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Textbook of

Cardiovascular & Thoracic NURSING

As per the INC Syllabus for MSc Nursing

P Hariprasath

2nd Edition

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4

CHAPTER

Nursing Management of Patients with Hypertension

LEARNING OBJECTIVES

At the end of this chapter, the learner should be able to:

- Define hypertension and types of hypertension.
- Identify risk factors for hypertension.
- Relate the pathophysiologic mechanisms associated with primary hypertension to the clinical manifestations and complications.
- Describe treatment approaches for hypertension, including lifestyle modifications and medication therapy.

- Types of Hypertension
- Risk Factors for Hypertension
- Causes
- Normal Regulation of Blood Pressure
- Pathophysiology of Primary Hypertension
- Pathophysiology of Secondary Hypertension
- Classification
- Clinical Manifestations
- Complications
- Hypertensive Heart Disease
- Assessment and Diagnostic Studies
- Management of Hypertension

DEFINITION OF HYPERTENSION

According to American Heart Association, high absolute risk of hypertension, is defined as systolic blood pressure (BP) ≥130 mm Hg and/or diastolic BP ≥80 mm Hg or treatment for hypertension. This values should be based on the average of two or more accurate blood pressure measurements taken 1 to 4 weeks apart by a health care provider.

TYPES OF HYPERTENSION

- ***Accelerated hypertension:*** A significant recent increase in blood pressure over previous hypertensive levels, with evidence of vascular damage on fundoscopic examination.
- ***Malignant hypertension:*** Triad of blood pressure > 200/140 mm Hg, grade IV retinopathy (papilledema) and renal dysfunction.
- ***Hypertensive urgency:*** Here blood pressure is markedly elevated without evidence of some end organ damage.
- ***Hypertensive emergency:*** Here blood pressure is markedly elevated with evidence of some end organ damage.
- ***White coat hypertension:*** A transient increase in blood pressure in normal individuals, when BP is recorded by a doctor.
- ***Pseudohypertension:*** A false increase in blood pressure due to stiff and non-compliant vessels (Osler's sign), occurring in old age. Here intra-arterial BP is lower than the BP measured by sphygmomanometer**.**
- ***Transient hypertension:*** An increased blood pressure for a particular period of time and then droping back to normal. It is seen in acute cerebrovascular accidents, acute myocardial infarction, acute glomerulonephritis, pregnancy.
- ***Paradoxical hypertension:*** Here the patients show a sudden increase in BP even on antihypertensive drugs.
- **Hypertensive crisis:** It is a severe and abrupt elevation in BP, where systolic blood pressure more than 180 mm Hg and or diastolic blood pressure more than 120 mm Hg.
- **Isolated systolic hypertension:** Here the systolic blood pressure (SBP) ≥ 130 mm Hg, but the diastolic blood pressure (DBP) < 80 mm Hg.

RISK FACTORS FOR HYPERTENSION

- **Age:** After the age of 55, systolic blood pressure between 130 and 139 mm Hg and diastolic pressure between 85 and 89 mm Hg are associated with increased for clinical cardiovascular disease (CVD) in blacks.

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- ***Alcohol:*** Excessive alcohol intake is strongly associated with hypertension. Patients with hypertension should limit their daily intake to 1 oz of alcohol.
- ***Cigarette smoking:*** Smoking greatly increases the risk of cardiovascular disease. People with hypertension who smoke are at even greater risk for cardiovascular disease.
- ***Diabetes mellitus:*** Hypertension is more common in diabetics. When hypertension and diabetes coexist, complications (e.g. target organ disease) are more severe.
- ***Elevated serum lipids:*** Elevated levels of cholesterol and triglycerides are primary risk factors in atherosclerosis. Hyperlipidemia is more common in people with hypertension.
- ***Excess dietary sodium:*** High sodium intake can contribute to hypertension in some patients and can decrease the effectiveness of certain antihypertensive medications.
- ***Gender:*** Hypertension is more prevalent in men in young adulthood and early middle age (< 55 years of age). After the age of 55, hypertension is more prevalent in women.
- ***Family history:*** History of a close blood relative (e.g. parents, sibling) with hypertension is associated with an increased risk for developing hypertension.
- ***Obesity:*** Weight gain is associated with increased frequency of hypertension. The risk is greatest with central abdominal obesity.
- ***Sedentary lifestyle:*** Regular physical activity can help to control weight and reduce cardiovascular risk. Physical activity may decrease BP. Sedentary lifestyle increases risk of cardiovascular disease.
- ***Socioeconomic status:*** Hypertension is more prevalent in lower socioeconomic groups and among the less educated.
- ***Stress:*** People exposed to repeated stress may develop hypertension more frequently than others. People who develop hypertension may respond differently to stress than those who do not develop hypertension.

