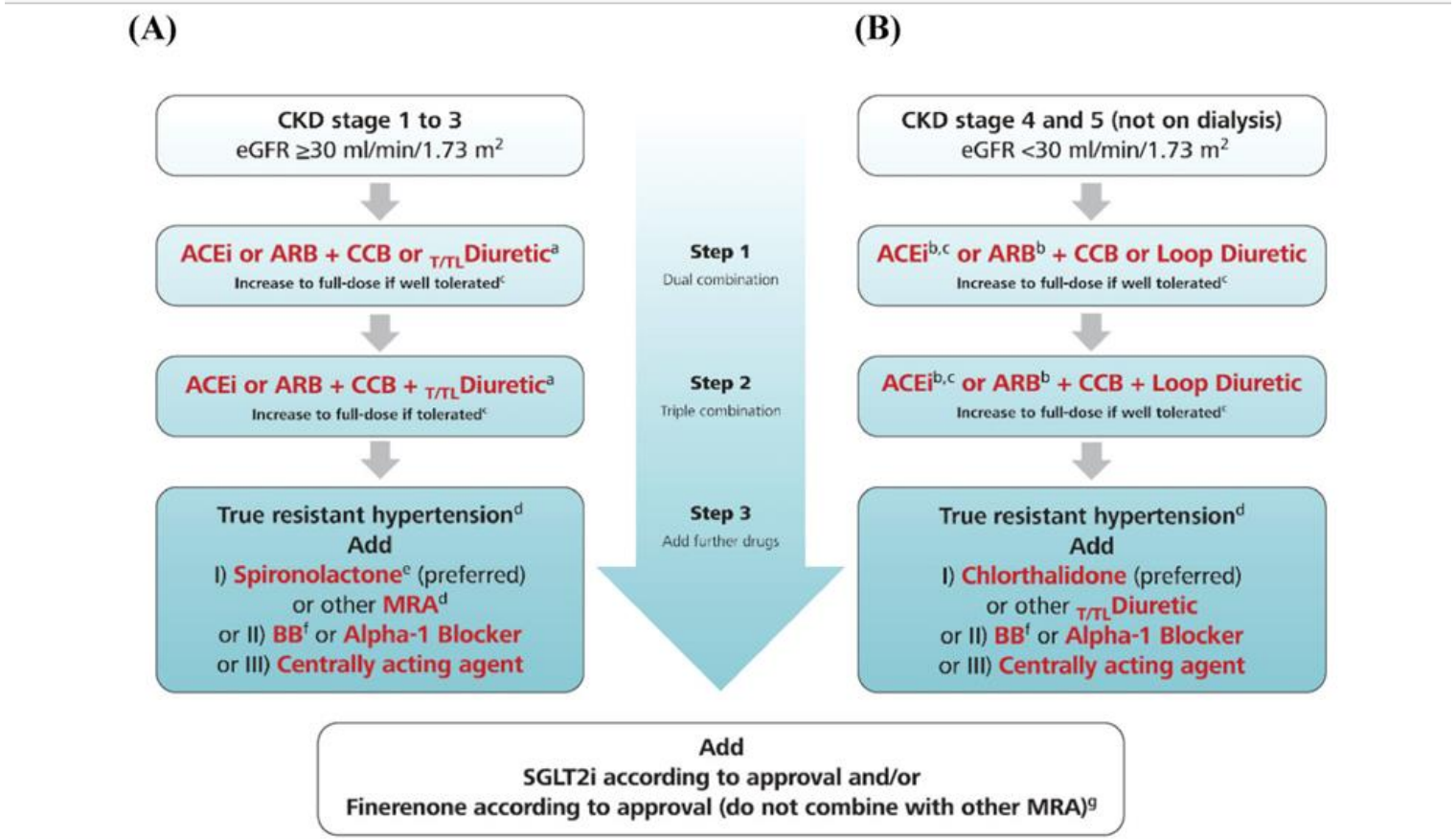


Figure 1: BP-lowering therapy in patients with hypertension and CKD. (A) Therapy for CKD G1–G3 (eGFR ≥30 mL/min/1.73 m²). (B) Therapy for CKD G4–G5 (eGFR <30 mL/min/1.73 m²) not on dialysis. (a) Transition from T/TL diuretic to loop diuretic should be individualized in patients with eGFR <45 mL/min/1.73 m². (b) Cautious start with low dose. (c) Check for dose adjustment according to renal impairment for drugs with relevant renal excretion rate. (d) When SBP is ≥140 mmHg or DBP is ≥90 mmHg provided that: maximum recommended and tolerated doses of a three-drug combination comprising a RAS blocker (either an ACEi or an ARB), a CCB and a T/TL diuretic were used, adequate BP control has been confirmed by ABPM or by HBPM if ABPM is not feasible, various causes of pseudo-resistant hypertension (especially poor medication adherence) and secondary hypertension have been excluded. (e) Caution if eGFR <45 mL/min/1.73 m² or serum potassium >4.5 mmol/L. (f) Should be used at any step as guideline directed medical therapy in respective indications or considered in several other conditions. (g) SGLT2i and finerenone should be used according to their approval for CKD treatment (from [14], with permission). T/TL: thiazide/thiazide-like.

Clinical Kidney Journal , 2024, vol. 17, Suppl 2, ii36–ii50

بیمار آقای 68 ساله با سابقه فشار خون بالا و بیماری ایسکمی قلبی و ESRD هفته ای سه بار تحت همودیالیز قرار دارد. جهت کنترل فشار خون بالا ، بیمار تحت درمان با آتنولول 25 میلی گرم سه بار در هفته می باشد که متاسفانه فشار بین نوبت های دیالیز 150/90 میلی متر جیوه است. جهت کنترل فشار خون بالای بیمار چه دارویی را پیشنهاد می کنید؟

Dialysis (HD/PD) & HTN

Hypertension Among Dialysis Patients

BP Goal	
Hemodialysis	**Peritoneal Dialysis**
Interdialytic BP of <140/80mmHg using HBPM Or Midweek intradialytic BP<140/80mmHg	<130/80mmHg using HBPM Or <130/80 mmHg using AOBP Or <140/90 mmHg using routine office-based measurement

UpToDate. 20205

Hypertension Treatment Among Dialysis Patients

Optimizing volume status and reducing target dry weight

BB: (?)

HD patients: 25mg thrice weekly; max. 100mg;

PD patients: 25mg daily; maximum 50 mg

reduce dose or stop dose increasing if HR≤ 50 beats/min

NDT. 2014;29: 672-81.

