

Endocrine Hypertension: Hormonal Imbalances and Cardiovascular Consequences

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DESCRIPTION

Hypertension, or high blood pressure, is a widespread health concern affecting millions worldwide. While lifestyle factors like diet and exercise play significant roles, a subset of hypertension, known as endocrine hypertension, stems from hormonal imbalances within the endocrine system. This comprehensive exploration delves into the intricate relationship between hormones and hypertension, and enlighten its causes, effects, and management strategies [1].

Endocrine hypertension

Endocrine hypertension refers to high blood pressure resulting from hormonal abnormalities, primarily involving the adrenal glands, thyroid gland, and Renin Angiotensin Aldosterone System (RAAS). These hormonal imbalances disrupt the body's regulatory mechanisms, leading to elevated blood pressure levels [2]. One of the most common causes of endocrine hypertension is primary aldosteronism, characterized by excess aldosterone production from the adrenal glands. Aldosterone promotes sodium retention and potassium excretion, leading to fluid retention and increased blood volume, ultimately raising blood pressure. Other endocrine disorders contributing to hypertension include Cushing's syndrome, characterized by excessive cortisol production; pheochromocytoma, involving adrenaline and noradrenaline-secreting tumors; and thyroid disorders such as hyperthyroidism [3].

Effects of endocrine hypertension

The consequences of endocrine hypertension extend beyond elevated blood pressure levels, impacting various organ systems and increasing the risk of cardiovascular complications. Chronic hypertension can lead to heart disease, stroke, kidney damage, and vision loss if left untreated [4]. The excess hormone secretion associated with endocrine disorders can exacerbate

these risks. For instance, aldosterone induced sodium retention can lead to fluid overload, exacerbating heart failure and contributing to cardiovascular trials. Moreover, hormonal imbalances may manifest as additional symptoms, including weight gain, fatigue, muscle weakness, palpitations, and mood disturbances, further compromising patients' quality of life [5].

Diagnosis of endocrine hypertension

Diagnosing endocrine hypertension requires a comprehensive evaluation of hormonal levels and underlying conditions. Initial assessments typically involve measuring blood pressure and screening for secondary causes of hypertension, such as renal artery stenosis and obstructive sleep apnea [6]. Laboratory tests, including serum electrolytes, plasma renin activity, aldosterone-to-renin ratio, and cortisol levels, help identify hormonal imbalances. Imaging studies such as CT scans, MRI, and nuclear medicine scans may be employed to localize adrenal or thyroid tumors. Confirmatory tests, such as the saline suppression test for aldosterone excess or the clonidine suppression test for catecholamine-secreting tumors, may be performed to establish the diagnosis definitively [7].

Management of endocrine hypertension

The management of endocrine hypertension revolves around addressing the underlying hormonal abnormalities while controlling blood pressure to reduce the risk of cardiovascular complications. Treatment strategies may vary depending on the specific endocrine disorder and patient's overall health status [8]. For primary aldosteronism, mineralocorticoid receptor antagonists like spironolactone or eplerenone are often prescribed to block the effects of aldosterone, leading to sodium excretion and blood pressure reduction. Surgical intervention, such as adrenalectomy, may be considered for unilateral adrenal adenomas causing aldosterone excess [9].

In cases of Cushing's syndrome, treatment aims to reduce

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cortisol production through surgery, radiation therapy, or medication such as ketoconazole or metyrapone. Pheochromocytomas are typically managed with surgical resection of the adrenal tumor, followed by alpha and beta-adrenergic blockade to control catecholamine release [10]. Thyroid disorders contributing to hypertension, such as hyperthyroidism, are managed with antithyroid medications, radioactive iodine therapy, or thyroidectomy, depending on the underlying etiology. Additionally, lifestyle modifications including dietary changes, weight management, regular exercise, and stress reduction techniques are crucial components of hypertension management in all patients, including those with endocrine-related hypertension.

CONCLUSION

Endocrine hypertension represents a complex interplay between hormonal dysregulation and cardiovascular health, necessitating a multifaceted approach to diagnosis and management. By unraveling the underlying hormonal imbalances and implementing targeted therapeutic strategies, healthcare providers can effectively control blood pressure and mitigate the risk of associated complications in patients with endocrine-related hypertension.

REFERENCES

1. Wang T, Ma S, Guan Y, Du J, Liu G, Zhao X. Double function of noninvasive intracranial pressure monitoring based on flash visual evoked potentials in unconscious patients with traumatic brain injury. *J Clin Neurosci*. 2016;27(S):63-67.
2. Berezcki D, Liu M, Prado GF, Fekete I. Cochrane report: A systematic review of mannitol therapy for acute ischemic stroke and cerebral parenchymal hemorrhage. *Stroke*. 2000;31(11):2719-2722.
3. Hemphill III JC, Greenberg SM, Anderson CS, Becker K, Bendok BR, Cushman M, et al. Guidelines for the management of spontaneous intracerebral hemorrhage: A guideline for healthcare professionals from the American heart association/American stroke association. *Stroke*. 2015;46(S):2032-2060.
4. Kothari RU, Brott T, Broderick JP, Barsan WG, Sauerbeck LR, Zuccarello M, et al. The abs of measuring intracerebral hemorrhage volumes. *Stroke*. 1996;27(8):1304-1305.
5. Gross T, Morell S, Amsler F. Gender-specific improvements in outcome 1 and 2 years after major trauma. *J Surg Res*. 2019;235(S):459-469.
6. Tao Z, Gao L. Management of patients with severe traumatic brain injury guided by intraventricular intracranial pressure monitoring: A report of 136 cases. *Chin J Traumatol*. 2010;13(3): 146-151.
7. Zhao YL, Zhou JY, Zhu GH. Clinical experience with the noninvasive Icp monitoring system. *Acta Neurochir Suppl*. 2005;95(S):351-355.
8. Simona S, Marini C, Toni D, Olivieri L. Incidence and 10-year survival of intracerebral hemorrhage in a population-based registry. *Stroke*. 2009;40(2):394-399.
9. Davis SM, Broderick J, Hennerici M, Brun NC. Hematoma growth is a determinant of mortality and poor outcome after intracerebral hemorrhage. *Neurology*. 2006;66(8):1175-1181.
10. Qerama E, Korshoej AR, Petersen MV, Brandmeier R, von Oettingen G. Latency-shift of intra-operative visual evoked potential predicts reversible homonymous hemianopia after intra-ventricular meningioma surgery. *Clin Neurophysiol Pract*. 2019;4(S):224-229.