CAUSES

The causes of hypertension can be classified as primary and secondary.

Primary Hypertension

Primary (essential or idiopathic) hypertension is elevated BP without an identified cause and accounts for 90 to 95% of all cases of hypertension. But few contributing factors are identified they are:

- Dysfunction of sympathetic nervous system (SNS)
- Dysfunction renin-angiotensin- aldosterone system
- Vasoconstriction
- Increased sodium intake
- Obesity
- Diabetes mellitus
- Insulin resistance
- Stress
- Dyslipidemia
- Excessive alcohol consumption.

Secondary Hypertension

Secondary hypertension is elevated BP with a specific cause that often can be identified and corrected. This type of hypertension accounts for 5 to 10% of hypertension.

- Renal disease such as renal artery stenosis, primary sodium retention, renin- producing tumors parenchymal disease.
- Endocrine disorders such as pheochromocytoma, cushing syndrome, acromegaly, hypothyroidism, hyperthyroidism, and hyperaldosteronism.
- Coarctation or congenital narrowing of the aorta.
- Neurologic disorders such as brain tumors, tetraplegia, Guillain–Barre syndrome and head injury.
- Sleep apnea.
- Cirrhosis of liver.
- Pregnancy-induced hypertension.

NORMAL REGULATION OF BLOOD PRESSURE

Sympathetic Nervous System

The nervous system, which reacts within seconds after a decrease in arterial pressure, increases BP primarily by activation of the sympathetic nervous system (SNS). Increased SNS activity increases HR and cardiac contractility, produces widespread vasoconstriction in the peripheral arterioles, and promotes the release of renin from the kidneys. The net effect of SNS activation is to increase arterial pressure by increasing both CO and SVR.

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The neurotransmitter norepinephrine (NE) is released from SNS nerve endings. NE activates receptors located in the sinoatrial node, myocardium, and vascular smooth muscle. The response to NE depends on the type and density of receptors present. SNS receptors are classified as α_1, α_2, β_1, and β_2. α-adrenergic receptors located in peripheral vasculature cause vasoconstriction when stimulated by NE.

β_1-adrenergic receptors in the heart respond to NE and epinephrine with increased HR *(chronotropic),* increased force of contraction *(inotropic),* and increased speed of conduction. The smooth muscle of the blood vessels has α_1-adrenergic and β_2-adrenergic receptors. β_2-adrenergic receptors are activated primarily by epinephrine released from the adrenal medulla and cause vasodilation. Diminished responsiveness of cardiovascular cells to SNS stimulation is one of the most significant cardiovascular effects of aging.

Vascular Endothelium

The vascular endothelium is a single cell layer that lines the blood vessels. Previously considered inert, it is now known to produce vasoactive substances and growth factors. Nitric oxide, an endothelium-derived relaxing factor (EDRF), helps to maintain low arterial tone at rest, inhibits growth of the smooth muscle layer, and inhibits platelet aggregation. Other substances released by the vascular endothelium with local vasodilator effects include prostacyclin and endothelium-derived hyperpolarizing factor.

Endothelin (ET), produced by the endothelial cells, is an extremely potent vasoconstrictor. There are three subclasses of endothelins—ET-1, ET-2, and ET-3. ET-1 is the most potent endothelin in producing vasoconstriction. ET-1 also causes adhesion and aggregation of neutrophils and stimulates smooth muscle growth. Endothelial cell function and dysfunction is an area of ongoing investigation. There is some evidence that vascular endothelial dysfunction may contribute to atherosclerosis and primary hypertension. The prevention or reversal of endothelial dysfunction may become important for therapeutic interventions in the future.

Renal System

The kidneys contribute to BP regulation by controlling sodium excretion and extracellular fluid (ECF) volume. Sodium retention results in water retention, which causes an increased ECF volume. This increases the venous return to the heart, increasing the stroke volume, which elevates the BP through an increase in CO.

The renin-angiotensin-aldosterone system (RAAS) also plays an important role in BP regulation. In response to SNS stimulation, decreased blood flow through the kidneys, or decreased serum sodium concentration, renin is secreted from the juxtaglomerular apparatus in the kidney. Renin is an enzyme that converts angiotensinogen to angiotensin I. Angiotensin-converting enzyme (ACE) converts angiotensin I into angiotensin II (A-II), which can increase BP by two different mechanisms. First, A-II is a potent vasoconstrictor and increases vascular resistance, resulting in an immediate increase in BP. Second, over a period of hours or days, A-II increases BP indirectly by stimulating the adrenal cortex to secrete aldosterone, which causes sodium and water retention by the kidneys resulting in increased blood volume and increased CO.

Angiotensin II also functions at a local level within the heart and blood vessels. The local vasoactive effects of A-II include vasoconstriction and tissue growth that results in remodeling of the vessel walls. These changes are associated with the development of primary hypertension and also the long-term effects of hypertension (e.g. atherosclerosis, renal disease, cardiac hypertrophy).

