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Primary aldosteronism and cardiovascular risk. Review

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Primary aldosteronism (PA) is a common cause of secondary hypertension associated with excess cardiovascular morbidity [1]. Primary aldosteronism is underdiagnosed because it does not have a specific, easily identifiable symptom, and clinicians may be poorly informed about the disease [2]. Primary aldosteronism is the most common and treatable cause of secondary hypertension [3] and is associated with excessive and autonomous production of aldosterone. Three characteristic features of PA are high blood aldosterone, depressed renin level, and hypertension [4]. Hypokalemia was initially considered the main feature of PA; however, recent studies have shown that hypokalemia is present in only 28 % of patients with PA [5].

The most common causes of PA are bilateral idiopathic hypertrophy (BIH) (60 % of cases) and aldosterone-producing adenomas (APA; called Conn's syndrome) (35 % of cases, although these percentages change over time) [6]. Rare causes of PA are unilateral hyperplasia, familial hyperaldosteronism (types I—IV), aldosterone-producing carcinoma, and ectopic aldosterone production [1]. Previously, PA was thought to be a rare cause of mild to moderate hypertension (< 1 %) and hypokalemia was one of the main diagnostic criteria, but recent studies have shown that PA may be the most common cause of secondary hypertension. hypertension [1].

Primary aldosteronism is now thought to be a factor in 5—10 % of all hypertensive patients. Both PA and resistant hypertension (RH) are high-risk phenotypes associated with increased cardiovascular morbidity and mortality compared with non-PA and non-RH patients [7]. Some studies have shown that the prevalence of PA depends on the severity of hypertension; the higher the degree of hypertension, the higher the prevalence of PA [8]. Primary aldosteronism can occur in 20 % of patients with RH [9].

In recent years, researchers have identified many genetic abnormalities underlying sporadic and familial forms of PA, and as such are useful markers for improved diagnosis and treatment in the future [10]. Recent advances in the understanding of PA, especially regarding APA, emphasize the significant role of somatic mutations [11]. Somatic mutations present in most APA are generally responsible for autonomous overproduction of aldosterone. Often, the same mutations are found in patients with BIH and familial hyperaldosteronism (FH). There are several types of FH, each associated with different genetic mutations [12].

Most of the mutations found in patients with PA are located in genes encoding ion channels and pumps. New knowledge about the genetic mutations associated with the pathogenesis of PA may lead to the development of new pharmacological treatments targeting the mutated proteins [13], while understanding the mutations that occur in FH may improve the diagnosis of positive patients in the early stages of the disease, to the appearance of symptoms or cardiovascular complications [1].

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In recent years, numerous studies have shown that patients with PA have a high rate of cardiovascular (CV) events [14—16]. Patients with PA are more likely to experience myocardial infarction, heart failure, or stroke, and have a higher prevalence of atrial fibrillation (AF) independent of blood pressure (BP) [17, 18]. Compared with age-, sex-, and BP-matched essential hypertensive patients, patients with PA often demonstrate significant left ventricular hypertrophy (LVH) [19, 20] and increased aortic stiffness [21]. The pathogenesis is unclear and may be related to excessive blood aldosterone concentrations, independent of BP, with increasing evidence that long-term exposure to high aldosterone levels may promote fibrosis and inflammation of the arterial wall and/or cardiac myocytes [22].

Aldosterone may also cause endothelial dysfunction by increasing oxidative stress [23]. Thus, excess aldosterone is associated with increased myocardial fibrosis, myocardial hypertrophy, and arterial stiffness [24]. A meta-analysis based on 12 case-control studies concluded that PA is significantly associated with subclinical arteriosclerosis and arterial stiffness based on comparison of CV markers including carotid intima-media thickness (CCA-IMT), flow-mediated dilation (FMD), nitroglycerin-mediated dilation (NMD), aortic pulse wave velocity (aortic-PWV), augmentation index (ALX), ankle-brachial index (ABI) and the prevalence of carotid plaques in patients, especially with PA [14].

