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Antihypertensive Drugs: An Overview of Uses, Doses, Side-effects, Nursing Intervention Protocols, and Contraindications-An Updated Review

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Abstract:

Background: Hypertension (HTN) is a major contributor to cardiovascular diseases, classified by the 2017 American College of Cardiology (ACC) and American Heart Association (AHA) guidelines into different stages. Stage 1 and 2 HTN require interventions such as lifestyle modifications and pharmacological treatment, depending on the patient's risk factors, including atherosclerotic cardiovascular disease (ASCVD) risk, chronic kidney disease, and type 2 diabetes mellitus.

Aim: This review aims to provide an updated overview of antihypertensive drugs, focusing on their uses, doses, side effects, contraindications, nursing intervention protocols, and effectiveness in treating hypertension.

Methods: A review of existing literature on antihypertensive agents, including diuretics, calcium channel blockers (CCBs), ACE inhibitors, ARBs, and beta-blockers. The pharmacodynamics, indications, side effects, and clinical considerations associated with these drug classes were explored.

Results: First-line therapies include thiazide-type diuretics, CCBs, ACE inhibitors, and ARBs. Diuretics, especially chlorthalidone, show superior efficacy in lowering blood pressure (BP). CCBs, including dihydropyridines like amlodipine, are effective vasodilators, while ACE inhibitors and ARBs offer cardioprotective effects, particularly in high-risk populations. Beta-blockers are typically reserved for specific conditions like heart failure or post-myocardial infarction. Combination therapies enhance BP control when monotherapy fails.

Conclusion: Antihypertensive drugs, particularly thiazide diuretics, CCBs, ACE inhibitors, and ARBs, are vital in managing hypertension and preventing cardiovascular events. When monotherapy is ineffective, combination therapy offers superior results, particularly in high-risk patients. Clinicians should consider patient-specific factors such as race, age, and comorbidities when prescribing antihypertensive medications.

Keywords: Hypertension, antihypertensive drugs, thiazide diuretics, calcium channel blockers, ACE inhibitors, ARBs, beta-blockers, combination therapy.

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Introduction:

Hypertension (HTN) is recognized as one of the most significant contributors to the prevalence of cardiovascular diseases. The 2017 guidelines issued by the American College of Cardiology (ACC) and the American Heart Association (AHA) classified HTN into various stages. Normal blood pressure (BP) is defined as a systolic BP below 120 mm Hg and a diastolic BP below 80 mm Hg. Elevated BP is characterized by a systolic BP between 120 and 130 mm Hg and a diastolic BP below 80 mm Hg. Stage 1 HTN is diagnosed with a systolic BP ranging from 130 to 139 mm Hg or a diastolic BP between 80 and 89 mm Hg. Stage 2 HTN is marked by a systolic BP of at least 140 mm Hg or a diastolic BP of at least 90 mm Hg, while hypertensive crises involve a systolic BP exceeding 180 mm Hg and/or a diastolic BP exceeding 120 mm Hg. The 2019 ACC/AHA guidelines for the primary prevention of cardiovascular disease recommend that all patients with elevated BP initiate lifestyle modifications. These include weight management, adherence to a heart-healthy diet, increased physical activity, reduced sodium intake, and moderated alcohol consumption. For individuals with Stage 1 HTN, antihypertensive medications are advised if their 10-year ASCVD (atherosclerotic cardiovascular disease) risk exceeds 10%, with a target BP below 130/80 mm Hg to reduce the likelihood of cardiovascular events. Those with a 10-year ASCVD risk below 10% are encouraged to adopt lifestyle modification measures as an initial approach. For patients with Stage 2 HTN, antihypertensive therapy is recommended regardless of their ASCVD risk, with the goal of achieving a BP level below 130/80 mm Hg. In patients with chronic kidney disease, the target BP remains 130/80 mm Hg. Similarly, for patients diagnosed with type 2 diabetes mellitus (T2DM), antihypertensive therapy is recommended if BP exceeds 130/80 mm Hg, aiming for a BP below this threshold.

Pharmacological Treatments:

Pharmacological treatment often begins with monotherapy, especially after unsuccessful lifestyle interventions. Combination therapy becomes necessary for cases where monotherapy proves inadequate. Lowering BP to a systolic level below 130 mm Hg has demonstrated significant reductions in cardiovascular risks, thereby preventing complications such as heart failure, diabetes, coronary artery disease, and stroke. The efficacy of initial monotherapy can be influenced by factors such as age and race. Various classes of antihypertensive drugs are available, with thiazide-type diuretics, calcium channel blockers (CCBs), and angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs) being the preferred first-line therapies.