Prostaglandins (PGE_2 and PGI_2) secreted by the renal medulla have a vasodilator effect on the systemic circulation. This results in decreased SVR and lowering of BP. The natriuretic peptides (atrial natriuretic peptide [ANP] and β-type natriuretic peptide [BNP]) secreted by cardiac cells antagonize the effects of antidiuretic hormone (ADH) and aldosterone by promoting natriuresis (excretion of sodium in urine) and diuresis resulting in reduced blood volume and a decrease in BP.

PATHOPHYSIOLOGY OF PRIMARY HYPERTENSION

- ***Heredity:*** Genetic abnormalities associated with several rare forms of hypertension have been identified. However, the contribution of genetic factors to BP levels in the general population is very small.
- ***Water and sodium retention:*** Excessive sodium intake may initiate the hypertension in some people. A high sodium intake causes water retention which results in increased extracellular volume and thereby the venous supply to the heart is increased. Increased venous return thereby, increases the cardiac output and blood pressure. People with low sodium intake show little or no hypertension and no progressive increase in BP with age. When sodium is restricted in hypertensive people, their BP falls.
- ***Altered renin-angiotensin mechanism:*** High plasma renin activity results in the increased conversion of angiotensinogen to angiotensin I and it is converted into angiotensin II by angiotensin-converting enzyme (ACE). Angiotensin II cause direct arteriolar constriction, promotes vascular hypertrophy, and induces aldosterone secretion. Thus altered renin-angiotensin mechanisms may contribute to the development and maintenance of hypertension.

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- ***Stress and increased sympathetic nervous system activity:*** Physiologic responses to stress, which are normally protective, may persist to a pathologic degree, resulting in prolonged increase in SNS activity. Increased SNS stimulation produces increased vasoconstriction, increased HR, and increased renin release. Increased renin activates the angiotensin mechanism and increases aldosterone secretion, both leading to elevated BP.
- ***Insulin resistance and hyperinsulinemia:*** Abnormalities of glucose, insulin, and lipoprotein metabolism are common in primary hypertension. High insulin concentration in the blood stimulates SNS activity and impairs nitric oxide-mediated vasodilation. Additional effects of insulin include vascular hypertrophy and increased renal sodium reabsorption.
- ***Endothelial cell dysfunction:*** Vascular endothelial cells are known to be the source of multiple vasoactive substances. Endothelin produces pronounced and prolonged vasoconstriction. The role of endothelial dysfunction in the pathogenesis of hypertension is still under research.

PATHOPHYSIOLOGY OF SECONDARY HYPERTENSION

Renal Parenchymal Disease

Factors contributing to the development of hypertension in patients with renal parenchymal disease are loss of nephrons leading to retention of sodium and water, decreased release of vasodilator substance such as nitric oxide, activation of renin-angiotensin mechanisms.

Renovascular Disease

The most common renovascular disease is renal artery stenosis which leads to renal ischemia, release of renin and results in increased blood pressure.

Primary Hyperaldosteronism

It is characterized by excess secretion of aldosterone which results in retention of sodium, chloride and water which leads to increased extracellular volume and thereby the venous return to the heart is increased. Increased venous return leads to increases in the cardiac output and blood pressure.

Pheochromocytoma

In this condition excessive amount of catecholamine is released in blood, resulting in increased BP.

CLASSIFICATION

Classification of hypertension has been described in **Table 4.1**.

CLINICAL MANIFESTATIONS

Hypertension is often called the "silent killer" because it is frequently asymptomatic until it becomes severe and target organ disease **(Table 4.2)** has occurred. A patient with severe hypertension may experience a variety of symptoms secondary to effects on blood vessels in the various organs and tissues or to the increased workload of the heart. These secondary symptoms include fatigue, reduced activity tolerance, dizziness, palpitations, angina and dyspnea.

Table 4.1: Classification of hypertension.

CLASSIFICATION OF HYPERTENSION			
BP Classification	**SBP (mm Hg)**		**DBP (mm Hg)**
Normal	<120	And	<80
Elevated	120-129	And	<80
Hypertension Stage 1	130-139	Or	80-89
Hypertension, Stage 2	140 or higher	Or	90 or higher
Hypertensive crisis	180 or higher	And/or	Higher than 120

(SBP: systolic blood pressure; DBP: diastolic blood pressure)

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COMPLICATIONS

The most common complications of hypertension are target organ diseases occurring in the heart (hypertensive heart disease), brain (cerebrovascular disease), peripheral vasculature (peripheral vascular disease), kidney (nephrosclerosis), and eyes (retinal damage).

Table 4.2: Clinical manifestations of target organ disease.

