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En la revista *Journal of Hypertension* se publicado la Guía de Práctica Clínica para el tratamiento de la hipertensión arterial de la Sociedad Europea de Hipertensión (*ESH*), avalada por la Asociación Renal Europea (*ERA*) y la Sociedad Internacional de Hipertensión (*ISH*), que consta de 22 secciones que sobre temas relacionados con el diagnóstico y el tratamiento de la hipertensión.

Los cambios más relevantes con respecto a la guía previa de 2018 son:

- Mayor importancia de las mediciones de presión arterial fuera de la consulta.
- Utilización de los suplementos de potasio como recomendación relacionada con el estilo de vida.
- Consejos más explícitos sobre el inicio de tratamiento con medicamentos betabloqueantes.
- Consideraciones sobre la denervación renal, en adición o como alternativa al aumento de la medicación, en pacientes con hipertensión resistente no controlada.
- Enfoque simplificado del tratamiento con antihipertensivos en pacientes con insuficiencia cardíaca.
- Recomendaciones para el tratamiento de pacientes con enfermedad renal crónica, incluyendo el uso de los inhibidores selectivos del transportador renal de glucosa (iSGLT-2 o gliflozinas) y la finerenona, que es un antagonista mineralocorticoide no esteroideo.
- Terapia antihipertensiva para prevenir el deterioro cognitivo y la progresión a demencia.

Además, se hacen pequeñas modificaciones en recomendaciones sobre la medición, clasificación, intervenciones en el estilo de vida, comienzo y elección del tratamiento con medicamentos antihipertensivos, uso de combinaciones a dosis fijas en una sola forma farmacéutica y aumento de la dosis en adultos con control inadecuado.

Relacionado con este tema se puede consultar el BTA del 2020 sobre tratamiento de la hipertensión arterial: nuevas guías, que incluye consideraciones sobre la guía europea anterior a esta actualización.

A continuación, y a modo de ejemplo, se reproducen la figura 12 sobre estrategia general para bajar la presión arterial en pacientes con hipertensión; y las tablas: 15, 20 y 25 sobre: contraindicaciones y condiciones que requieren un uso cauteloso de fármacos para reducir la presión arterial; medicamentos y otras sustancias que pueden aumentar la presión sanguínea; y, tipos de medicamentos, dosis y

características para el tratamiento de emergencia de la hipertensión, respectivamente.