Diuretics:

Thiazide and thiazide-like diuretics are frequently the initial choice for managing hypertension. According to the JNC8 guidelines, these diuretics can be employed either as monotherapy or in combination with other antihypertensive agents across all age groups and races, provided there is no evidence of chronic kidney disease. The Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT) endorsed thiazide diuretics as a first-line treatment unless contraindicated. Research comparing various thiazide-type diuretics has highlighted the superior efficacy of chlorthalidone and indapamide over hydrochlorothiazide in preventing cardiovascular diseases. Chlorthalidone is particularly recommended as monotherapy, demonstrating greater effectiveness in controlling BP and reducing mortality and morbidity. Studies have shown that transitioning from hydrochlorothiazide to chlorthalidone results in significant reductions in systolic BP.

Calcium Channel Blockers (CCGs):

Calcium channel blockers (CCBs) are also recommended as first-line antihypertensive agents in the JNC8 guidelines, either as monotherapy or combined with other medications. These agents are effective across all demographics except in cases of chronic kidney disease, where ACE inhibitors or ARBs are preferred. CCBs are categorized into dihydropyridines, such as nifedipine and amlodipine, which primarily act as vasodilators, and non-dihydropyridines, which have additional effects on cardiac contractility and conduction. The dihydropyridines are predominantly used for HTN management, while non-dihydropyridines are reserved for arrhythmias. In patients of African descent without heart failure or chronic kidney disease, CCBs or thiazide diuretics are recommended as initial therapy. Long-acting nifedipine has demonstrated a stronger antihypertensive effect compared to amlodipine, though both are effective in reducing cardiovascular risk.

ACE Inhibitors:

ACE inhibitors and ARBs are particularly suitable for patients with heart failure or chronic kidney disease, especially those with proteinuria. These classes are also recommended for non-Black patients as first-line therapy. Beyond their antihypertensive effects, ACE inhibitors and ARBs provide cardioprotective benefits, especially in individuals at high cardiovascular risk. Research has shown that ACE inhibitors such as ramipril and perindopril significantly reduce mortality and cardiovascular events in specific patient populations. Similarly, ARBs such as losartan have proven effective in lowering BP and reducing morbidity and mortality.

Beta-Blockers:

Beta-blockers are not generally recommended as first-line treatment for hypertension unless specific conditions such as heart failure or myocardial infarction are present. While they offer benefits in reducing cardiovascular morbidity and mortality among younger patients, they are less protective in older adults and have been associated with a higher risk of stroke.

Combination Therapy:

Combination therapy should be considered when a patient fails to respond to monotherapy for hypertension (HTN). Specifically, combining two antihypertensive medications is a viable treatment option for patients with stage 2 hypertension [6]. One study indicated that the reduction in blood pressure achieved by combining drugs from two different classes is approximately five times more significant than doubling the dose of a single medication [18]. The combination of an angiotensin receptor blocker (ARB) with a diuretic or an ACE inhibitor with a calcium channel blocker (CCB) has been shown to be more effective than the combination of a beta-blocker with a diuretic [19]. The latter combination is associated with a higher incidence of diabetes. Beta-blocker combinations should be reserved for patients with specific conditions, such as heart failure, tachycardia, or those recovering from a myocardial infarction (MI). Additionally, a combination of a thiazide diuretic with a potassium-sparing diuretic is equally effective as CCB monotherapy in managing hypertension and results in a lower incidence of hypokalemia compared to hydrochlorothiazide monotherapy [19]. Combination formulations involving CCBs and diuretics are less common, with ARB or ACE inhibitor-based combinations being preferred for patients requiring a combined approach. These combinations are particularly beneficial for patients with chronic kidney disease (CKD) [19][20]. The combination of benazepril and amlodipine is more effective than benazepril and hydrochlorothiazide in reducing the incidence of cardiovascular events and in slowing the progression of nephropathy in high-risk patients [21][22]. However, combining ACE inhibitors and ARBs is not recommended due to an increased incidence of side effects without additional benefits [14][23]. In cases where a combination of two medications does not achieve the desired treatment goal, the addition of a third agent is often necessary. Typically, this involves introducing a third agent from the first-line antihypertensive drug classes, such as thiazide-like diuretics, CCBs, ACE inhibitors, or ARBs. If the three-drug regimen fails, clinicians should consider treatment for resistant hypertension, adding a fourth antihypertensive agent from a different class [19].