Cardiac
• Coronary artery disease • Left ventricular hypertrophy • Left ventricular dysfunction or heart failure
Cerebrovascular
• Transient ischemic attack or stroke • Hypertensive encephalopathy
Peripheral vascular
• One or more major pulses in the extremities (except for dorsalis pedis) reduced or absent • Intermittent claudication • Abdominal or carotid bruits/thrills • Aneurysm
Renal
• Nephrosclerosis • Renal failure • Serum creatinine ≥ 1.5 mg/dl (130 µmol/L) • Proteinuria (1+ or greater) • Microalbuminuria
Retinopathy
• Generalized or focal narrowing of retinal arterioles • Arteriovenous nicking • Hemorrhages or exudates, with or without papilledema

HYPERTENSIVE HEART DISEASE

Coronary Artery Disease

Hypertension is a major risk factor for coronary artery disease (CAD). In people older than 50 years, systolic hypertension is a much more important risk factor for cardiovascular disease than diastolic hypertension. The mechanisms by which hypertension contributes to the development of atherosclerosis are not fully known. The "response-to-injury" hypothesis of atherogenesis suggests that hypertension disrupts the coronary artery endothelium, thus exposing the intimal layer to activated white blood cells and platelets. Growth factors released by the vascular endothelium and platelets may induce smooth muscle proliferation within the lesion. These arteriolar changes result in a stiffened arterial wall and a narrowed internal lumen, and account for a high incidence of CAD and the resulting problems of angina and myocardial infarction (MI).

Left Ventricular Hypertrophy

Sustained high BP increases the cardiac workload and produces left ventricular hypertrophy (LVH). Initially, LVH is an adaptive or compensatory mechanism that strengthens cardiac contraction and increases cardiac output. However, increased contractility increases myocardial work and oxygen consumption. When the heart can no longer meet the demands for myocardial oxygen, heart failure develops. Progressive LVH, especially in association with CAD, is associated with the development of heart failure.

Heart Failure

Heart failure occurs when the heart's compensatory adaptations are overwhelmed and the heart can no longer pump enough blood to meet the metabolic needs of the body. Contractility is depressed, and stroke volume and cardiac output are decreased.

Cerebrovascular Disease

Atherosclerosis is the most common cause of cerebrovascular disease. Hypertension is a major risk factor for cerebral atherosclerosis and stroke. Even in mildly hypertensive people, the risk of stroke is 4 times higher than in normotensive people. Adequate control of BP diminishes the risk of stroke.

Hypertensive encephalopathy may occur after a marked rise in BP if the cerebral blood flow is not decreased by autoregulation. Autoregulation is a physiologic process that maintains constant cerebral blood flow despite fluctuations in arterial blood pressure. Normally as pressure in the cerebral blood vessels rises, the vessels constrict to maintain constant flow. When arterial blood pressure exceeds the body's ability to autoregulate, the cerebral vessels suddenly dilate, capillary permeability increases, and cerebral edema develops, producing a rise in intracranial pressure. If left untreated, patients die quickly from brain damage.

Peripheral Vascular Disease

As it does with other vessels, hypertension speeds up the process of atherosclerosis in the peripheral blood vessels, leading to the development of peripheral vascular disease, aortic aneurysm, and aortic dissection.

Nephrosclerosis

Renal dysfunction is the direct result of ischemia caused by the narrowed lumen of the intrarenal blood vessels. Gradual narrowing of the arteries and arterioles leads to atrophy of the tubules, destruction of the glomeruli, and eventual death of nephrons. Initially intact nephrons can compensate, but these changes may eventually lead to renal failure.

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Retinal Damage

The appearance of the retina provides important information about the severity and duration of the hypertensive process. The retina is the only place in the body where the blood vessels can be directly visualized. Therefore damage to retinal vessels provides an indication of concurrent vessel damage in the heart, brain, and kidney. An ophthalmoscope is used to visualize the blood vessels of the eye. Manifestations of severe retinal damage include blurring of vision, retinal hemorrhage, and loss of vision.

ASSESSMENT AND DIAGNOSTIC STUDIES

History Collection

Family history, lifestyle (exercise, salt intake, smoking habit) and other risk factors should be recorded. A careful history will also identify those patients with drug or alcohol-induced hypertension and may elicit the symptoms of other causes of secondary hypertension such as pheochromocytoma (paroxysmal headache, palpitation and sweating) or complications such as coronary artery disease (e.g. angina, breathlessness).

Physical Assessment

Radio-femoral delay (coarctation of the aorta), enlarged kidneys (polycystic kidney disease), abdominal bruits (renal artery stenosis) and the characteristic facies and habitus of Cushing's syndrome are all examples of physical signs that may help to identify one of the causes of secondary hypertension. Examination may also reveal features of important risk factors such as central obesity and hyperlipidemia.