Investigators reported increased CCA-IMT and aortic-PWV in patients with PA. In addition, compared to normotensive controls, patients with PA had increased ALX scores and decreased FMD, which was associated with increased aortic stiffness and impaired endothelial dysfunction [25]. According to data from the German Conn Syndrome Registry, cardiovascular mortality and morbidity are more common in patients with PA than in patients with comparable essential hypertension [26]. However, overall mortality was reported to be not significantly different from hypertensive controls, although cardiovascular mortality was the leading cause of death in PA (50 % vs. 34 % in hypertensive controls) [26].

Primary aldosteronism occurs mainly in patients aged 20 to 60 years. There are no pathognomonic symptoms in PA patients. The three hallmarks of PA are high blood aldosterone, low/suppressed renin levels, and hypertension. Arterial hypertension in patients with PA is usually moderate or severe. A study by the Mayo Clinic in the United States showed that the mean BP in patients with PA was 184/112 ± 8/16 mm Hg [4]. Other studies have shown that a higher prevalence of PA is observed in patients with higher BP [27]. Thus, PA was diagnosed in 20.9 % of patients with «resistant hypertension» [28]. In addition, patients with APA have higher aldosterone levels and higher BP than patients with BIH [4]. Hypokalemia is recognized as the main feature of PA, but occurs in only 28 % of the total PA population [29]. The prevalence of hypokalemia was reported in 48 % of patients with APA and 17 % of patients with BIH [27]. Thus, normokalemic hypertension is currently the most common manifestation of the disease.

Other symptoms associated with PA include headaches, vision problems, fatigue, muscle cramps, muscle weakness, numbness, frequent urination, and thirst. Some of these symptoms are directly related to hypokalemia. For example, polyuria results from a renal concentration defect caused by hypokalemia.

For many years, PA was considered a rare cause of hypertension, and screening was limited to patients with hypertension and hypokalemia, which were considered the main features of PA. Recent studies have clearly shown that PA is the most common cause of secondary hypertension with a prevalence of 5—10 % in hypertensive patients [30]. Hypertension in patients with PA is usually moderate to severe, and hypokalemia occurs in 28 % of patients with PA [29]. Thus, a normokalemic hypertensive patient should not be excluded from PA screening [31, 32], and the degree of hypertension should be taken into account during PA screening. In addition, a certain group of young people (aged < 35 years) have persistent and significant hypokalemia but are not hypertensive. APA may be present in these cases. The absence of arterial hypertension is probably the result of the early onset of the disease, its short duration and effective mechanisms of prevention of arterial hypertension [4]. In addition, a review [32] highlighted that PA is very common in mild to moderate hypertensive, prehypertensive and even normotensive populations. Thus, screening for PA is a challenging task.

According to the Endocrine Society's Clinical Practise Guidelines [33], it is recommended to consider PA in patients with sustained BP > 150/100 mm Hg, confirmed on separate days, RH, Bp < 140/90 mm Hg, requiring four or more anti-hypertensive medications, hypertension with spontaneous or diuretic-induced hypokalemia, hypertension with an adrenal mass, hypertension with sleep apnoea, hypertension and a family history of early onset hypertension or stroke at

a young age, hypertension with a first-degree relative who has primary aldosteronism.

Regardless of these factors, PA is known to be associated with higher cardiovascular morbidity and mortality than essential hypertension [34, 35] and is still largely underdiagnosed [36]. Currently, less than 1 % of adults diagnosed with primary hypertension are screened for PA [37]. Early detection and specific treatment can reduce the risk of cardiovascular diseases. Thus, it is necessary to improve the degree of PA screening [38]. Increasing awareness and knowledge of PA among primary care physicians who have the initial contact with the hypertensive patient is crucial [39].