Loop diuretics are more effective than thiazides in patients with a low estimated glomerular filtration rate (less than 30 mL/min). These agents are approved for the treatment of peripheral edema, including that associated with congestive heart failure and other non-cardiac causes of edema, such as liver and kidney diseases [24]. However, loop diuretics are not considered first-line agents for hypertension treatment [25]. Potassium-sparing diuretics, specifically mineralocorticoid receptor antagonists like spironolactone and eplerenone, are not typically used as first-line treatments for hypertension. These medications are beneficial when added to other antihypertensive drugs in patients with resistant hypertension. They are particularly effective when incorporated into a triple-drug regimen, though caution is advised when combined with ACE inhibitors or ARBs due to the increased risk of hyperkalemia [26][27]. Additionally, spironolactone has been found to be more effective than doxazosin and bisoprolol in lowering blood pressure when added to first-line antihypertensive agents in the treatment of resistant hypertension [28]. Hydralazine may be considered as an adjunct in managing resistant hypertension, either alone or in combination with nitrates in patients with heart failure. Hydralazine can increase sympathetic tone and sodium retention, so it is often combined with a beta-blocker and loop diuretics to counter these effects [9]. Clonidine, a central alpha-2 agonist, is not a first-line treatment but can be added as an additional agent when combination therapy fails. The transdermal form of clonidine is preferred due to ease of use [9]. Minoxidil is typically used when hydralazine fails to control blood pressure. It is generally effective but may cause fluid retention, which can be managed with a loop diuretic. Additionally, minoxidil increases sympathetic tone, so a beta-blocker may also be necessary [9]. Alpha-blockers are not recommended as first-line agents for hypertension, as they are less effective in preventing cardiovascular events compared to other first-line antihypertensive medications [6].

Mechanism of Action:

The mechanism of action for thiazide and thiazide-like diuretics is not fully understood, although it is known that these diuretics primarily inhibit sodium transport in the distal tubule by blocking the Na/Cl channels [29]. While thiazides may have a minor effect on the proximal tubule by impairing sodium transport, their main action is localized to the distal tubule. Initially, thiazide diuretics cause volume depletion, leading to a reduction in cardiac output; however, this effect typically resolves within 6 to 8 weeks due to a reverse autoregulation mechanism, while blood pressure remains controlled. Thiazide diuretics can acutely activate the renin-angiotensin system, resulting in increased systemic vascular resistance, which may hinder the efficacy of the diuretic. This increase in renin-angiotensin activity is often mitigated with prolonged thiazide therapy, and the addition of an ACE inhibitor or ARB can further enhance blood pressure control. Thiazide diuretics also possess a modest vasodilatory effect, though the precise mechanism remains unclear.

Calcium channel blockers (CCBs) exert their effects by inhibiting the entry of calcium ions (Ca2+) into cells, primarily through binding to L-type voltage-gated calcium channels in the heart muscle. This inhibition results in peripheral vasodilation, a characteristic observed mainly with dihydropyridine CCBs, or a negative inotropic effect on the heart muscle with non-dihydropyridine CCBs, which inhibits sinoatrial and atrioventricular node activity, slowing cardiac contractility and conduction [30]. ACE inhibitors reduce blood pressure by blocking the angiotensin-converting enzyme, thereby decreasing the production of angiotensin II and increasing bradykinin levels through the inhibition of its degradation, leading to vasodilation [31]. In contrast, angiotensin II receptor blockers (ARBs) work by inhibiting the binding of angiotensin II to the AT1 receptors, effectively preventing the actions of angiotensin II. Unlike ACE inhibitors, ARBs do not affect bradykinin levels. Beta-blockers function by preventing catecholamines from binding to beta-1, beta-2, and beta-3 receptors. Beta-1 receptors, predominantly located in the heart, are the primary targets for most beta-blockers, while beta-2 receptors are found in the bronchial and peripheral vascular smooth muscles, and beta-3 receptors are located in adipose tissue. Cardioselective beta-blockers, such as metoprolol succinate, metoprolol tartrate, atenolol, betaxolol, and acebutolol, specifically block beta-1 receptors, reducing the likelihood of bronchospasm. By inhibiting catecholamine binding, beta-blockers exert a negative inotropic effect, decreasing heart rate and reducing oxygen consumption [32].

Loop diuretics act by increasing sodium excretion at the level of the medullary and cortical portions of the thick ascending limb of the nephron, which results in a reduction in volume and consequently lowers blood pressure [24]. Potassium-sparing diuretics, including spironolactone and eplerenone, act on the principal cells of the late distal tubule and the collecting duct. These drugs inhibit sodium reabsorption at this site, thereby reducing potassium and hydrogen ion excretion, and are classified as mineralocorticoid receptor antagonists that block the mineralocorticoid receptor. Hydralazine, an arteriolar vasodilator, works by inhibiting the release of calcium from smooth muscle cells in the blood vessels, reducing the cytoplasmic concentration of calcium, leading to vasodilation [33]. Clonidine, a central alpha-2 adrenergic agonist, reduces sympathetic outflow from the central nervous system by stimulating alpha-2 receptors in the rostral ventrolateral medulla. This results in decreased plasma norepinephrine levels and a reduction in cardiac output. Minoxidil, another arteriolar vasodilator, opens ATP-sensitive potassium channels in the smooth muscle of blood vessels, leading to vasodilation. Alpha-blockers function by inhibiting alpha-1 receptors, which results in reduced vascular smooth muscle contraction and subsequent vasodilation [34].

