Journal of Molecular Pathophysiology, 2022
VOL 11, NO. 9, PAGES 01-02

OPINION ARTICLE

Open Access

Pathophysiology of Hypertension and Its Cause

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ARTICLE HISTORY
Received: 12-Aug-2022, Manuscript No. JMOLPAT-22-74875;
Editor assigned: 15-Aug-2022, PreQC No: JMOLPAT-22-74875 (PQ);
Reviewed: 01-Sep-2022, QC No: JMOLPAT-22-74875;
Revised: 08-Sep-2022, Manuscript No: JMOLPAT-22-74875 (R).
Published: 16-Sep-2022

Description

Pathophysiology

A subspecialty of medicine called pathophysiology discusses how the body works in relation to illnesses and ailments. The study of the pathophysiology of hypertension aims to provide a mechanistic explanation for the disease's causes. Hypertension is a chronic condition marked by elevated blood pressure. The two main types of hypertension are essential (sometimes called primary or idiopathic) and secondary. Essential hypertension makes up about 90–95 percent of hypertension. Some experts characterize essential hypertension as having no known cause, while others say it is brought on by eating too much salt and not enough potassium. The term "secondary hypertension" refers to hypertension that is brought on by an underlying condition with a known cause, such as chronic kidney disease, aortic or kidney artery stenosis, or endocrine problems including elevated levels of aldosterone, cortisol, or catecholamine [1]. A key risk factor for hypertensive heart disease, coronary artery disease, and stroke, aneurysm of the aorta, peripheral artery disease, and chronic kidney disease is persistent hypertension. The two factors that determine arterial pressure are cardiac output and peripheral resistance. Heart rate and stroke volume together determine cardiac output; stroke volume is influenced by the size of the vascular compartment and myocardial contractility. Functional and anatomical alterations in tiny arteries and arterioles impact peripheral resistance [2,3].

Genetics

Mendelian variants of high blood pressure can result from single gene mutations; twelve genes have been found to be responsible for these monogenic forms of hypertension. Through modifications to kidney salt processing, these mutations affect blood pressure. Blood pressure within families is more similar than between families, indicating a type of inheritance, and this is not because of shared environmental factors. A statistically significant relationship between blood pressure and many chromosomal areas, including those connected to familial combination hyperlipidemia, was discovered using genetic analytic tools. These results imply that there are several genetic loci with modest effects on blood pressure in the overall population. However, the overall rarity of recognizable single-gene causes of hypertension is consistent with a complex origin for essential hypertension [4,5].

Autonomic nervous system

Pressure, volume and chemoreceptor inputs from the autonomic nervous system are crucial for maintaining cardiovascular homeostasis. It accomplishes this via controlling renal function, peripheral vasculature, and cardiac output, which in turn affect vascular resistance, fluid retention, and fluid retention. Hypertension is a result of sympathetic nervous system over activity, which raises blood pressure [6,7].

Modifications in bar reflex and chemo reflex pathways at both the peripheral and central levels have a role in the causes of increased sympathetic nervous system activity in hypertension. When arterial pressure is stabilized, the peripheral resetting of arterial baroreceptors which occurs in hypertensive individuals, returns to normal. In addition, the aortic bar reflex is centrally reset in hypertension individuals, which suppresses sympathetic inhibition after aortic baroreceptor nerve activity. Angiotensin II's central action appears to be mediated, at least in part, by this bar reflex resetting. Reactive oxygen species and endothelin are additional small-molecule mediators that inhibit baroreceptor action and support an accentuated sympathetic drive in hypertension. According to several researches, hypertension patients react to norepinephrine injections more vasoconstrictive than normotensive controls. Furthermore, hypertension patients do not typically respond to elevated levels of circulating norepinephrine by downregulating

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the noradrenergic receptor; it is thought that this aberrant response is inherited genetically [8]. Stress exposure increases sympathetic output, and persistent stress-induced vasoconstriction may lead to vascular hypertrophy, which causes progressively higher blood pressure and peripheral resistance. Since they experience higher amounts of stress from daily life, this may help to explain why hypertension is more common in lower socioeconomic groups. When exposed to laboratory stressors like cold pressor testing and mental stress, which may predispose a person to hypertension, they exhibit heightened vasoconstrictor and sympathetic responses. Particularly for young African Americans, this is true. Exaggerated stress reactions could be a factor in this group's higher incidence of hypertension. By electrically activating the baroreflex using a pacemaker-like device, resistant hypertension can be treated.

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