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Reconsidering Neurogenic Essential Hypertension

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Description

The use of diuretics, angiotensin-converting enzyme inhibitors, and angiotensin receptor blockers to lower blood pressure (BP) through effects on blood volume and the renin-angiotensin system has become increasingly popular in the treatment of essential hypertension. However, in many people, these drugs, whether used alone or in combination, fail to bring blood pressure back to normal. In such instances, hypertension is likely to be maintained in part by pathophysiologic processes other than volume and the renin-angiotensin system, necessitating medication targeting other mechanisms.

Neurogenic hypertension is one such type of hypertension that is frequently neglected. The goal of this paper is to bring this underappreciated phenomenon back into the spotlight, to provide a clinically focused summary of its putative causes and manifestations, and to highlight the potentially significant treatment implications of recognising this type of hypertension. These findings highlight the importance of continuing to focus clinical and scientific emphasis on neurogenically caused hypertension [1].

Despite breakthroughs in treatment, only 45 percent of treated hypertensives have their essential hypertension under control. Because of the, hypertension can respond beautifully variety of underlying hypertension mechanisms, most single medications only decrease blood pressure (BP) in 50% to 60% of patients. Not unexpectedly to one medication while being entirely unresponsive to another in the same person. A logical goal would be to treat people with drugs that target their hypertension's underlying mechanism. This is, unfortunately, a difficult task. The Sixth Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VI) drug selection guidelines reflect this, focusing on demographics, co-morbidities, and side effects rather than underlying hypertensive mechanisms. Even in individuals with severe essential hypertension, we often don't know the mechanism or the appropriate treatment option [2].

The central role of blood volume and the Renin-angiotensin System (RAS) in the development of essential hypertension has been highlighted in recent years, and drugs targeting these systems, such as diuretics, angiotensin-converting enzyme inhibitors (ACEIs), and Angiotensin Receptor Blockers (ARBs), have played an increasingly prominent role in treatment. However, despite the fact that these drugs are highly successful when used alone or in combination, they fail to regulate blood pressure in a large number of individuals. This limitation emphasises the necessity for a wider range of therapeutic options. The link between essential hypertension and enhanced Sympathoadrenal System (SAS) activity, as well as the antihypertensive efficacy of SAS-targeted drugs, suggests that essential hypertension may potentially have a neurogenic component. Despite a large body of basic research on neurogenic BP regulation pathways, the clinical significance of neurogenically driven hypertension has received little attention. The goal of this review is to draw attention to the clinical importance of neurogenic hypertension, to illustrate clinical scenarios in which it can be suspected, and to highlight the potentially significant treatment implications of its diagnosis [3].

Clinical features of non-neurogenic essential hypertension

The regulation of volume and RAS expression are well known to be related to human hypertension, and antihypertensive medications aiming at them are commonly employed. These types of hypertension appear to be caused by both hereditary and lifestyle factors. Hypertension usually develops gradually, with lifestyle factors such as nutrition, weight, and exercise status influencing the onset age and severity. Salt sensitivity of blood pressure, lower levels of plasma renin activity, and response to salt restriction, diuretics, and calcium channel blockers are all characteristics of volume-dependent. It is most common among African Americans, the elderly, and those with renal failure. RAS-dependent essential hypertension, on the other hand, is less susceptible to diuretics and more responsive to ACEIs and ARBs [4].

The majority of patients react to either a diuretic or an ACEI (or ARB) alone, or a combination of the two. Noncompliance with medicine, alcohol misuse, and intake of sympathomimetic drugs may all be factors in certain nonresponders. Isolated systolic hypertension, which has a different pathophysiology than essential hypertension, can be difficult to manage. In the absence of such causes, hypertension that does not respond to a diuretic/ACEI combination is thought to be secondary hypertension, but diagnostic examination is frequently inconclusive. Pathogenetic pathways other than volume and the RAS, such as neurogenic hypertension, should certainly be considered in this context [5].

Neurogenic hypertension

The SAS is heavily engaged in the regulation of blood pressure. Afferent nerves from artery baroreceptors, central nervous system routes such as the brainstem, medulla, hypothalamus, limbic system, and spinal cord, and peripheral sympathetic nerves are among its components. The nucleus tractus solitarius and the caudal ventrolateral medulla both have inhibitory effects on sympathetic outflow, but the rostral ventrolateral medulla has excitatory effects. Sympathetic outflow is known to be affected by excitatory and inhibitory inputs from other central nervous system areas, such as the hypothalamus and limbic cortex. Unfortunately, the brain mechanisms regulating blood pressure in normotensive and hypertensive people are complex and poorly understood [6].