If BP not controlled, CCBs (amlodipine 10mg)

If not controlled; ACEi or ARB

for patients who still makes urine consider ACEi/ARB as firs-line antihypertensive because of potential for preserving residual renal function

If resistant: other agents such as minoxidil

UpToDate. 20205

Post Kidney Transplantation & HTN

بیمار خانم 45 ساله ، 2 سال پیش بدلیل گلومرونفریت کاندید پیوند کلیه شده ، در حال حاضر 2 ماه از زمان پیوند کلیه بیمار گذشته و بیمار تحت درمان با تاکرولیموس ، مایکوفنولات مفتیل و پردنیزولون می باشد. اخیرا دیابت و هایپرتنشن به تابلوی علائم بیمار اضافه شده و متوسط فشار خون بیمار 155/95 میلیمتر جیوه است. اقدام درمانی مناسب جهت کنترل فشار خون بیمار چیست؟

Hypertension After Kidney Transplantation

- HTN is common after kidney transplantation because of
  - pre-existing kidney disease,
  - effects of immunosuppressive medications (CNIs) (among KT as well as other SOT),
  - presence of allograft pathology

- High proportion of masked hypertension (up to 40%); abnormal dipping status, and high prevalence of nocturnal hypertension (reaching up to 70%–80%) are reported among KT patients.

Clinical Kidney Journal , 2024, vol. 17, Suppl 2, ii36–ii50

Hypertension After Kidney Transplantation

- Both 2023 ESH and 2021 KDIGO Guidelines use the same target of <130/80 mmHg as a reasonable target for KTRs (CoR II, LoE B).

Clinical Kidney Journal , 2024, vol. 17, Suppl 2, ii36–ii50

Hypertension After Kidney Transplantation

- In a systematic review/meta-analysis on 71 RCTs:
- CCBs (26 trials) reduced the risk for graft loss [RR 0.58; 95% CI 0.38-0.89], increased GFR [MD 3.08 mL/min (95% CI 0.38-5.78)] and reduced BP.
- ACEIs (13 trials) reduced the risk for graft loss [RR 0.62; 95% CI 0.40-0.96] but decreased renal function and increased the risk for hyperkalaemia.
- ARBs (10 trials) did not modify the risk of death, graft loss and non-fatal CV events and increased the risk for hyperkalaemia.
- When pooling ACEI and ARB data, the risk for graft failure was lower in renin-angiotensin system (RAS) blockade as compared with control treatments.
- In direct comparison with ACEIs or ARBs (11 trials), CCBs increased GFR [MD 11.07 mL/min (95% CI 6.04-16.09)] and reduced potassium levels but were not more effective in reducing BP.

Nephrol Dial Transplant. 2020; 35(5):878-87.

Hypertension After Kidney Transplantation

- There are few available data on mortality, graft loss and rejection. Very few studies performed comparisons with other active drugs.

- **Conclusions:**
- CCBs could be the preferred first-step antihypertensive agents in kidney transplant patients, as they improve graft function and reduce graft loss.
- No definite patient or graft survival benefits were associated with RAS inhibitor use over conventional treatment.

Nephrol Dial Transplant. 2020; 35(5):878-87.

HTN & DM

بیمار آقای 58 ساله با سابقه 4 ساله دیابت ملیتوس تحت درمان با داروهای آتورواستاتین ، مت فورمین 1000
میلی گرم دو بار در روز و سیتاگلیپتین 100 میلی گرم در روز می باشد.متاسفانه در طی چند ماه گذشته کنترل
قند خون بیمار مناسب نبوده و A1C بیمار 8.2 درصد است و هایپرتنشن هم به تابلوی علائم بیمار اضافه شده
است. متوسط فشار خون بیمار 155/95 میلی متر جیوه است. در آزمایشات بیمار پروتئینوری خفیف گزارش
شده است. اقدام درمانی مناسب جهت کنترل فشار خون بیمار چیست؟

- More than 80% of adults with T2D also have hypertension.
- Further, CVD risk in adults with both T2D and hypertension is more than double the risk for either condition alone.
- Hypertension accelerates CKD, particularly when moderate or severe albuminuria is present.
- HTN + DM +/- CKD & IHD
- Goal of BP is less than 130 /80 mmHg and the optimal goal is less than 120/80 for reducing CV mortality and morbidity

Any of the recommended antihypertensive drug classes (ACEi, ARB, CCB, and diuretics) are useful in the treatment of hypertension in diabetes.

ACEi or ARB is recommended as part of treatment in patients with diabetes and CKD, defined by an eGFR< 60 ml/ml/1.73m2 who also have moderate or severe albuminuria, defined as 30 mg albumin per g creatinine or greater. An ACEi or ARB is also appropriate for less severe CKD (stage 1 or 2) when moderate or severe albuminuria is present.

SGLT2i and GLP-1 receptor agonists have been demonstrated to slow decline in kidney function whether or not diabetes is present and may have some beneficial effects on BP.

5.3.1. Diabetes

Recommendations for Diabetes Referenced studies that support the recommendations are summarized in the Evidence Table.		
COR	**LOE**	**Recommendations**
1	A	1. In adults with T2D and hypertension, antihypertensive drug treatment should be initiated at an SBP of ≥130 mm Hg with a treatment goal of <130 mm Hg, with encouragement to achieve an SBP <120 mm Hg to reduce CVD morbidity and mortality.[1–5]
1	C-LD	2. In adults with T2D and hypertension, antihypertensive drug treatment should be initiated at a DBP of ≥80 mm Hg with a treatment goal of <80 mm Hg to reduce CVD morbidity and mortality.[6]
1	A	3. In adults with T2D and hypertension, all first-line classes of antihypertensive agents (ie, thiazide-type diuretics, long-acting CCB, ACEi, and ARB) are useful and effective for BP lowering.[7–9]
1	A	4. In adults with diabetes and hypertension, ACEi or ARB are recommended in the presence of CKD as identified by eGFR <60 mL/min/1.73 m^2 or albuminuria ≥30 mg/g and should be considered when mild albuminuria (<30 mg/g) is present to delay progression of diabetes-related kidney disease.[10–12]

HTN & HFrEF

بیمار آقای 71 ساله با سابقه فشار خون بالا ، دیس لیپیدمی و بیماری ایسکمی قلبی و نارسایی قلبی

مزمن با کاهش کسر تخلیه (LVEF = 35 %) تحت درمان با آسپرین 80 روزی یک عدد، آتورواستاتین

40 روزی یک عدد، بیزوپرولول روزی 5 میلی گرم ، امپاگلیفلوزین 10 میلی گرم روزانه ، والزارتان 160

میلی گرم روزانه و اپلرنون 25 میلی گرم روزانه می باشد. متاسفانه با دریافت رژیم دارویی ذکر شده

هنوز متوسط فشار خون وی 150/90 میلیمتر جیوه است. اقدام درمانی مناسب جهت کنترل فشار خون

بالای بیمار چیست؟

- Hypertension is the most common medical comorbidity in patients with HF, and its prevalence among patients with heart failure with reduced ejection fraction (HFrEF), defined as left ventricular ejection fraction ≤40%, continues to rise.