Administration:

Thiazide-type diuretics are administered exclusively in oral form. Hydrochlorothiazide is available in tablet doses of 12.5 and 25 mg, with the daily dosage typically reaching up to 50 mg, while chlorthalidone is available in 25 and 50 mg tablets, with a maximum daily dose of 100 mg [29]. Dihydropyridine calcium channel blockers are given orally, with the maximum dose for amlodipine being 10 mg daily [35], and nifedipine extended-release tablets having a maximum daily dose of 120 mg [36]. Non-dihydropyridine CCBs, such as diltiazem and verapamil, are available in both oral and intravenous forms, with diltiazem being especially useful for controlling heart rate in arrhythmic conditions [37]. The maximum oral dose for diltiazem is 480 mg daily [37], while verapamil is similarly available in both oral and IV forms, with oral verapamil having a maximum daily dose of 480 mg [38]. ACE inhibitors, with the exception of enalapril, are administered orally, while enalapril is also available in an intravenous form [31]. All ARBs are administered orally [39]. Beta-blockers are available in both oral and intravenous forms [32]. Loop diuretics are available in both oral and intravenous preparations, and potassium-sparing diuretics are primarily used orally [24]. Hydralazine is available in both oral and intravenous formulations, with the maximum oral dose being 300 mg daily [33]. Clonidine is typically administered in a transdermal form, as the oral form is associated with a higher risk of rebound hypertension. The maximum dose for transdermal clonidine is 0.3 mg weekly, while the oral immediate-release form can be taken up to 0.3 mg three times daily [40]. Minoxidil is administered orally for hypertension treatment, and alpha-blockers are also used orally for the management of hypertension [41].

Adverse Effects:

Thiazide and thiazide-like diuretics are associated with various side effects, most of which are dose-dependent. The most common metabolic adverse effects include hypokalemia and hyponatremia, followed by hyperuricemia, hypomagnesemia, hyperlipidemia, and elevated glucose levels [42][29]. A study identified chlorthalidone as increasing the risk of hospitalization due to severe hypokalemia in older individuals. Non-dose-related adverse effects of thiazides may also include sexual dysfunction and sleep disturbances. Calcium channel blockers (CCBs) often result in peripheral edema, particularly with dihydropyridine types. Long-acting nifedipine is associated with a higher incidence of edema compared to amlodipine, and the extent of edema is related to the dose of the CCB. This edema is not attributed to sodium or fluid retention, nor does it lead to heart failure [9]. Since it does not rise from volume overload, it is unresponsive to diuretic therapy. However, the combination of CCBs with ACE inhibitors or angiotensin receptor blockers (ARBs) has shown a lesser degree of edema. Dihydropyridine CCBs may cause lightheadedness, flushing, headaches, and gingival hyperplasia [30]. Non-dihydropyridine CCBs are associated with bradycardia and constipation, affecting up to 25% of patients. CCBs also inhibit platelet aggregation, leading to an increased risk of gastrointestinal bleeding, necessitating caution in older adults and those at high risk of bleeding.

The most common adverse effects of ACE inhibitors include cough, hypotension, fatigue, and azotemia, with reversible renal impairment being particularly frequent when patients experience volume depletion due to factors such as diarrhea or vomiting. Cough occurs in approximately 20% of patients taking ACE inhibitors and may persist for up to 28 days after discontinuation. In contrast, ARBs cause less frequent coughing, and no significant difference in cough incidence has been found when comparing losartan to hydrochlorothiazide. ARBs are also safe for patients with asthma, as candesartan does not increase the incidence of cough compared to CCBs. Ramipril has been linked to a higher rate of cough than telmisartan

[14]. ACE inhibitors are typically associated with mild hyperkalemia, with an increased risk in patients with renal failure, diabetes, or congestive heart failure [43]. Ramipril and telmisartan have similar rates of hyperkalemia, acute kidney injury, and syncope, although telmisartan is linked to a higher incidence of symptomatic hypotension [14]. Angioedema, a rare side effect, occurs in 0.3% of patients on ramipril, and ARBs are less likely to cause this reaction [14]. In Black patients, ARBs have been shown to reduce the incidence of both cough and angioedema [6].

Beta-blockers are commonly associated with bradycardia, constipation, depression, fatigue, and sexual dysfunction. They can also exacerbate bronchospasm and symptoms of peripheral vascular disease, including a flare-up of Raynaud's syndrome [32]. Loop diuretics frequently result in electrolyte imbalances, particularly hypokalemia, hyponatremia, hypomagnesemia, and hypochloremia [24]. Other metabolic side effects include dehydration, hyperuricemia, and hyperlipidemia, while ototoxicity and hearing loss are potential complications of loop diuretic therapy [25]. Mineralocorticoid receptor antagonists primarily cause hyperkalemia. They may also induce metabolic acidosis due to impaired hydrogen ion excretion and can lead to erectile dysfunction and gynecomastia in men, along with irregular menstrual cycles in women [9]. Hydralazine can cause headaches, flushing, palpitations, dizziness, and hypotension due to sympathetic system stimulation [33]. It is also linked to drug-induced lupus erythematosus, hemolytic anemia, and other immune-related conditions [33]. Common side effects of clonidine include drowsiness, headache, dizziness, irritability, nausea, vomiting, constipation, upper abdominal pain, and bradycardia, though more serious effects, such as angioedema, atrioventricular block, and severe hypotension, may also occur [40]. Minoxidil is associated with hirsutism [44]. Alpha-blockers can cause tachycardia and orthostatic hypotension as a result of venous dilation [34].