Non-specific findings may include left ventricular hypertrophy (apical heave), accentuation of the aortic component of the second heart sound, and a fourth heart sound. The optic fundi are often abnormal and there may be evidence of generalized atheroma or specific complications such as aortic aneurysm or peripheral vascular disease.

Diagnostic Studies

- Urinalysis for blood, protein and glucose
- Blood urea, electrolytes and creatinine
- Blood glucose level
- Serum total cholesterol and high-density lipoprotein (HDL) cholesterol, LDL cholesterol, triglycerides
- 12-lead ECG (left ventricular hypertrophy, coronary artery disease)
- ***Thyroid function test:*** T_3, T_4, TSH to rule out hypothyroidism and hyperthyroidism
- ***Chest X-ray:*** To detect cardiomegaly, heart failure, coarctation of the aorta
- ***Ambulatory BP recording:*** To assess borderline or 'white coat' hypertension
- ***Echocardiogram:*** To detect or quantify left ventricular hypertrophy
- ***Renal ultrasound:*** To detect possible renal disease
- ***Renal angiography:*** To detect or confirm presence of renal artery stenosis
- ***Urinary catecholamines:*** To detect possible pheochromocytoma
- ***Urinary cortisol and dexamethasone suppression test:*** To detect possible Cushing's syndrome
- ***Plasma renin activity and aldosterone:*** To detect possible primary aldosteronism.

MANAGEMENT OF HYPERTENSION

- ***Lifestyle modifications:*** It includes weight reduction, DASH (Dietary Approaches to Stop Hypertension) eating plan, and dietary sodium reduction, moderation of alcohol consumption, regular aerobic physical activity, and avoidance of tobacco use (smoking and chewing).
- ***Weight reduction:*** Weight reduction has a significant effect on lowering BP in many people, and the effect is seen with even moderate weight loss. Weight reduction through a combination of dietary calorie restriction and moderate physical activity is recommended for overweight hypertensive patients.
- ***DASH eating plan:*** This diet involves eating several servings of fish each week, eating plenty of fruits and vegetables, increasing fiber intake, and drinking a lot of water. The DASH diet significantly lowers BP. These decreases have been compared to those achieved with BP-lowering medication.
- ***Dietary sodium reduction:*** It has been recommended that healthy adults restrict salt intake to less than 6 g of salt (NaCl) or less than 2.4 g of sodium per day. This involves avoiding foods known to be high in sodium and not adding salt in the preparation of foods or at meals.

  The patient and family, especially the member who prepares the meals, should be taught about sodium-restricted diets. Instructions should include reading labels of over-the-counter drugs, packaged foods, and health products (e.g. toothpaste containing baking soda) to identify hidden sources of sodium. It is helpful to review the patient's normal

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diet and to identify foods high in sodium. Analysis of a 3-day diet history will help identify foods high in sodium in the patient's usual diet. Sodium restriction may be enough to control BP in some patients with hypertension.

- ***Moderation of alcohol consumption:*** Excessive alcohol consumption is strongly associated with hypertension. Consumption of three or more alcoholic drinks daily is also a risk factor for heart disease and stroke. Men should limit their intake of alcohol to no more than 2 drinks per day and women and lighter weight men to no more than 1 drink per day. 1 drink = 1.5 oz alcohol. Excessive alcohol consumption that results in cirrhosis is the most frequent cause of secondary hypertension.
- ***Physical activity:*** It is recommended that all adults have regular aerobic physical activity (e.g. brisk walking) at least 30 minutes per day atleast 5 days in a week. Moderately intense activity such as brisk walking, jogging, and swimming can lower BP, promotes relaxation, and decrease or control body weight. Regular activity of this type can reduce SBP in the hypertensive patient by approximately 4 to 9 mm Hg.
- ***Avoidance of tobacco products:*** Nicotine in tobacco causes vasoconstriction and increases BP in hypertensive people. The cardiovascular benefits of discontinuing tobacco use can be seen within 1 year in all age-groups. Everyone, especially a hypertensive patient, should be strongly advised to avoid tobacco use.
- ***Stress management:*** Stress can raise BP on a short-term basis and has been implicated in the development of hypertension. Relaxation therapy, guided imagery, and biofeedback may be useful in helping patients manage stress, thus decreasing BP.

Drug therapy (for further details refer Chapter 3)

- ***Diuretics***
  - Thiazide diuretics
  - Loop diuretics
  - Potassium-sparing diuretics
  - Aldosterone receptor blockers
- ***Adrenergic inhibitors***
  - Central-acting α-adrenergic antagonists
  - Peripheral-acting α-adrenergic antagonists
  - α_1-adrenergic blockers
  - β-adrenergic blockers.
- ***Direct vasodilators***
- ***Angiotensin inhibitors***
  - Angiotensin-converting enzyme inhibitors
  - Angiotensin-II receptor blockers
- ***Calcium channel blockers***

Nursing Management

Encouraging the Patient to do Lifestyle Modifications

It includes weight reduction, DASH (dietary approaches to stop hypertension) eating plan, and dietary sodium reduction, moderation of alcohol consumption, regular aerobic physical activity, and avoidance of tobacco use (smoking and chewing).