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ABSTRACT

Primary aldosteronism (PA), a significant and curable cause of secondary hypertension, occurs in 5—10 % of hypertensive patients, with prevalence dependent on the severity of hypertension. The main etiologies of PA include bilateral idiopathic hypertrophy (BIH) and aldosterone-producing adenoma (APA), while less common causes include unilateral hyperplasia, familial hyperaldosteronism (FH) types I—IV, aldosterone-producing carcinoma, and ectopic aldosterone synthesis. This condition, characterized by excessive secretion of aldosterone, leads to increased reabsorption of sodium and water along with potassium loss, culminating in the distinct clinical signs of elevated aldosterone, suppressed renin, and hypertension. It should be noted that hypokalemia is present in only 28 % of patients with PA and is not the main indicator. The association of PA with an increased risk profile for cardiovascular disease, regardless of blood pressure level, is notable. Patients with PA show an increased rate of cardiovascular events compared to patients with essential hypertension, adjusted for age, sex, and blood pressure level. Despite its prevalence, PA often remains undiagnosed, highlighting the need for enhanced screening protocols. The diagnostic process for PA involves a three-pronged assessment: the aldosterone/renin ratio (ARR) as the initial screening tool, followed by confirmatory and subtype tests. A positive ARR requires confirmatory testing to rule out false positives. Examination to detect PA should be carried out in patients with arterial hypertension:

moderate (> 160—179/100—109 mm Hg) or severe (> 180/110 mm Hg); resistant to treatment; with idiopathic or diuretic-induced hypokalemia; with an accidentally diagnosed tumor of the adrenal glands; if first-generation relatives are diagnosed with PA, or family history indicates early onset of arterial hypertension, or cerebrovascular disorders at a young age (< 40 years); with accompanying obstructive sleep apnea.

Keywords: primary aldosteronism, hypertension, cardiovascular risk in primary aldosteronism, screening.

РЕЗЮМЕ

Первинний альдостеронізм та серцево-судинний ризик. Огляд

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В огляді розглянуто первинний альдостеронізм (ПА) як головну причину вторинної гіпертензії та серцево-судинний ризик. Первинний альдостеронізм має місце в 5—10 % пацієнтів з артеріальною гіпертензією. Основні етіологічні чинники ПА — двобічна ідіопатична гіпертрофія та аденома, що продукує альдостерон. До менш поширених причин належать однобічна гіперплазія надниркової залози, сімейний гіперальдостеронізм типу I—IV, карцинома, що продукує альдостерон, та ектопічна продукція альдостерону. Цей стан характеризується надмірною секрецією альдостерону, призводить до збільшення реабсорбції натрію та води разом із втратою калію, що завершується чіткими клінічними ознаками підвищеного рівня альдостерону, зниженого рівня реніну та артеріальної гіпертензії. Гіпокаліємія трапляється лише у 28 % хворих на ПА і не належить до основних показників. Детально розглянуто зв'язок ПА з підвищеним ризиком розвитку серцево-судинних захворювань незалежно від рівня артеріального тиску. У хворих на ПА реєструють підвищену частоту серцево-судинних подій порівняно з пацієнтами з есенціальною гіпертензією з поправкою на вік, стать і рівень артеріального тиску. Попри поширеність, ПА часто залишається недіагностованим, що свідчить про потребу в розширених протоколах скринінгу. Діагностика ПА передбачає оцінку величини співвідношення альдостерон/ренін (початковий інструмент скринінгу), а потім — проведення підтверджувальних тестів. При порушенні співвідношення альдостерон/ренін слід виконати підтверджувальне тестування, щоб запобігти помилковим результатам. Обстеження на предмет виявлення ПА слід проводити в пацієнтів із артеріальною гіпертензією: помірною (> 160—179/100—109 мм рт. ст.) або тяжкою (> 180/110 мм рт. ст.), резистентною до лікування, з ідіопатичною або спричиненою діуретиками гіпокаліємією, випадково діагностованою пухлиною надниркових залоз, якщо у родичів першого покоління діагностовано ПА, або сімейний анамнез вказує на раннє виникнення артеріальної гіпертензії, або церебрально-судинні порушення в молодому віці (< 40 років), із супутнім обструктивним апное сну.

Ключові слова: первинний альдостеронізм, артеріальна гіпертензія, серцево-судинний ризик при первинному альдостеронізмі, скринінг.

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