- Hypertension is known to be a major risk factor for HFrEF directly through alterations in cardiac structure and function in response to chronic pressure overload and indirectly through its associations with ischemic heart disease.

- A goal SBP <130/80 mmHg should at least be attained in patients with hypertension and HFrEF.

- In patients with HFrEF and hypertension, up titration of HF GDMT to the maximally tolerated dose is recommended for hypertension control.

- Diuretics should be added as needed for volume overload.

- Dihydropyridine CCB may be used to treat hypertension in patients with elevated BP despite the optimization of GDMT.

Table 18. GDMT for Patients With Hypertension and HFrEF

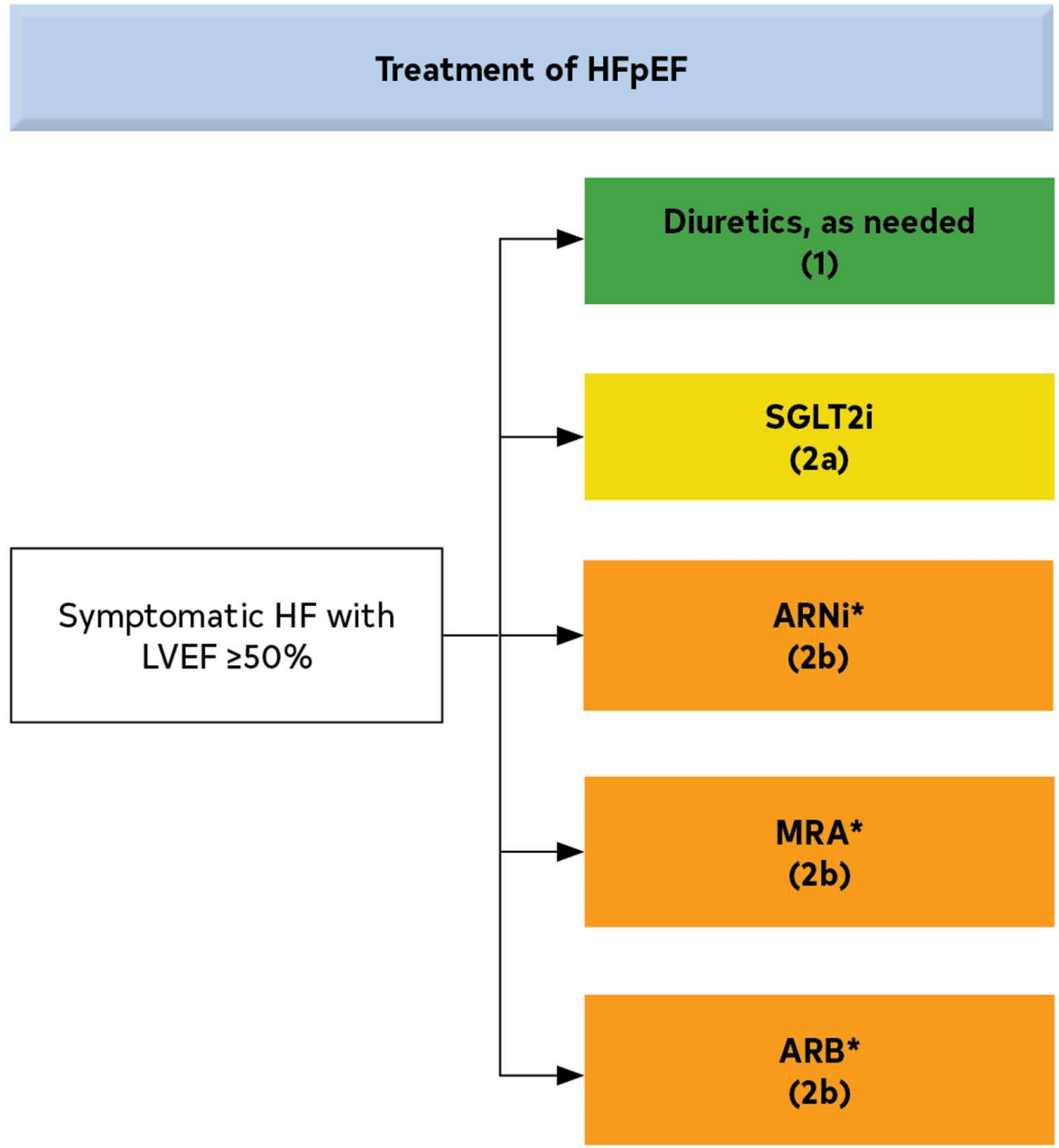
Drug Class	Notes on Use
BB	In patients with HFrEF, even if asymptomatic, use 1 of the 3 BBs proven to reduce mortality and hospitalizations (bisoprolol, carvedilol, metoprolol succinate).
MRA	In patients with symptomatic HFrEF, spironolactone or eplerenone is recommended to reduce morbidity and mortality if eGFR is >30 mL/min/1.73 m^2 and potassium is <5.0 mEq/L.
RAASi with ACEi or ARB or ARNi	In patients with HFrEF and NYHA functional class II to III symptoms, ARNi is recommended to reduce morbidity and mortality. When the use of ARNi is not feasible, ACEi or ARB is recommended to reduce morbidity and mortality.
SGLT2i	SGLT2i are recommended in patients with symptomatic HFrEF to reduce hospitalization and cardiovascular mortality irrespective of the presence of type 2 diabetes.
Additional GDMT to be added as indicated	
Hydralazine and isosorbide dinitrate	For patients self-identified as Black with NYHA functional class III to IV HFrEF who are receiving optimal medical therapy, the combination of hydralazine and isosorbide dinitrate is recommended to improve symptoms and reduce morbidity and mortality. In patients with current or previous symptomatic HFrEF who cannot be given first-line agents, such as ARNi, ACEi, or ARB, because of drug intolerance or renal insufficiency, a combination of hydralazine and isosorbide dinitrate might be considered to reduce morbidity and mortality.

Modified with permission from Heidenreich et al.[5] Copyright 2022 American Heart Association, Inc., and American College of Cardiology Foundation.

ACEi indicates angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blockers; ARNi, angiotensin receptor-neprilysin inhibitors; BB, beta blocker; eGFR, estimated glomerular filtration rate; GDMT, guideline-directed medical therapy; HFrEF, heart failure with reduced ejection fraction; MRA, mineralocorticoid receptor antagonist; NYHA, New York Heart Association; RAASi, renin-angiotensin-aldosterone system inhibitors; and SGLT2i, sodium-glucose cotransporter inhibitors.