**Figure 12. General bP lowering strategy in patients with Hypertension**

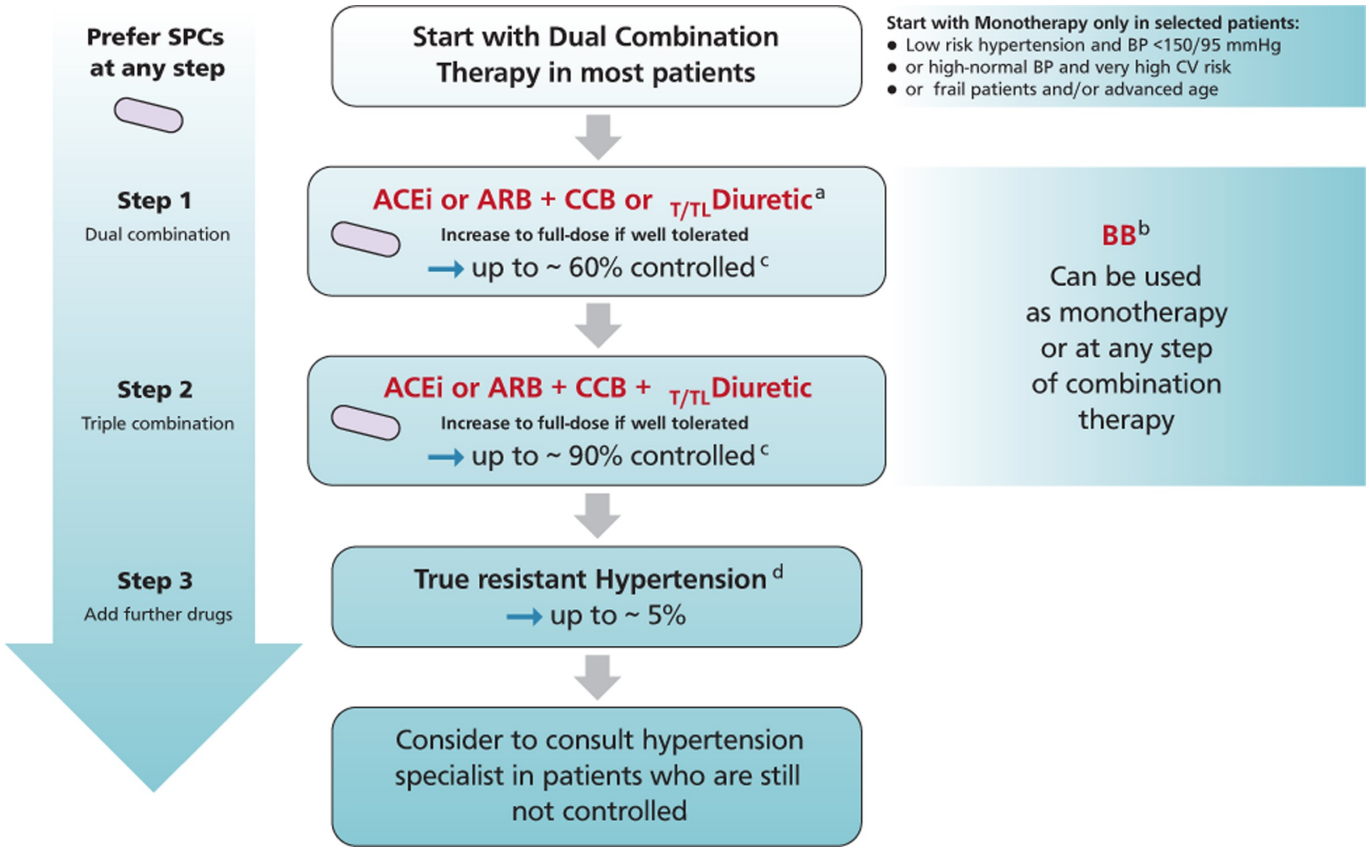

**TABLE 15. Compelling contraindications and conditions requiring cautious use of BP-lowering drugs**

| Drug class | Contraindications | Cautious use |
|---|---|---|
| ACEi | • Pregnancy<br>• Women planning pregnancy<br>• Previous angioneurotic edema<br>• Severe hyperkalemia (e.g. potassium >5.5 mmol/l)<br>• Bilateral renal artery stenosis or stenosis in solitary (functional) kidney | • Women of child-bearing potential without reliable contraception |
| ARB | • Pregnancy<br>• Women planning pregnancy<br>• Severe hyperkalemia (e.g. potassium >5.5 mmol/l)<br>• Bilateral renal artery stenosis or stenosis in solitary (functional) kidney | • Women of child-bearing potential without reliable contraception |
| Beta-blocker | • Severe asthma<br>• Any high-grade sino-atrial or atrioventricular block<br>• Bradycardia (e.g. heart rate <60 bpm) | • Asthma<br>• Glucose intolerance<br>• Athletes and physically active patients |
| DHP-CCB | | • Tachyarrhythmia<br>• Heart failure (HFrEF, class III or IV)<br>• Preexisting severe leg edema |
| Non-DHP-CCB (verapamil, diltiazem) | • Any high-grade sino-atrial or AV block<br>• Severe LV dysfunction (LV EF <40%), HFrEF<br>• Bradycardia (e.g. heart rate <60 bpm)<br>• Co-medications susceptible to significant drug interactions mediated by P-gp or CYP3A4 | • Constipation |
| Thiazide/Thiazide-like diuretics | • Hyponatremia<br>• CKD due to obstructive uropathy<br>• Sulfonamide allergies | • Gout<br>• Glucose intolerance<br>• Pregnancy<br>• Hypercalcemia<br>• Hypokalemia<br>• Cancer patients with bone metastasis |
| MRA | • Severe hyperkalemia (e.g. potassium >5.5 mmol/l)<br>• eGFR <30 ml/min/1.73 $m^2$ | • Co-medications susceptible to significant drug interactions mediated by P-gp or CYP3A4 for eplerenone |

