Continue

إرشادات jcem الألدوستيرونية pdf

As a library, NLM provides access to scientific literature. Inclusion in an NLM database does not imply endorsement of, or agreement with, the contents by NLM or the National Institutes of Health. Learn more: PMC Disclaimer | PMC Copyright Notice Int J Endocrinol Metab Disord. Author manuscript; available in PMC: 2016 Dec 21. Published in final edited form as: Int J Endocrinol Metab Disord. 2016 Jul 11;2(3):10.16966/2380-548X.129. doi: 10.16966/2380-548X.129 Primary Aldosteronism is the single most common cause of secondary hypertension and is associated with increased target organ injury. The Endocrine Society has recently released the updated Clinical Practice Guideline for Primary Aldosteronism entitled “The Management of Primary Aldosteronism: Case Detection, Diagnosis, and Treatment: An Endocrine Society Clinical Practice Guideline”. We review the updated Clinical Practice Guideline, highlighting the new recommendations and the implications that they may have in clinical practice. The recognition by the Endocrine Society’s Task Force that Primary Aldosteronism is a public health issue and that the population at risk for screening should be significantly expanded will surely have an impact in the clinical practice which hopefully will translate in better detection, diagnosis and treatment of patients with Primary Aldosteronism. Keywords: Primary Aldosteronism, Adrenocorticotrophic hormone, Aldosterone-producing adenoma The Endocrine Society has recently released the updated Clinical Practice Guideline for Primary Aldosteronism (PA) entitled “The Management of Primary Aldosteronism: Case Detection, Diagnosis, and Treatment: An Endocrine Society Clinical Practice Guideline” [1]. The updated guideline includes both specific new recommendations for case detection, diagnosis, and treatment of PA as well as more profound philosophical changes due to our current better understanding of this disease. The steroid hormone aldosterone is the main mineralocorticoid and is synthetized by the zona glomerulosa of the adrenal gland cortex [2,3]. The main physiological regulators of aldosterone synthesis and secretion are angiotensin II (the end product of the renin-angiotensin system), plasma potassium and the adrenocorticotrophic hormone (ACTH), beside a plethora of regulators that fine-tune aldosterone secretion under different physiological conditions [4]. Aldosterone binds to the renal mineralocorticoid receptor (MR) to exert its classical biological action stimulating sodium reabsorption and potassium excretion modulating gene expression in target cells [5,6]. Besides aldosterone classical epithelial actions that translate in the regulation of extracellular volume and blood pressure, the mineralocorticoid also exerts multiple biological actions in non-epithelial tissues [5]. Autonomous excess aldosterone synthesis and secretion is the hallmark of PA which is characterized by hypertension, and target organ injury and dysfunction, with the latest ones being even worse than the ones observed in hypertensive patients from other etiologies than PA [7-10]. The Clinical Practice Guideline for PA was first published by the Endocrine Society in 2008 [11]. Eight years later and with a substantial increase of our understanding on susceptible populations, prevalence, genetics, target-organ damage and therapies for PA, the 2008 Guideline [11] obviously needed an update. For that purpose, the Endocrine Society commissioned a Task Force composed of eight experts on PA to address this demand and generated the updated Guideline [1]. The updated Guideline not only include experts in the Task Force from four continents but it is also sponsored by scientific societies from all around the world including American Heart Association, American Association of Endocrine Surgeons, European Society of Endocrinology, European Society of Hypertension, International Association of Endocrine Surgeons, International Society of Endocrinology, International Society of Hypertension, Japan Endocrine Society, and The Japanese Society of Hypertension. Furthermore, the Task Force did not receive any corporate funding or remuneration to keep it as unbiased as possible. We review the Clinical Practice Guideline for PA emphasizing what is new in the updated one. Primary Aldosteronism (PA) is defined as a group of disorders in which aldosterone production is inappropriately high for sodium status, relatively autonomous of the major regulators of secretion (angiotensin II, plasma potassium concentration), and nonsuppressible by sodium loading. Excess aldosterone, inappropriately high for the salt intake status, causes hypertension, cardiovascular and renal damage, sodium retention, suppression of plasma renin, and increased potassium excretion that (if prolonged and severe) may lead to hypokalemia. PA is commonly caused by an adrenal adenoma, unilateral or bilateral adrenal hyperplasia, or in rare cases adrenal carcinoma or inherited conditions of familial hyperaldosteronism. It is critical to keep PA definition in mind since it will guide us in the analysis of PA case detection, diagnosis, and treatment. Multiple epidemiological studies in the last two decades have shown that PA has a prevalence of >5% (possible even >10%) of hypertensive patients, both in general and specialty settings [12]. First described in the mid 1950’s [13] and although reported by Jerome Conn as representing a significant fraction of hypertensive patients [14], PA went under the radar in the following decades, not because it was not present, but due to screening deficiencies relying in hypokalemia as a PA hallmark. Latter studies showed that hypokalemia is present in only~10-40% of patients with PA [15,16]. With better screening methods and not relying in the assumption that hypokalemia is a sine qua non hallmark of PA, a series of epidemiological studies have clearly shown that PA is much more common than previously thought [12]. The guidelines are a call for physicians to realize that PA patients are a significant proportion of their daily hypertension-related consults. If we assume, in general terms, that the worldwide incidence of hypertension is~22% [17] and between 5-10% of them suffer from PA [12], we can conclude that between 1.1% and 2.2% of the general population suffers from PA. These impressive prevalence numbers are the driving forces that probably lead the Task Force to declare PA as a major health care issue that impacts not only the individual but the society as a whole. Nowadays, nobody doubts that the main physiological effect of aldosterone in the kidney is to increase sodium reabsorption and potassium excretion. It is intuitive to think that an excess aldosterone, such as in PA, will cause an increased potassium excretion leading to hypokalemia. However, that is not the case in the majority of PA cases. Several epidemiological studies have shown that hypokalemia is present in only~10-40% of patients with PA [15,16]. The cause for the lack of hypokalemia in PA, particularly in the less florid cases, is not clear but what it is absolutely clear nowadays is that the use of hypokalemia as a PA screening test should be discontinued. PA is the single most common cause of secondary hypertension. Moreover, PA patients have higher cardiovascular morbidity and mortality than patients with essential hypertension with similarly elevated blood pressure [18-21]. Consequently, PA is much more than another cause of hypertension and targeted specific therapies, either surgical or medical, is preferred over non-specific blood pressure reducing therapies. Screening for PA is recommended for subjects who meet one of the following criteria: Subjects with sustained blood pressure (BP) above 150/100 mm Hg on each of three measurements obtained on different days; Subjects with hypertension (BP>140/90 mm Hg) resistant to three conventional antihypertensive drugs (including a diuretic); Subjects with controlled BP (