Providing Basic Information on Disease Process

- Explain the meaning of high BP, risk factors, and their influences on the cardiovascular, cerebral, and renal systems.
- Stress that there cannot be total cure, only control, of hypertension can be done; emphasize the consequences of uncontrolled hypertension.
- Emphasize the fact that there may be no correlation between high BP and symptoms; the patient cannot tell by the way he feels whether BP is normal or elevated.
- Have the patient recognize that hypertension is chronic and requires persistent therapy and periodic evaluation. Effective treatment improves life expectancy; therefore, follow-up health care visits are mandatory.
- Inform the patient regarding various diagnostic and therapeutic activities to minimize anxiety and to obtain cooperation.
- Solicit the assistance of the patient's spouse, family, and friends provide information regarding the total treatment plan.

Education on Pharmacological Management of Hypertension

- Explain that the drugs used for effective control of elevated BP will likely produce adverse effects.
- Alert the patient regarding orthostatic hypotension that may occur initially with some drug therapy.
- Instruct the patient to get up slowly to offset the feeling of dizziness.
- Encourage the patient to sit or lie down immediately if he feels faint.

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- Alert the patient to expect initial effects, such as anorexia, light-headedness, and fatigue, with many medications.
- Inform the patient that the goal of treatment is to control BP, reduce the possibility of complications, and using minimum number of drugs with the lowest dosage necessary to accomplish the goal.
- Educate the patient to be aware of serious adverse effects and report them immediately so that adjustments can be made in individual pharmacotherapy.

Improving Patient Adherence to the Treatment

A major problem in the long-term management of the patient with hypertension is poor adherence with the treatment plan. It is important to determine the reasons why a patient is not adhering to treatment. The reasons for nonadherence are complex and can include inadequate patient teaching, low health literacy, unpleasant side effects of drugs, return of BP to normal range while on drugs, high cost of drugs, and lack of insurance. Develop a plan with the patient and caregiver to improve adherence. The plan should be compatible with the patient's habits, cultural beliefs, and lifestyle.

Controlling of Blood Pressure

- Encourage the patient to consult a dietitian to help develop a plan for improving nutrient intake or for weight loss.
- Encourage restriction of sodium and fat.
- Emphasize increase intake of fruits and vegetables.
- Implement regular physical activity.
- Advise patient to limit alcohol consumption and avoidance of tobacco.
- Assist the patient to develop and adhere to an appropriate exercise regimen.

Prevention and Treatment of Complications

Symptoms suggesting that hypertension is progressing to the extent that target organ damage is occurring must be detected early so that appropriate treatment can be initiated. When the patient returns for follow-up care, all body systems must be assessed to detect any evidence of vascular damage. An eye examination with an ophthalmoscope is particularly important because retinal blood vessel damage indicates similar damage elsewhere in the vascular system. The heart, nervous system, and kidneys are also carefully assessed. Any significant findings are promptly reported to determine whether additional diagnostic studies are required. Based on the findings, medications may be changed to improve blood pressure control.

Encouraging Self-management

- Enlist the patient's cooperation in redirecting lifestyle in keeping with the guidelines of therapy, acknowledge the difficulty, and provide support and encouragement.
- Plan the patient's medication schedule so that the many medications are given at proper and convenient times; set up a daily checklist on which the patient can record the medication taken.
- Be sure the patient knows the generic and brand names for all medications and throws away old medications and dosages so they will not be mixed up with current medications.
- Instruct the patient regarding proper method of taking BP at home and at work. Inform patient of desired range and the readings that are to be reported.
- Determine recommended dietary plans and provide dietary education as appropriate.
- Most patients have a basic knowledge of nutrients and minerals, such as fats and sodium, and can be taught to read labels.
- Many patients may require consultation with a dietitian to understand how diet may affect BP and health.

Discharge and Home Care Guidelines

- The nurse can help the patient to achieve blood pressure control through education about managing blood pressure.
- Assist the patient in setting blood pressure goals.
- Provide assistance with social support.
- Encourage the involvement of family members in the education program to support the patient's efforts to control hypertension.
- Provide written information about expected effects and side effects.
- Encourage and teach patients to measure their blood pressures at home.
- Emphasize strict compliance of follow-up care.