Thiazide-type diuretics are contraindicated in patients who are anuric or allergic to sulfonamides [29]. Calcium channel blockers are contraindicated in patients with hypersensitivity to the drug [30]. Non-dihydropyridine CCBs should not be used in patients with heart failure, reduced ejection fraction, sick sinus syndrome, or second- or third-degree AV block. Dihydropyridine CCBs are contraindicated in patients with cardiogenic shock, severe aortic stenosis, and unstable angina, and special caution is advised when used in patients with hepatic impairment [36][35]. ACE inhibitors are contraindicated in patients with a history of hypersensitivity or angioedema related to ACE inhibitors, pregnancy, or concurrent use of aliskiren [45]. Relative contraindications for ACE inhibitors include patients with volume depletion, abnormal renal function, or aortic valve stenosis [45]. ARBs should not be used in pregnancy [39], and their combination with ACE inhibitors is also relatively contraindicated. Other relative contraindications for ARB therapy include patients with volume depletion, those taking other medications that cause hyperkalemia, or those with abnormal renal function [39]. Beta-blockers are contraindicated in patients with asthma, particularly nonselective beta-blockers, and should be avoided in cases of hypotension, bradycardia, and cocaine-induced coronary artery spasms [32]. Loop diuretics are contraindicated in patients with hypersensitivity to sulfonamides, anuric patients, and those with hepatic coma [24]. Potassium-sparing diuretics should be avoided in patients with chronic kidney disease or hyperkalemia, and caution is required when used with ACE inhibitors, ARBs, or aliskiren [46]. They are also contraindicated in patients with hypersensitivity to this class of drugs. Clonidine is contraindicated in patients with hypersensitivity to alpha-2 agonists, and it should be avoided in those with depression or recent myocardial infarction [40]. Hydralazine is contraindicated in patients with a history of hydralazine allergy, and it should be used with caution in patients with coronary artery disease or rheumatic mitral valve disease [47][33]. Minoxidil is contraindicated in pregnant or breastfeeding women and those with hypersensitivity to the drug [44]. Contraindications for alpha-blockers include patients with a history of orthostatic hypotension or those using phosphodiesterase inhibitors [41].

Monitoring and Toxicity:

Thiazide and loop diuretics can cause hypokalemia, while potassium-sparing diuretics are known to induce hyperkalemia. As such, it is essential to monitor electrolytes in patients on diuretics, and uric acid levels should also be assessed in patients using thiazides and loop diuretics. Additionally, patients on loop diuretics should be monitored for ototoxicity and deafness. The use of ACE inhibitors and ARBs is often associated with hyperkalemia, and caution is warranted when these agents are combined with potassium-sparing diuretics. Potassium levels must be carefully monitored in patients with chronic kidney disease undergoing treatment with ACE inhibitors, ARBs, or potassium-sparing diuretics. Moreover, ACE inhibitors and ARBs have the potential to cause acute kidney injury, necessitating ongoing renal function monitoring. For patients receiving dihydropyridine calcium channel blockers (CCBs), it is important to monitor hypotension and edema. Non-dihydropyridine CCBs and beta-blockers can lead to bradycardia, and heart rate should be closely monitored, particularly when these two drug classes are combined. Furthermore, in patients on sotalol, monitoring of the QTc interval is recommended. Alpha-blockers should be monitored for tachyarrhythmias and orthostatic hypotension. Complete blood count and antinuclear antibody (ANA) levels should be assessed in patients receiving hydralazine, with special caution if systemic symptoms such as arthralgia or fever arise [42-46].