**TABLE 20. Medications and other substances that may increase BP**

| Medication/substance | Proposed mechanism | Comments |
|---|---|---|
| NSAIDs | Inhibition of COX-1 and 2 decreasing PG I2 and E2 synthesis with subsequent reduction in urinary Na excretion and an increased systemic vascular resistance. | Mild, dose-dependent increase in BP. Increased risk with age, preexisting hypertension, salt-sensitive patients, patients with renovascular hypertension. |
| Paracetamol (acetaminophen) | Presumably via inhibition of cyclooxygenases and reduced production of prostaglandins. | Increased relative risk of 1.34 of hypertension with almost daily paracetamol use. |
| Estrogens and progestins | Increased renin synthesis (by estrogens) leading to RAS activation and subsequent Na and water retention. | Mild, sustained increase in BP (6/3 mmHg increase with high doses of estrogen (>50 μg of estrogen and 1–4 μg progestin) but can be severe, common in premenopausal women, cause hypertension in 5% of women. |
| Glucocorticoids | Enhanced Na reabsorption and fluid retention via stimulation of mineralocorticoid receptors.<br>Increased systemic vascular resistance due to upregulation of AT1 receptors on vascular smooth muscle cells. | Dose-dependent, low doses have less effect on BP, more common in older patients, or with a family history of primary hypertension. |
| Calcineurin inhibitors | Reduced NO production, ET-1 overproduction, systemic and renal vasoconstriction, renal Na retention. | Dose-dependent, mild-to-moderate increase in BP. Severe hypertension has been reported. Increased risk with preexisting hypertension, elevated creatinine levels and maintenance therapy with corticosteroids. See Section 20.8.2 |
| Antidepressants<br>SNRIs | Increased noradrenaline release causing adrenergic activation and increased SNS activity. | Dose-dependent, mild (2/1 mmHg) increase in BP. |
| Nasal decongestants | Vasoconstriction due to stimulation of alpha-1 receptors on vascular smooth muscles. | Dose-dependent, sustained increase in BP. |
| Erythropoietin-stimulating agents | Increased thromboxane, reduced prostacyclin levels and activation of the local RAS.<br>Increased ET-1 production, decreased NO synthesis with subsequent vasoconstriction. | Dose-dependent, mild increase in BP, increased risk with preexisting hypertension, or when the initial hematocrit level is low. See Section 20.8.2 |
| Stimulants<br><br>- Modafinil<br>- Amphetamines<br>- Methylphenidate | <br><br>Block noradrenaline or dopamine reuptake.<br>Promote release of catecholamines | Caffeine may cause persistent BP effects with regular consumption.<br><br>Genetic polymorphisms may affect BP response. |
| VEGF inhibitors | Decreased NO production via VEGFR-2 antagonism and stimulation of ET-1 receptors promoting vasoconstriction. | A class effect. The incidence of hypertension is dose-related, risk is increased by preexisting hypertension, old age and overweight. See Section 20.8.2. |
| Substances of abuse<br><br>- MDMA<br>- PCP<br>- Methamphetamine<br><br>- Cocaine<br><br>- Alcohol | Increased release and inhibited reuptake of monoamine neurotransmitters with subsequent SNS activation.<br>Increased CNS catecholamine release with decreased neuronal uptake.<br><br>Cocaine induces an increase in arterial wall stiffness and atherosclerosis.<br><br>Alcohol increases SNS and RAS activity. | Cocaine induces acute but not chronic increase in BP.<br><br>Alcohol causes a dose-dependent, sustained increase in BP independent from obesity or salt intake. |
| Herbal products<br><br>- Licorice<br>- Ephedra<br>- St. John's wort<br>- Yohimbine<br>- Ginseng (high doses)<br>- Ma huang | Chronic excessive liquorice use mimics hyperaldosteronism by stimulating the mineralocorticoid receptor and inhibiting cortisol metabolism.<br>Ephedra activates the alpha-1 receptor increasing SNS activity. | Licorice: Dose-dependent, sustained increase in BP characterized by hypokalemia, metabolic alkalosis and suppressed plasma renin activity and aldosterone levels<br>Yohimbine causes acute, dose-dependent increase in BP. |
| Diet pills<br><br>- Sibutramine<br>- Phenylpropanolamine | Increased levels of norepinephrine with subsequent activation of noradrenergic transmission | Mild increase in BP. |

