evaluate for secondary hypertension, including primary or secondary aldosteronism (Section 3.2.3.1, "Primary Aldosteronism") and other endocrine causes. A basic metabolic panel should be checked 2 to 4 weeks after initiation or dose titration of specific antihypertensive medication classes, including diuretics, ACEi, ARB, and MRA. Common lab disturbances relate to changes in potassium, sodium, or creatinine. In addition to secondary causes of hypertension, hypokalemia may be caused by kaliuresis from thiazide-type and loop diuretics. Hyperkalemia may be caused by ACEi, ARB, MRA, and potassium-sparing diuretics especially when used in combination or in the setting of CKD. ACEi and ARB should not be used concurrently due to several trials demonstrating an increased risk for AKI or renal dysfunction.[1-3] Hyponatremia may be caused by diuretics, in particular thiazide-type diuretics. Strategies to mitigate electrolyte disturbances related to antihypertensive medications include dietary changes, electrolyte supplementation, and combination use of medications with complementary effects on electrolytes (eg, ACEi plus thiazide-type or loop diuretic, which may normalize potassium levels) (Section 5.2.6, "Medication Interactions"). Treatment of hyperkalemia, other than emergency treatment for life-threatening hyperkalemia, can also be managed with initiation of potassium-lowering binders (including patiromer and sodium zirconium cyclosilicate), noting the importance of taking them (primarily patiromer) mid-day apart from other medications to avoid interfering with absorption.[4,5] If severe or life-threatening electrolyte imbalances occur, the causative medication should be discontinued and the imbalance treated immediately.

5.2.9. Kidney Dysfunction/Injury

Synopsis

Estimated GFR using serum creatinine should be measured 2 to 4 weeks after initiation or dose titration of antihypertensive medications. Renin-angiotensin-aldosterone system inhibitor (RAASi) (including ACEi, ARB, and MRA) may lead to an expected reduction, or dip, in eGFR of up to 30% via vasodilation of efferent arterioles.[1-3] This expected short-term dip in eGFR is associated with preservation of kidney function in the long-term[4-10] and should not lead to discontinuation of the RAASi unless the decline in eGFR is persistently >30%. A referral to a nephrologist is appropriate for evaluation for other causes of AKI, CKD progression, and possible renal artery stenosis. The presence of new kidney dysfunction/injury may also be observed with the addition or dose increase of diuretics. This should prompt evaluation of volume status to rule out hypovolemia and other possible causes of kidney dysfunction. It may be appropriate to initially hold or reduce the diuretic dose and then advance more slowly.

5.3. Comorbidities

Synopsis

Hypertension-related target organ damage describes adverse structural or functional changes in major organ systems, including the heart, vasculature, kidneys, brain, and retina due to hypertension.[1,2] Common forms of target organ damage include left ventricular hypertrophy, HF, subclinical and clinical atherosclerosis, CKD (ie, reduced eGFR or albuminuria), and cerebrovascular disease (eg, stroke, dementia, retinopathy).[3] Numerous studies demonstrate an association between hypertension and target organ damage,[4-9] and longitudinal data indicate at least 1 form of hypertension-related target organ damage is present in >50% of individuals with hypertension.[1] Recent studies also demonstrate relationships between the severity of hypertension and the number of organs affected by hypertension,[10] as well as the number of affected organs and increased CVD risk.[11] Although there are strong data linking hypertension to target organ damage, recommendations on screening and management of different types of target organ damage beyond hypertension treatment are lacking.[12] The goals of preventing target organ damage and its progression from asymptomatic to symptomatic target organ damage can be achieved by focusing on hypertension control.[13] Future studies are needed to inform how hypertension-related target organ damage should be diagnosed and managed among patients with hypertension.

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.[1,7-9]

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