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SHORT REVIEW

Effect of primary aldosteronism on diabetes mellitus and hypertension: a short narrative review

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Abstract

Introduction: Primary aldosteronism (PA) is a condition of excess aldosterone levels due to a pathology of the adrenal gland. Diabetes and hypertension increase cardiovascular, renal, and cerebrovascular morbidity and mortality. If diabetes and hypertension coexist with underlying PA, complications increase. PA is usually not screened in every individual due to the cost and technical difficulties associated with the test. There are certain guidelines to screen for PA, like resistant hypertension with more than three antihypertensive agents, family history of PA, young-onset hypertension, diuretic-induced hypokalemia, etc. PA increases insulin resistance. It is still debatable whether PA poses a risk for new-onset diabetes mellitus or not. However, there is a consensus that PA increases metabolic and vascular complications in diabetic and hypertensive patients. The main aim of this article is to highlight a few aspects of aldosteronism and its impact on diabetes and hypertension.

Method: It is a short narrative review article. Articles were searched over PubMed and Google Scholar using the search words Primary Aldosteronism "And "Or" Diabetes mellitus. Full articles were only reviewed.

Result: PA is one of the common causes of secondary hypertension. It is not screened in all patients. Reviewed research papers support screening for PA in patients with resistant hypertension with more than three antihypertensive agents, family history of PA, young-onset hypertension, and diuretic-induced hypokalemia.

Conclusion: It is better to screen and treat PA in diabetic and hypertensive patients as per the screening guidelines released by the relevant Societies of Endocrinologists and the location of health institutes.

Keywords: Aldosterone; Diabetes; Primary aldosteronism; Resistant hypertension

INTRODUCTION

Aldosterone is a mineralocorticoid hormone that maintains the homeostasis of extracellular volume, blood pressure, and potassium levels. The production of aldosterone is controlled by extracellular potassium levels, Angiotensin II, and adrenocorticotropic hormone (ACTH). Aldosterone is produced by the adrenal gland and, to some extent, by visceral fat.[1] It maintains salt homeostasis and blood pressure. Excess production of aldosterone is known as aldosteronism. If the pathology for aldosteronism is within the adrenal gland, then it is known as primary aldosteronism.

A meta-analysis showed that in patients with PA, the prevalence of impaired fasting glucose was 31.2%, impaired glucose tolerance was 26.19%, and diabetes mellitus was 15.22%.[2] Excess of aldosterone can lead to resistant hypertension.[1] It increases the inflammatory and proliferative process in various cells, which is key to neurovascular complications associated with diabetes and hypertension.[2]

In resource-limited countries like Nepal, it is not always possible to screen for PA in every patient with diabetes and hypertension. This review article, first of all, aims to discuss briefly about PA (definition and pathophysiological effects). The next aim of this article is to discuss on impact of PA on diabetes and hypertension. Lastly, this article aims to provide a guide to reader about when to do PA screen among patients with diabetes and hypertension.

METHOD

It is based on a review of articles searched over PubMed and Google Scholar using Primary Aldosteronism "And", "Or" Diabetes mellitus as keywords to search. The latest pertinent articles till May 2024 were reviewed to prepare this article. Only full articles were reviewed. Articles only with abstracts, older than 30 years, were not included.

Primary Aldosteronism (PA)

Aldosteronism is a condition where there is an excess level of aldosterone in the body. It can be primary or secondary aldosteronism. Primary aldosteronism is due to a problem in the adrenal glands.[3] It results from the autonomous production of aldosterone by unilateral adrenal lesions like adrenal adenoma or bilateral adrenal lesions due to idiopathic hyperaldosteronism. It is also known as Conn's syndrome.[3] Secondary aldosterone results from overproduction of aldosterone due to problems in other parts of the body, like renal hypoperfusion.[1]

Aldosterone acts by activating mineralocorticoid receptors (MR). MR are expressed not only in renal tissues but also in cardiomyocytes, adipocytes, endothelial cells, macrophages, and vascular smooth muscle cells.[1] Aldosterone increases the absorption of sodium and excretion of potassium via MR. In these cells, aldosterone causes inflammation, proliferation, and migratory processes.[1]

Approximately one in five patients with PA has diabetes mellitus (DM).[3] Though the exact mechanism for DM in PA is a matter of discussion, insulin resistance and subclinical hypercortisolism might be the reasons.[4]

There is no consensus on when to screen for PA. Some expert guidelines recommend screening tests for PA in groups with a high prevalence of the disease, like treatment-resistant hypertension, hypertension associated with spontaneous diuretic-induced hypokalemia, family history of early onset hypertension or young onset (<40years) cerebrovascular accidents, adrenal incidentaloma, or family history of PA.[5] Urine steroid metabolites can help to screen for PA and aldosterone-producing adenomas noninvasively.[6]

Effects of Primary Aldosteronism

Metabolic syndrome is associated with PA more than essential hypertension(EH).[7] Dysregulated glucose metabolism was higher in people with PA than EH. It can be a part of metabolic syndrome, too.[7] Aldosterone excess, along with metabolic syndrome associated with diabetes, increases the likelihood of diabetes-related complications.[8]

Modest dysregulation of aldosterone production with the background of renin suppression, hypertension, or eukalemia can be primary aldosteronism.[9] Cardiovascular and renal complication goes parallel and silently along with aldosteronism.[9]

Aldosterone may increase insulin resistance by inducing MR in adipocytes and skeletal muscles.[1] It indirectly increases insulin resistance by increasing proinflammatory cytokines and reducing beneficial adipokines.[1] Aldosterone decreases extracellular potassium levels. Potassium is required for insulin secretion.[1,3] Aldosterone increases insulin resistance, probably because of hypokalemia, though the exact mechanism is not known.[1] Subclinical hypercortisolism can also increase glucose intolerance.[10]

Cpeptide has been shown to increase after blockage with spironolactone, which supports the hypothesis that a decrease in insulin secretion could be mediated by aldosterone.[11]

In adipocytes, aldosterone blocks insulin-induced glucose uptake. It impairs insulin sensitivity and induces insulin resistance.[2] Higher Blood urea nitrogen (BUN) and excessive secretion of urinary calcium may also be potential risk factors for DM in PA patients apart from older age, dyslipidemia, and overweight.[12]

There was a higher incidence of DM in PA patients than in patients with hypertension.[13,14] Diabetes and hypertension usually coexist, but underlying PA worsens hypertension, glycemia, and cardiovascular risk. PA is also prevalent in the Asian-Indian population with DM and hypertension.[15] The overproduction of aldosterone increases the risk of renal, cardiovascular, and cerebrovascular complications in patients with hypertension and diabetes.[16] PA should be screened in diabetic individuals who are taking more than three antihypertensive drugs.[17]

PA is common in diabetic patients with resistant hypertension.[17] There is an increased risk of developing new-onset diabetic mellitus in individuals with PA. Adrenalectomy in patients with PA decreases the risk of metabolic diseases in comparison to mineralocorticoid receptor antagonists.[18]

Aldosterone antagonism improves endothelial function in chronic heart failure and thus reduces overall mortality. It decreases blood pressure in patients with hypertension and diabetes. However, aldosterone antagonists like spironolactone could not improve endothelial function in these groups significantly.[11]

CONCLUSION

Primary aldosteronism, when it coexists with diabetes and hypertension, increases cardiovascular, cerebrovascular, and other complications. It is one of the common causes of secondary hypertension. So, PA should be screened and treated in patients who are at risk for developing PA.

DECLARATIONS

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Conflict of interest
None

Ethical clearance
Not applicable.

Consent for study
Not applicable

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