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Role of Aldosterone in Hypertension Patients

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Introduction

It has been recognized for several decades (since the initial report by Conn in 1955) that primary hyperaldosteronism is a crucial explanation for high blood pressure, being typically characterized by high (or occasionally normal) aldosterone levels, suppressed renin levels, and an elevated ARR. The underlying causes of primary hyperaldosteronism include bilateral adrenal hyperplasia (in about 60% of cases), an aldosterone secreting adrenal adenoma (a cause amenable to surgical treatment), and rarer forms like glucocorticoid remediable aldosteronism (GRA) and familial hyperaldosteronism Type I.

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A discussion of the latter forms is beyond the scope of this review; these entities are reviewed intimately in references. It's critical to think about the likelihood of hyper aldosteronism during a patient with a high pre-test probability of high blood pressure because hypertensive patients with hyperaldosteronism have worse cardiovascular morbidity relative to those with hyperpiesia, with rates of stroke and non-fatal myocardial infarct being 4–6 fold higher within the former compared to the latter; the previous could also be curable once in a while surgically (when thanks to an adenoma), thereby avoiding lifelong antihypertensive treatment; and it's going to influence the selection of antihypertensive treatment because patients with hyper aldosteronism respond better to MR blockers like spironolactone.

Whereas there's little dispute that hyper aldosteronism is a crucial etiologic factor to be considered in patients with a high likelihood of high blood pressure, the prevalence estimates among hypertensive patients has varied considerably within the literature. Formerly it had been widely believed that primary hyper aldosteronism was quite infrequent among patients with hypertension, constituting but 2% of all cases (with a variety of 0.5–2%). Recent estimates are considerably higher, partially due to the popularity that hypokalemia isn't always a concomitant of hyper aldosteronism, and also greater awareness and use of the ARR for the diagnosis of the condition. newer estimates suggest that the prevalence of primary hyper aldosteronism may vary supported the sampling frame of hypertensive patients and their severity of hypertension, and also the standards used for the diagnosis of the condition. Thus, a recent meta-analysis estimated that the prevalence of hyper aldosteronism is 4.3% among medical care patients with hypertension (8 studies), but higher among referred patients with hypertension in clinical centers (estimated at 9%, supported 12 studies), and even higher among patients with refractory hypertension (in whom prevalence could also be as high as 15–20%).

As noted above, the prevalence of primary hyperaldosteronism also varies supported the precise biochemical criteria wont to diagnose the condition. A high ARR has been widely wont to detect identifiable secondary causes of hypertension, thanks to the very fact that primary hyperaldosteronism has been known to be one among its commonest causes. Yet, sole reliance on the ARR typically overestimates the prevalence of hyperaldosteronism due to various factors which will influence the ratio and impact its sensitivity and specificity for the diagnosis of the condition. Using data from the Study of Health in Pomerania (SHIP) and therefore the Cooperative Health Research within the Region of Augsburg (KORA) studies, Hannemann et al reported the prevalence of primary hyperaldosteronism among patients with hypertension, consistent with the subsequent three definitions: 1) increased ARR; 2) increased ARR and decreased plasma renin concentrations (PRC); and 3) increased ARR with increased serum aldosterone and decreased PRC. The prevalence of primary hyperaldosteronism using the aforementioned definitions are 5.9, 2.6, and 0.2% among patients with hypertension who aren't on antihypertensive medications, and 8.5, 4.9, and 0.5% among patients with hypertension who are on antihypertensive medications, for definitions 1), 2), and 3), respectively. Thus, if one uses very stringent criteria for the diagnosis of primary hyperaldosteronism (all 3 conditions, i.e., increased ARR, elevated aldosterone, and decreased PRC), the prevalence is far less than that using less strict criteria.

Recent guidelines from the Endocrine society emphasize that additionally to estimation of the ARR under standardized conditions, one among 4 confirmatory tests (oral sodium loading, saline infusion, fludrocortisone suppression, and therefore the captopril challenge test) be wont to establish a diagnosis of primary hyperaldosteronism, the selection of the confirmatory test partially being determined by local expertise, costs, and laboratory practices.

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