HYPERTENSIVE CRISIS

Hypertensive crisis is defined as systolic blood pressure more than 180 mm Hg and or diastolic blood pressure more than 120 mm Hg. Hypertensive crisis is categorized as "hypertensive urgency" if the blood pressure is high without damage to organs, and as "hypertensive emergency" if the blood pressure is high with damage to organs.

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Causes

- Exacerbation of chronic hypertension
- Renovascular hypertension
- Preeclampsia, eclampsia
- Pheochromocytoma
- Drugs such as oral contraceptive pills, over-the-counter painkillers, and steroids, as well as cocaine or methamphetamines
- Rebound hypertension (from abrupt withdrawal of some antihypertensive drugs such as clonidine and propranolol
- Head injury
- Salt rich diet
- Acute aortic dissection.

Clinical Manifestations

Neurological symptoms

Hypertensive encephalopathy: A syndrome in which a sudden rise in BP is associated with severe headache, nausea, vomiting, seizures, confusion, and coma. The manifestations of encephalopathy are the result of increased cerebral capillary permeability. This leads to cerebral edema and a disruption in cerebral function.

Renal symptoms

Renal insufficiency ranging from minor injury to complete renal failure can occur.

Cardiovascular symptoms

Rapid cardiac decompensation ranging from unstable angina to MI and pulmonary edema is also possible. Patients can have chest pain and dyspnea. Aortic dissection can develop and will cause sudden, excruciating chest and back pain and possibly reduced or absent pulses in the extremities.

Treatment

Hypertensive Emergency

For hypertensive emergency the therapeutic goals are reduction of the mean arterial pressure by 20 to 25% or to decrease mean arterial pressure to 110 to 115 mm Hg within the first hour of treatment, a further reduction to a goal pressure of about 160/100 mm Hg over a period of up to 6 hours, and then a more gradual reduction in pressure over a period of days. Lowering the BP too quickly may decrease cerebral, coronary, or renal perfusion. This could cause a stroke, MI, or renal failure. The medications of choice in hypertensive emergencies are those that have an immediate effect. Intravenous vasodilators, including sodium nitroprusside, nicardipine, clevidipine, labetalol and nitroglycerin have immediate actions that are short-lived and therefore frequently used for initial treatment.

Hypertensive Urgency

For hypertensive urgency oral agents can be given with the goal of normalizing blood pressure within 24 to 48 hours. Oral doses of fast-acting agents such as beta-adrenergic blockers (labetalol) ACE inhibitors (captopril), or alpha2-agonists (clonidine) are recommended for the treatment of hypertensive urgencies.

Nursing Management

- Assess the patient's BP and HR every 2 to 3 minutes during the initial administration of drugs.
- Use an arterial line or an automated, noninvasive BP machine to monitor the BP.
- Monitor the ECG for dysrhythmias and signs of ischemia.
- Measure urine output hourly to assess renal perfusion.
- Patients receiving IV antihypertensive drugs may be restricted to bed. Getting up (e.g., to use the commode) may cause severe cerebral ischemia and fainting.
- Ongoing assessment is essential to evaluate the effectiveness of these drugs and the patient's response to therapy. Frequent neurologic checks, including level of consciousness, pupillary size and reaction, and movement of extremities, help detect any changes in the patient's condition.
- Monitor cardiac, pulmonary, and renal systems for decompensation caused by the severe elevation in BP (e.g., angina, pulmonary edema, renal failure).

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CASE STUDY: TREATING HYPERTENSION IN PATIENTS WITH DIABETES

PRESENTATION

L.N. is a 49-year-old woman with a history of type 2 diabetes, obesity, hypertension, and migraine headaches. The patient was diagnosed with type 2 diabetes 9 years ago when she presented with mild polyuria and polydipsia. L.N. is 5′4″ and has always been on the large side, with her weight fluctuating between 165 and 185 lb.

Initial treatment for her diabetes consisted of an oral sulfonylurea with the rapid addition of metformin. Her diabetes has been under fair control with a most recent hemoglobin A1c of 7.4%.

Hypertension was diagnosed 5 years ago when blood pressure (BP) measured in the office was noted to be consistently elevated in the range of 160/90 mm Hg on three occasions. L.N. was initially treated with lisinopril, starting at 10 mg daily and increasing to 20 mg daily, yet her BP control has fluctuated.

One year ago, microalbuminuria was detected on an annual urine screen, with 1,943 mg/dL of microalbumin identified on a spot urine sample. L.N. comes into the office today for her usual follow-up visit for diabetes. Physical examination reveals an obese woman with a BP of 154/86 mm Hg and a pulse of 78 bpm.

QUESTIONS

1. What are the effects of controlling BP in people with diabetes?
2. What is the target BP for patients with diabetes and hypertension?
3. Which antihypertensive agents are recommended for patients with diabetes?