SNRI, serotonin–noradrenaline reuptake inhibitors.

**Table 25. Drug types, dose and characteristics for treatment of hypertension emergencies**

| Drug | Onset of action | Duration of action | Dose | Contraindications | Adverse effects |
|---|---|---|---|---|---|
| Esmolol | 1 min | 10–30 min | 0.5–1 mg/kg i.v. bolus; 50–300 µg/kg/min i.v. infusion | Second-degree or third-degree AV block, systolic heart failure, asthma, bradycardia | Bradycardia |
| Metoprolol | 1–2 min | 5–8 h | 2.5–5 mg i.v. bolus over 2 min; may repeat every 5 min to a maximum dose of 15 mg | Second-degree or third-degree AV block, systolic heart failure, asthma, bradycardia | Bradycardia |
| Labetalol[a] | 5–10 min | 3–6 h | 10–20 mg i.v. bolus in 1 min; incremental doses ≥20 mg may be administered i.v. at 10 min intervals (max 80 mg) or 1–3 mg/min i.v. infusion until goal BP is reached | Second-degree or third-degree degree AV block; systolic heart failure, asthma, bradycardia | Bronchocostriction, fetal bradycardia |
| Fenoldopam | 5–15 min | 30–60 min | 0.1–0.3 µg/kg/min i.v. infusion, increase every 15 min with 0.1 µg/kg/min increments until goal BP is reached | Caution in glaucoma | |
| Clevidipine | 2 min | 10 min | 1–2 mg/h i.v. infusion, increase every 2 min with 2 mg/h until goal BP, then titrate by smaller increments every 5–10 min | | Headache, reflex tachycardia |
| Nicardipine | 5–15 min | 4–6 h | 5–15 mg/h i.v. infusion, starting dose 5 mg/h, increase every 15–30 min with 2.5 mg until goal BP, maximum 15 mg/h | Liver failure | Headache, reflex tachycardia |
| Nitrogly-cerine | 1–5 min | 5–10 min | 5–200 µg/min i.v. infusion, 5 µg/min increase every 5 min | | Headache, reflex tachycardia |
| Nitro-prusside | Immediate | 1–3 min | 0.3–0.5 µg/kg/min i.v. infusion, increase by 0.5 µg/kg/min every 5 min until goal BP (maximum dose 10 µg/kg/min) | Liver/kidney failure (relative) | Cyanide intoxication |
| Enalaprilat | 5–15 min | 4–6 h | 0.62–1.25 mg i.v. bolus given over 5 min every 6 h | History of angioedema | |
| Urapidil | 3–5 min | 4–6 h | 12.5–25 mg i.v. bolus; 5–40 mg/h as continuous infusion | | |
| Clonidine | 30 min | 4–6 h | 0.2–0.5 µg/kg/min i.v. | | Sedation, rebound hypertension |
| Phentol-amine | 1–2 min | 10–30 min | 1–5 mg i.v. bolus or continuous i.v. infusion at a rate of 0.5–20 µg/kg/min | | Tachyarrhythmia, chest pain |

[a]Not available in serveral countries.