HTN & HFpEF

بیمار آقای 65 ساله با سابقه 12 ساله فشار خون بالا تحت درمان با آملودیپین 10 میلی گرم روزانه و
هیدروکلروتیازید 25 میلی گرم روزانه می باشد. بیمار سابقه دیس لیپیدمی ، COPD را از حدود 8 سال پیش
ذکر میکند. متاسفانه در طی سالهای اخیر بیمار کنترل مناسب در فشار خون نداشته و در اکوکاردیوگرافی انجام.
شده جهت بیمار هایپرتروفی بطن چپ در حد متوسط تا شدید همراه با درجاتی از دیاستولیک دیسفانکشن بطن
چپ در حد متوسط گزارش شده و کسر تخلیه بطن چپ 55 درصد در اکوکاردیوگرافی گزارش شده است. در
حال حاضر متوسط فشار خون بیمار 160/95 میلی متر جیوه بوده و شکایت اصلی بیمار تنگی نفس فعالیتی
می باشد. اقدام درمانی مناسب جهت کنترل فشار خون بیمار چیست؟


Treatment of HFpEF
Symptomatic HF with LVEF ≥50%
Diuretics, as needed (1)
SGLT2i (2a)
ARNi* (2b)
MRA* (2b)
ARB* (2b)

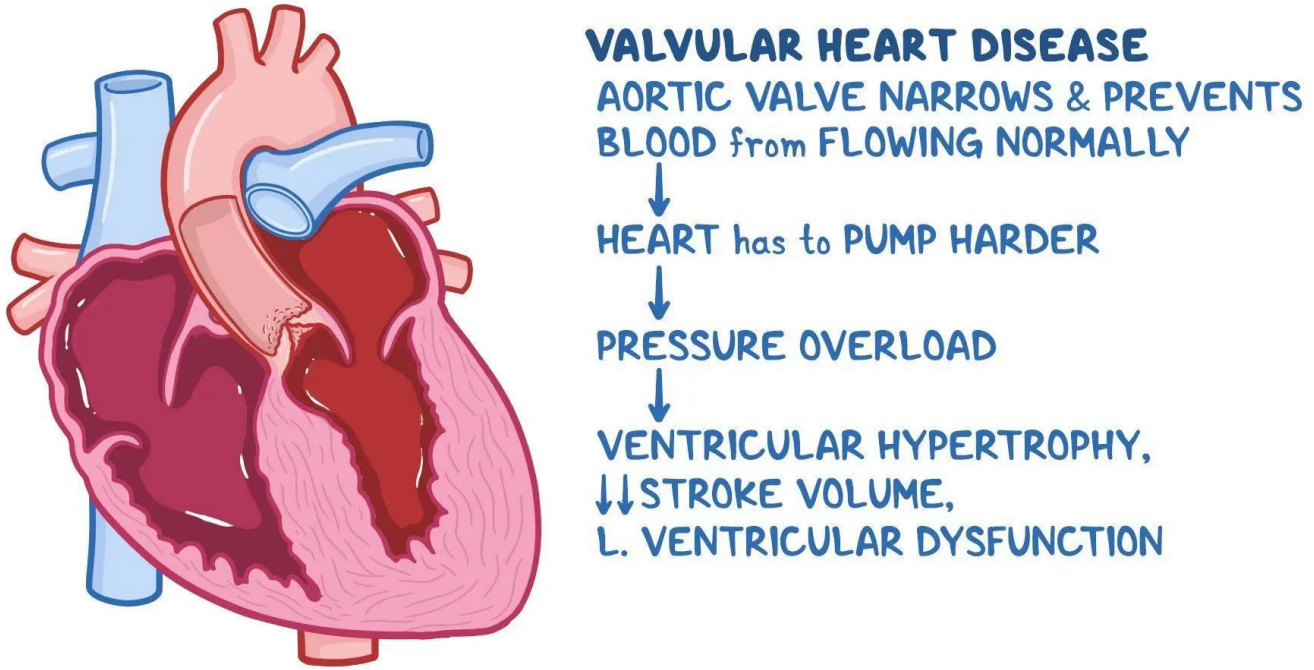
- Hypertension is a major risk factor for developing heart failure with preserved ejection fraction (HFpEF) and an important target for HF management to reduce hospitalization, CVD events, and mortality.
- Goal BP is less than 130/80 mmHg
- Appropriate use of diuretics is crucial to the success of other antihypertensive medications in the presence of HFpEF and should be used for signs and/or symptoms of volume overload.
- SGLT2i are used frequently for HFpEF treatment (with and without diabetes), unless contraindicated, to reduce the risk of hospitalization and cardiovascular mortality. SGLT2i may lower BP; therefore, adjustment in other antihypertensive medications may be indicated if signs or symptoms of hypotension are present.

- RAASi are indicated for management of HFpEF to attain an SBP of < 130/80 mmHg, especially with an MRA or ARNi, or ARB when ARNi is not feasible.

- BB are not recommended for hypertension management with HFpEF given negative chronotropic effects and should be restricted to specific comorbid conditions (eg, arrhythmia, ACS)

AS /AR & HTN

Aortic Stenosis



Asymptomatic AS

Diuretics:

Diuretics generally should be avoided particularly if the LV chamber is small due to concentric hypertrophy. Any further reduction in LV volume (eg, with a diuretics) could lead to a fall in cardiac output.

Vasodilators:

A fixed obstruction at the valve level might blunt the expected rise in cardiac output as afterload is reduced with a vasodilator possibly leading to hypotension and decreased coronary perfusion. However in asymptomatic AS , the valve leaflets are still flexible and the degree of leaflet opening can increase with increases in transvalvular flow rate even valve obstruction is sever.

BBs:

reduce contractility which may pose a risk for the LV overload. while low dose beta blockers may be considered in patients with asymptomatic hypertension especially in setting of AF

ACEIs / ARBs:

May have beneficial effect on LV fibrosis in addition to control hypertension

Symptomatic AS

Concomitant HTN and Sever AS present a double load to the LV which may exacerbate ventricular remodeling. No specific treatment regimen with patients with symptomatic AS and HTN has been established.

Diuretics: should be used with caution.

BBs: reduce contractility which may pose a risk for the LV overload. while low dose beta blockers may be considered in patients with asymptomatic hypertension especially in setting of AF , BBs should be avoided in patients with symptomatic AS and HF

Vasodilators (Hydralazine, TNG, DHP CCBs)may cause hypotension and coronary perfusion and should be used with caution.

ACEI/ARB : There are few data in this filed

Start with low dose of ACEI/ARB and titrate slowly and in uncontrolled HTN CCBs should be used with caution.

Recommendations for Medical Therapy of AS

Referenced studies that support the recommendations are summarized in Online Data Supplement 5.