Toxicity from thiazides and loop diuretics may lead to electrolyte disturbances, primarily hypokalemia and hyponatremia, along with metabolic acidosis, particularly hypochloremic acidosis. Severe dehydration is a potential consequence. Since no antidotes are available for these diuretics, the primary treatment involves volume and electrolyte replacement. In cases of potassium-sparing diuretics toxicity, which manifests severe hyperkalemia, treatment consists of discontinuing all medications causing elevated potassium levels, intravenous hydration, calcium gluconate, insulin with glucose, sodium bicarbonate, and potassium-binding resins. Non-dihydropyridine CCB toxicity results from reduced ionotropy on the cardiac muscle, leading to bradycardia and hypotension. Complete heart block and idioventricular rhythms may occur. Treatment involves intravenous hydration, atropine, and the use of an external pacemaker for bradycardia. Calcium chloride or gluconate may be administered if hydration does not improve blood pressure. In more severe cases, intravenous vasopressors may be necessary. Dihydropyridine CCBs, causing peripheral vasodilation and severe hypotension with less impact on heart rate, require intravenous hydration for hypotension, with atropine or external pacing for bradycardia. ACE inhibitors and ARBs can cause severe hypotension, hyperkalemia, and hyponatremia, for which no antidotes exist. IV hydration and management of hyperkalemia are recommended in these situations. Beta-blockers may result in hypotension, bradycardia, and AV block, requiring the administration of IV glucagon as an antidote, along with IV hydration and external pacing if necessary. Hydralazine toxicity can lead to severe hypotension, tachycardia, and skin flushing, with more severe cases causing cardiac shock or myocardial ischemia. The lack of antidotes means that IV hydration and vasopressors are critical interventions. Beta-blockers may be used for severe tachycardia. Clonidine toxicity can be presented as lethargy, hypotension, bradycardia, and miosis, and in extreme cases, respiratory depression. Management involves supportive care, including hydration, vasopressors, dopamine, and norepinephrine, with IV atropine for severe bradycardia and external pacing in resistant cases. Minoxidil toxicity can cause tachycardia and hypotension, treated with supportive care and vasopressors. Alpha-blocker toxicity can result in severe hypotension, for which IV hydration and vasopressors are the primary treatment options [46-50].

The management of hypertension (HTN) involves a broad range of antihypertensive medications, necessitating careful monitoring of adherence and potential adverse reactions. Effective treatment is essential for preventing cardiovascular diseases, and selecting the appropriate drug class is critical for achieving optimal control with minimal side effects. An interprofessional approach involving clinicians, nurses, and pharmacists is crucial in managing patients on these medications. Initially, the clinician establishes the antihypertensive regimen, followed by pharmacists who focus on monitoring drug-drug interactions, patient adherence, and medication reconciliation. Nurses play a pivotal role in monitoring adherence, identifying barriers to treatment efficacy, and evaluating patients' diets, activity levels, and home environments. Home-visiting nurses can monitor blood pressure and heart rate responses to treatment while detecting early adverse reactions. Pharmacists and nurses must communicate any concerns about adherence, adverse effects, or changes in the home environment to the clinician. This comprehensive interprofessional collaboration ensures the optimal delivery of care and maximizes the benefits of the prescribed regimen for the patient and their family [51].

Nursing Interventions:

Hypertension, a prevalent condition that affects a significant portion of the global population, is a major risk factor for cardiovascular diseases, stroke, and kidney failure. Nurses play a crucial role in the management of hypertension, as they are responsible for monitoring patient health, educating patients, and ensuring proper medication adherence. This article outlines the essential nursing intervention protocols in hypertensive diseases, focusing on the administration and management of antihypertensive drugs.

Assessment and Monitoring

The primary nursing intervention in hypertensive patients involves accurate and regular monitoring of blood pressure (BP). Nurses must assess BP using calibrated, validated devices at regular intervals, ensuring that patients' readings remain within the target range. Continuous monitoring is especially vital for patients on antihypertensive therapy to gauge the efficacy of the prescribed medication and adjust dosages accordingly. Blood pressure measurements should be taken in the seated position, after the patient has rested for at least five minutes, with the appropriate cuff size to ensure accuracy. The readings should be interpreted in the context of the patient's medical history, including other comorbidities such as diabetes or kidney disease. In addition to BP monitoring, nurses should assess other clinical parameters that are affected by hypertension, such as heart rate, respiratory rate, and renal function. Routine laboratory tests are essential, particularly for monitoring renal function (e.g., serum creatinine and glomerular filtration rate) and electrolytes (e.g., potassium levels), as antihypertensive medications, including diuretics, can alter these values. Nurses should also monitor for side effects of antihypertensive drugs, including dizziness, fatigue, or any signs of electrolyte imbalances, such as muscle weakness or cramps.

Medication Management

Antihypertensive drugs are commonly prescribed to manage high blood pressure, and their proper administration is a central nursing responsibility. Nurses must be knowledgeable about the various classes of antihypertensive drugs and their mechanisms of action, side effects, and contraindications. Common drug classes include diuretics, beta-blockers, ACE inhibitors, calcium channel blockers (CCBs), and angiotensin II receptor blockers (ARBs). The nursing intervention protocols include:

1. **Diuretics** (e.g., thiazide diuretics) are often the first line of treatment for hypertension. Nurses must monitor for signs of dehydration, hypokalemia, and metabolic alkalosis, common side effects of diuretics. They should educate patients on the importance of staying hydrated and maintaining potassium intake (e.g., consuming potassium-rich foods or taking potassium supplements, if recommended).