COMMENTARY

Diabetes mellitus is a major risk factor for cardiovascular disease (CVD). Approximately two-thirds of people with diabetes die from complications of CVD. Nearly half of middle-aged people with diabetes have evidence of coronary artery disease (CAD), compared with only one-fourth of people without diabetes in similar populations.

Patients with diabetes are prone to a number of cardiovascular risk factors beyond hyperglycemia. These risk factors, including hypertension, dyslipidemia, and a sedentary lifestyle, are particularly prevalent among patients with diabetes. To reduce the mortality and morbidity from CVD among patients with diabetes, aggressive treatment of glycemic control as well as other cardiovascular risk factors must be initiated.

Studies that have compared antihypertensive treatment in patients with diabetes versus placebo have shown reduced cardiovascular events. The United Kingdom Prospective Diabetes Study (UKPDS), which followed patients with diabetes for an average of 8.5 years, found that patients with tight BP control (< 150/< 85 mm Hg) versus less tight control (< 180/< 105 mm Hg) had lower rates of myocardial infarction (MI), stroke, and peripheral vascular events. In the UKPDS, each 10 mm Hg decrease in mean systolic BP was associated with a 12% reduction in risk for any complication related to diabetes, a 15% reduction for death related to diabetes, and an 11% reduction for MI. Another trial followed patients for 2 years and compared calcium-channel blockers and angiotensin-converting enzyme (ACE) inhibitors, with or without hydrochlorothiazide against placebo and found a significant reduction in acute MI, congestive heart failure, and sudden cardiac death in the intervention group compared to placebo.

The Hypertension Optimal Treatment (HOT) trial has shown that patients assigned to lower BP targets have improved outcomes. In the HOT trial, patients who achieved a diastolic BP of <80 mm Hg benefited the most in terms of reduction of cardiovascular events. Other epidemiological studies have shown that BPs > 120/70 mm Hg are associated with increased cardiovascular morbidity and mortality in people with diabetes. The American Diabetes Association has recommended a target BP goal of < 130/80 mm Hg. Studies have shown that there is no lower threshold value for BP and that the risk of morbidity and mortality will continue to decrease well into the normal range.

Many classes of drugs have been used in numerous trials to treat patients with hypertension. All classes of drugs have been shown to be superior to placebo in terms of reducing morbidity and mortality. Often, numerous agents (three or more) are needed to achieve specific target levels of BP. Use of almost any drug therapy to reduce hypertension in patients with diabetes has been shown to be effective in decreasing cardiovascular risk. Keeping in mind that numerous agents are often required to achieve the target level of BP control, recommending specific agents becomes a not-so-simple task. The literature continues to evolve, and individual patient conditions and preferences also must come into play.

While lowering BP by any means will help to reduce cardiovascular morbidity, there is evidence that may help guide the selection of an antihypertensive regimen. The UKPDS showed no significant differences in outcomes for treatment for hypertension using an ACE inhibitor or a β-blocker. In addition, both ACE inhibitors and angiotensin II receptor blockers (ARBs) have been shown to slow the development and progression of diabetic nephropathy. In the Heart Outcomes Prevention

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Evaluation (HOPE) trial, ACE inhibitors were found to have a favorable effect in reducing cardiovascular morbidity and mortality, whereas recent trials have shown a renal protective benefit from both ACE inhibitors and ARBs. ACE inhibitors and β-blockers seem to be better than dihydropyridine calcium-channel blockers to reduce MI and heart failure. However, trials using dihydropyridine calcium-channel blockers in combination with ACE inhibitors and β-blockers do not appear to show any increased morbidity or mortality in CVD, as has been implicated in the past for dihydropyridine calcium-channel blockers alone. Recently, the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT) in high-risk hypertensive patients, including those with diabetes, demonstrated that chlorthalidone, a thiazide-type diuretic, was superior to an ACE inhibitor, lisinopril, in preventing one or more forms of CVD.

L.N. is a typical patient with obesity, diabetes, and hypertension. Her BP control can be improved. To achieve the target BP goal of < 130/80 mm Hg, it may be necessary to maximize the dose of the ACE inhibitor and to add a second and perhaps even a third agent.

Diuretics have been shown to have synergistic effects with ACE inhibitors, and one could be added. Because L.N. has migraine headaches as well as diabetic nephropathy, it may be necessary to individualize her treatment. Adding a β-blocker to the ACE inhibitor will certainly help lower her BP and is associated with good evidence to reduce cardiovascular morbidity. The β-blocker may also help to reduce the burden caused by her migraine headaches. Because of the presence of microalbuminuria, the combination of ARBs and ACE inhibitors could also be considered to help reduce BP as well as retard the progression of diabetic nephropathy. Overall, more aggressive treatment to control L.N.'s hypertension will be necessary. Information obtained from recent trials and emerging new pharmacological agents now make it easier to achieve BP control targets.

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