COR	LOE	Recommendations
1	B-NR	1. In patients at risk of developing AS (Stage A) and in patients with asymptomatic AS (Stages B and C), hypertension should be treated according to standard GDMT, started at a low dose, and gradually titrated upward as needed, with appropriate clinical monitoring.[1–3]
1	A	2. In all patients with calcific AS, statin therapy is indicated for primary and secondary prevention of atherosclerosis on the basis of standard risk scores.[4–6]
2b	B-NR	3. In patients who have undergone TAVI, renin–angiotensin system blocker therapy (ACE inhibitor or ARB) may be considered to reduce the long-term risk of all-cause mortality.[7,8]
3: No Benefit	A	4. In patients with calcific AS (Stages B and C), statin therapy is not indicated for prevention of hemodynamic progression of AS.[4–6]

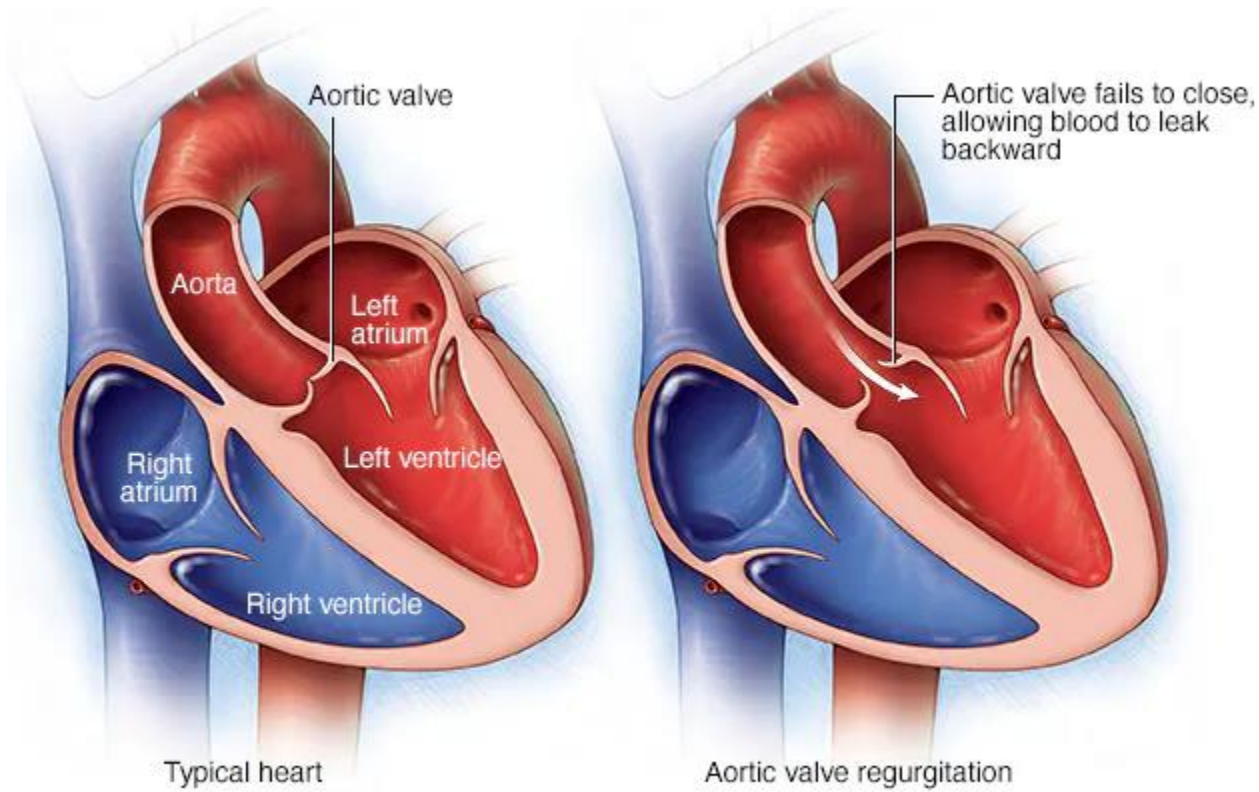
AR and HTN

- Chronic aortic regurgitation is often accompanied by a wide pulse pressure
- **BBs & NDHP CCBs:**

  medications that lower heart rate may paradoxically increase SBP

  Reduction in HR cause increase in stroke volume that may contribute to elevated systolic pressure and also leads to an increase in the duration of diastole which can contribute to an increase in the regurgitation volume.
- **ACEIs/ARBs, DHP CCBs**

  may be favored since they may reduce SBP in this setting.



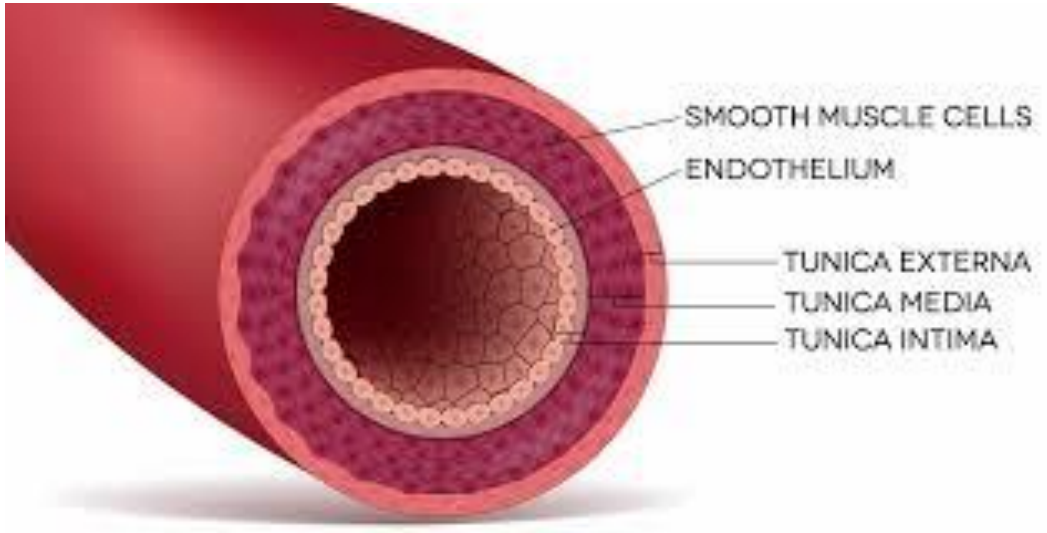
Recommendations for Medical Therapy of Chronic AR