2. **Beta-blockers** (e.g., metoprolol, atenolol) are used to reduce heart rate and lower BP. Nurses should monitor heart rate and assess for bradycardia. Special caution is necessary when administering these drugs to patients with asthma or other respiratory conditions due to the potential for bronchospasm. Educating patients about the risks of abrupt discontinuation of beta-blockers is essential, as this can lead to rebound hypertension or angina.

3. **ACE Inhibitors and ARBs** (e.g., lisinopril, losartan) are commonly used to treat hypertension, especially in patients with diabetes or chronic kidney disease. Nurses must monitor kidney function and electrolytes, as these medications can cause hyperkalemia and renal dysfunction. Patients should be informed about the potential side effect of a persistent dry cough with ACE inhibitors and should be advised to report any such symptoms promptly.

4. **Calcium Channel Blockers** (e.g., amlodipine) help reduce BP by relaxing blood vessel walls. Nurses should monitor side effects such as peripheral edema, dizziness, and palpitations. If patients experience swelling in the lower extremities, the nurse should assess for fluid retention and adjust the management plan accordingly.

5. **Other agents**, such as alpha-blockers, vasodilators, and direct renin inhibitors, may be used in more complex cases of hypertension or when other medications are not effective. Nurses must be vigilant in monitoring side effects such as hypotension, tachycardia, or orthostatic hypotension. They should

educate patients on how to manage orthostatic hypotension by slowly rising from a seated position to prevent falls.

Patient Education

A critical component of nursing intervention is patient education regarding lifestyle modifications and adherence to antihypertensive medication regimens. Nurses should provide patients with detailed information on the importance of lifestyle changes, such as weight loss, dietary modifications (e.g., reducing salt intake, increasing fruit and vegetable consumption, and following the DASH diet), and regular physical activity. Smoking cessation and limiting alcohol intake are also important aspects of hypertension management. Patients should be educated on the potential side effects of their medications and the importance of adhering to their prescribed drug regimen. Nurses should discuss the significance of taking medications regularly and not discontinuing treatment without consulting their healthcare provider. This is particularly important for patients on beta-blockers or ACE inhibitors, where sudden withdrawal can result in rebound effects. Nurses should assess any barriers to adherence, such as financial constraints, side effects, or forgetfulness, and collaborate with pharmacists to resolve these issues.

Collaboration and Follow-Up

Nurses should collaborate with the broader healthcare team, including physicians, pharmacists, and dietitians, to develop a comprehensive care plan tailored to the individual needs of each patient. This team approach ensures that the patient's condition is managed holistically, addressing all aspects of hypertension, including lifestyle changes, medication management, and the prevention of complications. Regular follow-up appointments are essential for reassessing BP control and adjusting medications as needed. Nurses play a key role in coordinating these follow-ups, ensuring that patients are informed of their BP goals and are aware of the importance of ongoing management. The management of hypertension requires a multifaceted approach, and nurses are at the forefront of ensuring the effective implementation of antihypertensive treatments. By closely monitoring patient health, managing medications, providing education on lifestyle modifications, and fostering collaboration with the healthcare team, nurses help improve patient outcomes and prevent the complications associated with uncontrolled hypertension. Proper nursing interventions are vital in achieving optimal BP control and enhancing the quality of life for hypertensive patients.

Conclusion:

Hypertension (HTN) remains a critical public health issue due to its role in cardiovascular diseases (CVD), necessitating effective treatment strategies. The updated 2017 ACC/AHA guidelines classify HTN into different stages, guiding treatment decisions based on a patient's cardiovascular risk profile. Stage 1 hypertension, characterized by a systolic BP between 130-139 mm Hg or diastolic BP between 80-89 mm Hg, can often be managed initially with lifestyle modifications. However, if the patient's 10-year ASCVD risk exceeds 10%, pharmacological treatment is recommended. Stage 2 hypertension, which involves BP readings exceeding 140/90 mm Hg, generally requires antihypertensive medication regardless of ASCVD risk. Pharmacological treatment begins with monotherapy, with first-line agents such as thiazide diuretics, calcium channel blockers (CCBs), ACE inhibitors, and angiotensin receptor blockers (ARBs). Thiazide diuretics, particularly chlorthalidone, are highly effective in controlling BP and reducing cardiovascular risks. CCBs, especially dihydropyridines like amlodipine, are effective vasodilators that work well across demographic groups. ACE inhibitors and ARBs are essential in treating patients with comorbidities such as chronic kidney disease or heart failure. These drugs offer additional cardioprotective benefits beyond BP lowering. While beta-blockers provide useful cardiovascular benefits in conditions such as heart failure or post-myocardial infarction, they are not recommended as first-line therapy for general hypertension management. Moreover, combination therapy is often necessary for patients with more severe hypertension (Stage 2) or those who do not achieve adequate BP control with a single drug. Combinations of diuretics with ACE inhibitors, ARBs, or CCBs are particularly effective in managing BP, reducing the risk of stroke, and improving patient outcomes. For resistant hypertension, triple or quadruple-drug regimens, including mineralocorticoid receptor antagonists, may be required to achieve the target BP. The review also highlights the importance of personalized treatment strategies that consider factors such as race, age, and comorbidities, which may influence drug efficacy. Clinicians should be cautious when combining antihypertensive agents to minimize side effects, such as hyperkalemia with ACE inhibitors and ARBs, or the increased risk of diabetes with beta-blocker diuretic combinations. In conclusion, managing hypertension effectively requires a multifaceted approach involving lifestyle changes and a careful selection of pharmacological treatments. Antihypertensive medications, especially when combined appropriately, offer significant benefits in preventing cardiovascular complications and improving long-term health outcomes for patients with hypertension.