Transient elevations in blood pressure, such as those that occur in reaction to stressors, are obviously mediated by the SAS. Although research examining plasma catecholamines, norepinephrine spillover, microneurography, and heart rate variability have confirmed that SAS tone is stronger in hypertensive than in normotensive populations, its involvement in persistent hypertension is less well understood. Furthermore, medicines that inhibit SAS activity or counteract its effects significantly lower blood pressure.

In human essential hypertension, the variables that increase sympathetic tone are poorly known. Although the importance of this action in human hypertension is unknown, angiotensin II promotes sympathetic outflow. Some researchers believe insulin resistance is involved, but it's unclear whether insulin resistance is a cause or a result of higher SAS tone. Another possible cause, impaired baroreflexes, is refuted by evidence. Increasing sympathetic tone may be caused by salt sensitivity and increased salt intake. Genetic variables are also being looked into. Janetta et al. first proposed a link between neurogenic hypertension and vascular compression of the medulla by ecstatic vessels, but later research has produced mixed results [7].

Clinical signs and symptoms of neurogenic hypertension

When the SAS is the primary cause of hypertension, it is referred to as

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neurogenic hypertension. The challenge is determining which individuals are likely to be affected by neurogenic mechanisms. The lack of therapeutically useful tools for evaluating SAS tone in individual individuals is a major impediment to such research. Catecholamine levels in the blood and urine do not accurately reflect differences in SAS tone between people. Sympathetic nerve activity measurements is time-consuming and uncomfortable, thus it's only used in study. The function of spectral heart rate analysis has yet to be determined. Clinical rather than biochemical indicators may merit more attention in this context as indicators of possible neurogenic hypertension [8].

Neurogenic hypertension and psychological aspects

The well-established link between emotional stressors and changes in blood pressure and SAS tone lends credence to the idea that neurogenic hypertension is linked to psychological variables. Anxiety is linked to increased SAS tone, and neurogenic hypertension is frequently suspected in anxious and tachycardia people who do have an adrenergically mediated hyperkinetic form of blood pressure elevation. Anxiety and tachycardia, on the other hand, are frequently a symptom of white coat hypertension (blood pressure rise limited to the doctor's office), rather than actual sustained hypertension as measured by ambulatory or home blood pressure monitoring. As a result, while there is a correlation between anxiety and hypertension, it is weaker than widely assumed [9].

Other psychological factors may play a role in hypertension, particularly neurogenic hypertension. Emotional defensiveness has been linked to hypertension, particularly severe hypertension. Recurrent paroxysmal hypertension, also known as pseudopheochromocytoma, appears to be neurogenic and psychosomatic in origin, and has been successfully treated as a result of this knowledge. The transience of the hypertensive episodes, elevations in catecholamine levels during episodes, and the efficacy of combination plus blocking in some patients all point to a neurogenic aetiology. Although patients universally state that episodes are unconnected to stress or mental anguish, there is an underlying psychosomatic aetiology. A pattern of repression of emotions is a common thread across patients, which can be linked to a history of severe abuse or trauma, or to a more widespread habit of repressing undesired emotions. Psychotherapy has been shown to be extremely effective in preventing recurring hypertension paroxysms, and antidepressants have been known to be extremely effective in preventing recurrent hypertensive paroxysms [10].

Conclusion

Essential hypertension is a diverse condition with better treatment outcomes when medications are chosen based on the pathophysiology of the particular patient. Greater understanding of the SAS's role, as well as the anticipated role of neurogenic pathways in some hypertension individuals, would undoubtedly aid in achieving this goal.

Although it is impossible to properly quantify SAS tone in individual individuals, the potential that widely available clinical signals could serve as indications of neurogenic hypertension is worth investigating. If we are to move from "cookbook" treatment to more physiologically appropriate individualised care of hypertensive patients, more research and clinical trials designed to identify individuals with neurogenically mediated hypertension and assess their responses to different antihypertensive regimens are needed.

Conflict Of Interest

None.

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