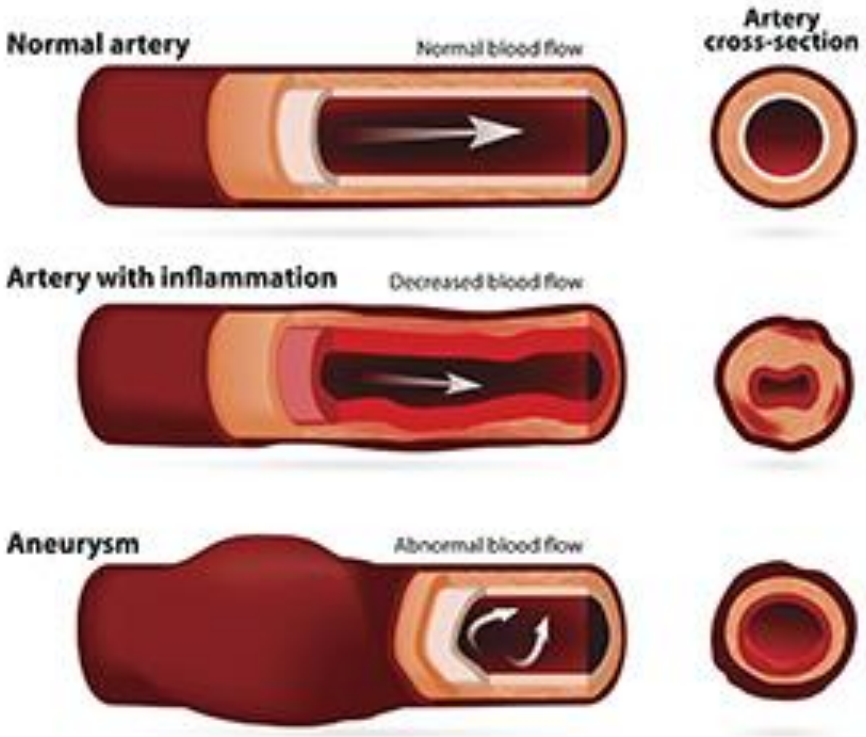
Referenced studies that support the recommendations are summarized in Online Data Supplement 14.

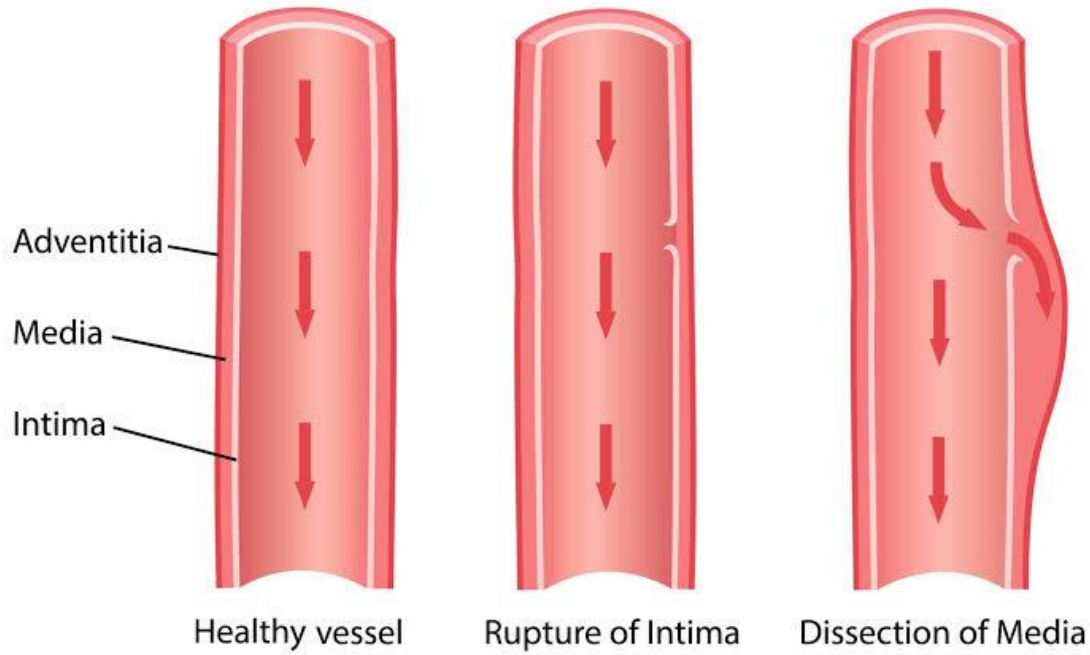
COR	LOE	Recommendations
1	B-NR	1. In asymptomatic patients with chronic AR (Stages B and C), treatment of hypertension (systolic blood pressure >140 mm Hg) is recommended.[1–3]
1	B-NR	2. In patients with severe AR who have symptoms and/or LV systolic dysfunction (Stages C2 and D) but a prohibitive surgical risk, GDMT for reduced LVEF with ACE inhibitors, ARBs, and/or sacubitril/valsartan is recommended.[4]

Aortic Disease & HTN

Develop the definitions for aneurysm and dissection to include: An aneurysm is a bulge in the aorta or a peripheral artery. A dissection of the aorta or its branches occurs when the inner lining of the artery is “torn” and begins to separate from the rest of the arterial wall.




Normal artery
Normal blood flow
Artery cross-section
Artery with inflammation
Decreased blood flow
Aneurysm
Abnormal blood flow


Adventitia
Media
Intima
Healthy vessel
Rupture of Intima
Dissection of Media

Goal BP:

The goal systolic pressure is 105 to 120 mmHg if tolerated

Choice of Antihypertensive drugs:

Beta blockers:

we suggest blood pressure control primarily using beta blockers with the aim of limiting further aortic expansion . Beta blockers reduce the inotropic state of the heart, decreasing left ventricular contractility (dP/dt), shear stress, and the impact force of ejected blood on the aorta.

ACEIs/ARBs:

ACE inhibition may have beneficial effects by modifying the inflammatory mediators and by decreasing vascular smooth muscle apoptosis and downregulation of TGF-beta

HTN & CCS

بیمار آقای 68 ساله با سابقه دیابت ، هایپرتنشن ، دیس لیپیدمی و بیماری ایسکمی قلبی پایدار تحت درمان با

آسپرین 80 روزانه ، آتورواستاتین 20 روزانه ، والزارتان 160 میلی گرم در روز ، کلشی سین 0.5 میلی گرم روزانه