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الأدوية المضادة لارتفاع ضغط الدم: لمحة عامة عن الاستخدامات، الجرعات، الآثار الجانبية، بروتوكولات تدخل التمريض، والموانع - مراجعة محدثة

الملخص:

الخلفية: يُعد ارتفاع ضغط الدم (HTN) من العوامل الرئيسية المساهمة في أمراض القلب والأوعية الدموية، وتم تصنيفه وفقًا لإرشادات الجمعية الأمريكية لأمراض القلب (ACC) والجمعية الأمريكية للقلب (AHA) لعام 2017 إلى مراحل مختلفة. تتطلب مرحلتا ارتفاع ضغط الدم 1 و2 تدابير تدخلية مثل تعديل نمط الحياة والعلاج الدوائي، وفقًا لعوامل الخطر لدى المريض، بما في ذلك خطر الإصابة بأمراض القلب والأوعية الدموية التصلبية(ASCVD) ، وأمراض الكلى المزمنة، وداء السكري من النوع 2.

الهدف: تهدف هذه المراجعة إلى تقديم لمحة محدثة عن الأدوية المضادة لارتفاع ضغط الدم، مع التركيز على استخداماتها، جرعاتها، آثارها الجانبية، موانع استخدامها، بروتوكولات التدخل التمريضي، وفعاليتها في علاج ارتفاع ضغط الدم.

الطرق: تم مراجعة الأدبيات الحالية حول العوامل المضادة لارتفاع ضغط الدم، بما في ذلك مدرات البول، حاصرات قنوات الكالسيوم(CCBs) ، مثبطات الإنزيم المحول للأنجيوتنسين(ACE) ، حاصرات مستقبلات الأنجيوتنسين(ARBs) ، وحاصرات بيتا. تم استكشاف الديناميكيات الدوائية، المؤشرات، الآثار الجانبية، والاعتبارات السريرية المرتبطة بهذه الفئات الدوائية .

النتائج: تشمل العلاجات الأولية مدرات البول من نوع الثيازيد، حاصرات قنوات الكالسيوم(CCBs) ، مثبطات الإنزيم المحول للأنجيوتنسين(ACE) ، وحاصرات مستقبلات الأنجيوتنسين .(ARBs) تُظهر مدرات البول، خاصة الكلورثاليدون، فعالية متفوقة في خفض ضغط الدم .(BP) تعتبر حاصرات قنوات الكالسيوم، بما في ذلك الديهيدروبيريدينات مثل الأمليوديبين، موسعات للأوعية فعالة، في حين توفر مثبطات الإنزيم المحول للأنجيوتنسين (ACE) وحاصرات مستقبلات الأنجيوتنسين (ARBs)تأثيرات وقائية للقلب، خاصة في الفئات السكانية عالية المخاطر. عادةً ما يتم الاحتفاظ بحاصرات البيتا للحالات الخاصة مثل فشل القلب أو بعد احتشاء عضلة القلب. تعزز العلاجات المجمعة التحكم في ضغط الدم عندما تفشل العلاجات الأحادية.

الخاتمة: تُعد الأدوية المضادة لارتفاع ضغط الدم، خاصة مدرات البول من نوع الثيازيد، وحاصرات قنوات الكالسيوم، ومثبطات الإنزيم المحول للأنجيوتنسين، وحاصرات مستقبلات الأنجيوتنسين، أساسية في إدارة ارتفاع ضغط الدم والوقاية من الأحداث القلبية الوعائية. عندما تكون العلاجات الأحادية غير فعالة، توفر العلاجات المجمعة نتائج أفضل، خاصة في المرضى ذوي المخاطر العالية. يجب على الأطباء أخذ عوامل المريض الخاصة مثل العرق والعمر والأمراض المصاحبة في الاعتبار عند وصف الأدوية المضادة لارتفاع ضغط الدم.

الكلمات المفتاحية: ارتفاع ضغط الدم، الأدوية المضادة لارتفاع ضغط الدم، مدرات البول من نوع الثيازيد، حاصرات قنوات الكالسيوم، مثبطات الإنزيم المحول للأنجيوتنسين، حاصرات مستقبلات الأنجيوتنسين، التمريض، حاصرات البيتا، العلاج المجمّع.