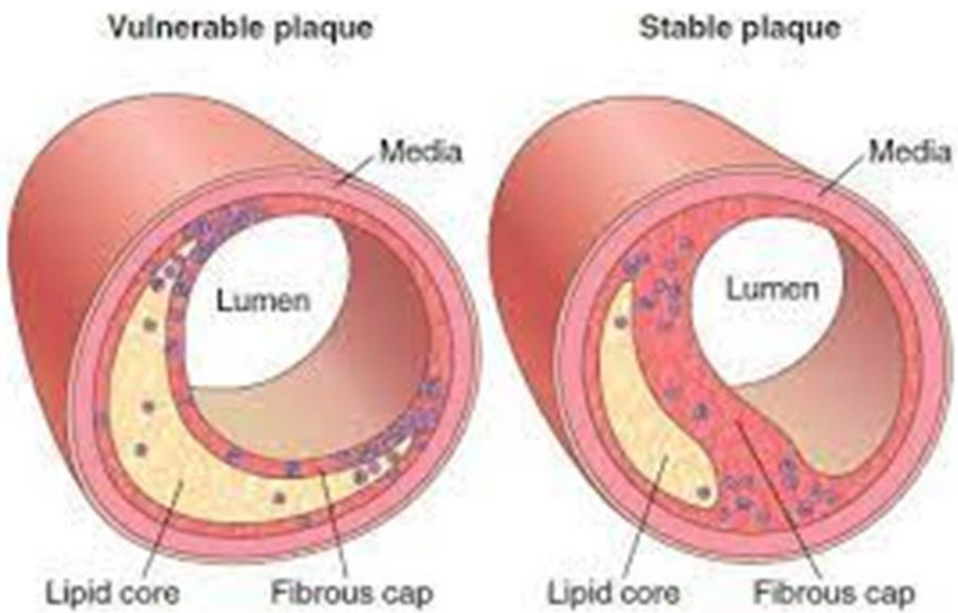
، متورال 25 میلی گرم دو بار در روز ؛ امپاگلیفلوزین 10 میلی گرم روزانه و مت فورمین 1000 میلی گرم دو بار

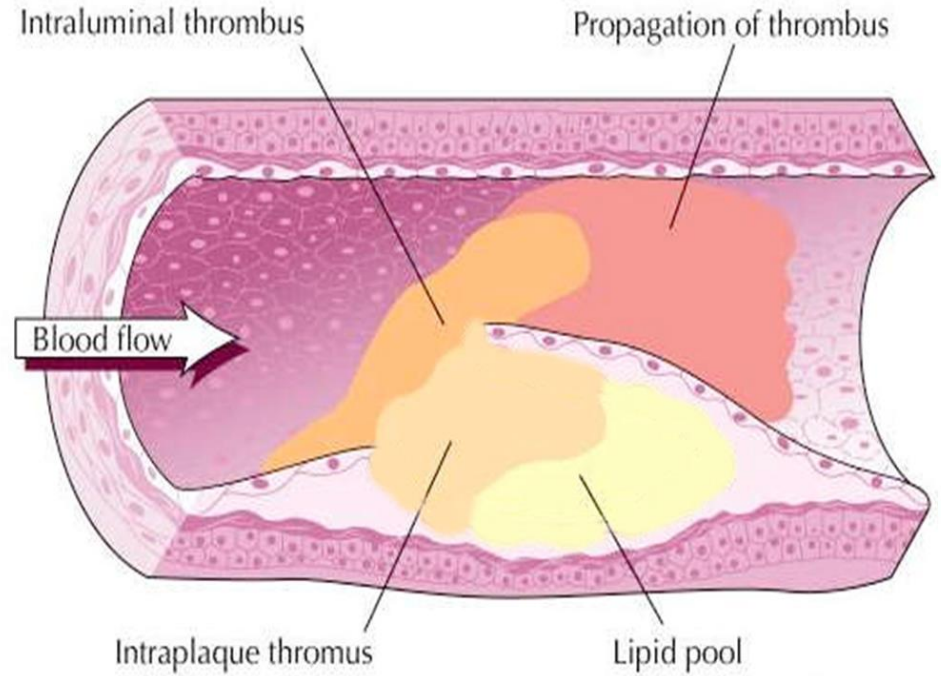
در روز و نیتروکانتین 2.6 دو بار در روز می باشد. علائم حیاتی بیمار در حال حاضر به شرح زیر است:

فشار خون : 150/90 میلی متر جیوه ضربان قلب: 80 در دقیقه

متاسفانه هنوز دردهای ایسکمی بیمار ادامه داشته و فشار خون و قند خون و LDL-C بیمار هم در حد مطلوب

نمی باشد. اقدام درمانی مناسب جهت کنترل فشار خون بیمار چیست؟


Vulnerable plaque
Media
Lumen
Lipid core
Fibrous cap
Stable plaque
Media
Lumen
Lipid core
Fibrous cap


Intraluminal thrombus
Propagation of thrombus
Blood flow
Intraplaque thromus
Lipid pool

داروهای موثر در پیشگیری اولیه و ثانویه وقایع قلبی عروقی در SIHD

داروهای آنتی پلاکت

داروهای آنتی کوآگولانت (دوز کم ریواروکسابان)

ACEIs/ARBs

استاتین ها

کلشی سین

درمان های ضد ایسکمی جهت کنترل علائم ایسکمی

بتا بلاکرها

مهار کننده های کانال کلسیمی

نیترات های طولانی اثر

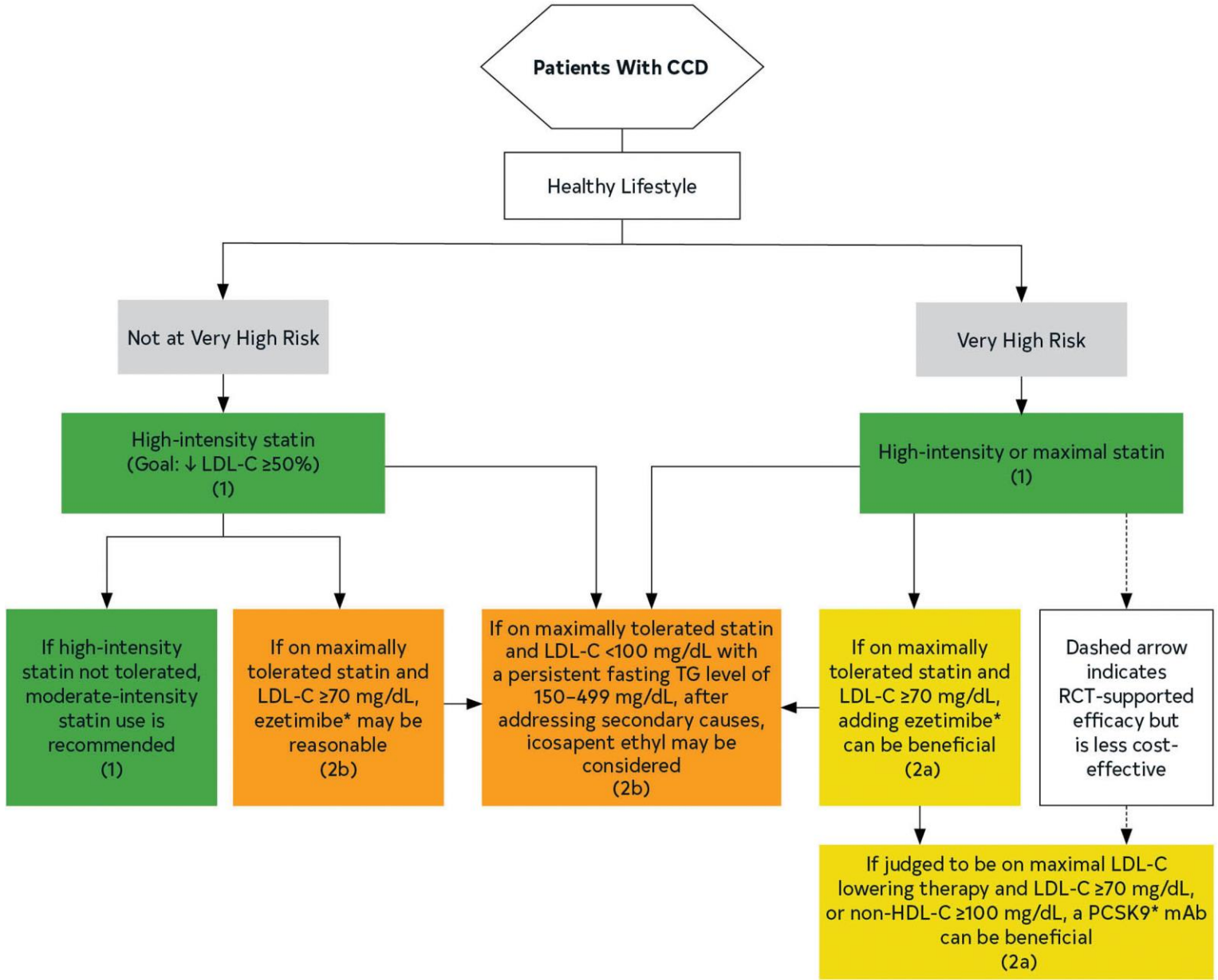
رانولازین

نیکوراندیل

Recommendations for Blood Pressure Management
Referenced studies that support the recommendations are summarized in the Online Data Supplement.

COR	LOE	Recommendations
1	A	1. In adults with CCD, nonpharmacologic strategies are recommended as first-line therapy to lower BP in those with elevated BP (120-129/<80 mm Hg) (see Table 12).*[1–9]
1	B-R	2. In adults with CCD who have hypertension, a BP target of <130/<80 mm Hg is recommended to reduce CVD events and all-cause death.*[10–14]
1	B-R	3. In adults with CCD and hypertension (systolic BP ≥130 and/or diastolic BP ≥80 mm Hg), in addition to nonpharmacological strategies, GDMT angiotensin-converting enzyme (ACE) inhibitors, angiotensin-receptor blockers (ARB), or beta blockers[15–17] are recommended as first-line therapy for compelling indications (eg, recent MI or angina), with additional antihypertensive medications (eg, dihydropyridine calcium channel blockers [CCB], long-acting thiazide diuretics, and/or mineralocorticoid receptor antagonists) added as needed to optimize BP control.*[13,18]

*Modified from the 2017 ACC/AHA Multisociety Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults.[19]


Patients With CCD
Healthy Lifestyle
Not at Very High Risk
Very High Risk
High-intensity statin (Goal: ↓ LDL-C ≥50%) (1)
High-intensity or maximal statin (1)
If high-intensity statin not tolerated, moderate-intensity statin use is recommended (1)
If on maximally tolerated statin and LDL-C ≥70 mg/dL, ezetimibe* may be reasonable (2b)
If on maximally tolerated statin and LDL-C <100 mg/dL with a persistent fasting TG level of 150–499 mg/dL, after addressing secondary causes, icosapent ethyl may be considered (2b)
If on maximally tolerated statin and LDL-C ≥70 mg/dL, adding ezetimibe* can be beneficial (2a)
Dashed arrow indicates RCT-supported efficacy but is less cost-effective
If judged to be on maximal LDL-C lowering therapy and LDL-C ≥70 mg/dL, or non-HDL-C ≥100 mg/dL, a PCSK9* mAb can be beneficial (2a)

Recommendations for Use of SGLT2 Inhibitors and GLP-1 Receptor Agonists
Referenced studies that support the recommendations are summarized in the Online Data Supplement.

COR	LOE	Recommendations
1	A	1. In patients with CCD who have type 2 diabetes, the use of either an SGLT2 inhibitor[1–8] or a GLP-1 receptor agonist[9–17] with proven cardiovascular benefit is recommended to reduce the risk of MACE.
Cost Value Statement: High Value	B-NR	2. In patients with CCD and type 2 diabetes, addition of a GLP-1 receptor agonist at US prices is projected to be of high value compared with standard of care.[18]
Cost Value Statement: Intermediate Value	B-NR	3. In patients with CCD and type 2 diabetes, addition of an SGLT2 inhibitor at US prices is projected to be of intermediate value compared with standard of care.[18]
1	A	4. In patients with CCD and heart failure with LVEF ≤40%, use of an SGLT2 inhibitor is recommended to reduce the risk of cardiovascular death and heart failure hospitalization[19–22] and to improve QOL,[23,24] irrespective of diabetes status.*
Cost Value Statement: Intermediate Value	B-NR	5. In patients with CCD and heart failure with LVEF ≤40%, addition of an SGLT2 inhibitor to GDMT, irrespective of diabetes status, is projected to be of intermediate value at US prices.[25,26]
2a	B-R	6. In patients with CCD and heart failure with LVEF >40%, use of an SGLT2 inhibitor can be beneficial in decreasing heart failure hospitalizations[27,28] and to improve QOL,[4,29] irrespective of diabetes status.
Cost Value Statement: Intermediate Value	B-NR	7. In patients with CCD and heart failure with LVEF >40%, addition of an SGLT2 inhibitor to GDMT, irrespective of diabetes status, is projected to be of uncertain value at US prices.[30]

*Modified from the 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure.[31]

بیمار آقای 65 ساله با سابقه دیابت از 20 سال پیش ، بیماری ایسکمی قلبی از 15 سال پیش اخیرا بدلیل عدم کنترل دیابت دچار نفروپاتی دیابتی با پروتئین اوری 50 mg/g شده و فشار خون بالا هم به تابلوی علائم بیمار اضافه شده است. در اکوکاردیوگرافی انجام شده تنگی دریچه آئورت همراه با هایپرتروفی بطن چپ گزارش شده است .
در حال حاضر متوسط فشار خون بیمار 150/90 میلی متر جیوه است. داروهای مصرفی بیمار شامل آسپرین روزانه 80 میلی گرم ، سینوریپا 1000/12.5 دو بار در روز، والزومیکس 160/5 دو بار در روز ، آتورواستاتین روزانه 40 میلی گرم می باشد. جهت کنترل فشار خون بیمار چه دارویی را پیشنهاد میدهید؟

THE END