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Consensus

SFE/SFHTA/AFCE consensus on primary aldosteronism, part 1: Epidemiology of PA, who should be screened for sporadic PA?

Consensus hyperaldostéronisme primaire SFE/SFHTA/AFCE, groupe 1 : épidémiologie de l'hyperaldostéronisme primaire (HAP), chez qui rechercher une forme sporadique d'HAP ?

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Abstract

Depending on the study, the prevalence of primary aldosteronism (PA) in patients with hypertension varies from 6 to 18%. Prevalence is higher in each of the following conditions, any one of which requires screening for PA: severe hypertension (systolic blood pressure [BP] ≥ 180 mmHg and/or diastolic BP ≥ 110 mmHg); resistant hypertension (systolic BP ≥ 140 mmHg and/or diastolic BP ≥ 90 mmHg despite adherence to a tritherapy including a thiazide diuretic); hypertension associated with hypokalemia (either spontaneous or associated with a diuretic); Hypertension or hypokalemia associated with adrenal incidentaloma. It should be borne in mind that PA can induce hypertension without hypokalemia or, less frequently, hypokalemia without hypertension. Finally, as cardiovascular and renal morbidity in PA is greater than in essential hypertension of equivalent level, screening for PA is indicated when cardiovascular or renal morbidity is more severe than predicted from BP level.

Keywords: Primary aldosteronism; Hypokalemia; Resistant hypertension; Severe hypertension; Adrenal incidentaloma; Cardiovascular morbidity

Résumé

Selon les études la prévalence de l'hyperaldostéronisme primaire (HAP) chez les patients hypertendus varie de 6 à 18 %. Cette prévalence est plus élevée dans chacune des conditions suivantes, qui justifient de rechercher systématiquement un HAP (une seule condition est suffisante) : hypertension artérielle (HTA) sévère (pression artérielle systolique [PAS] ≥ 180 mmHg ou pression artérielle diastolique [PAD] ≥ 110 mmHg) ; HTA résistante (PAS ≥ 140 mmHg et/ou PAD ≥ 90 mmHg malgré trithérapie comprenant un diurétique thiazidique) ; HTA associée à une hypokaliémie, qu'elle soit spontanée ou associée à la prise d'un diurétique ; HTA ou hypokaliémie associée à un incidentalome surrénalien. Il faut souligner qu'un HAP peut être responsable d'HTA sans hypokaliémie et, plus rarement, d'une hypokaliémie sans HTA. Par ailleurs, dans la mesure où la morbidité cardiovasculaire et rénale de l'HAP est plus élevée que celle observée dans une HTA de même niveau tensionnel, il apparaît justifié de rechercher un HAP lorsque le retentissement cardiovasculaire ou rénal de l'HTA apparaît disproportionné avec le niveau tensionnel.

Mots clés : Hyperaldostéronisme primaire ; Hypokaliémie ; Hypertension résistante ; Hypertension sévère ; Incidentalome surrénalien ; Morbidité cardiovasculaire

1. Introduction

The exact prevalence of primary aldosteronism (PA) is unclear, as reports have differed in methodology and study population.

Several clinical presentations, principally including hypertension, are associated with a high risk of PA. At-risk patients to

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be screened for PA show one of the following: severe hypertension (grade 3), resistant hypertension, hypertension associated with hypokalemia, or associated with adrenal incidentaloma. In addition it is necessary to screen for a familial form of PA in specific situations which include young age and familial history of hypertension, stroke or PA, as described in the following article by Zennaro et al.

2. PA prevalence in hypertension

Several studies in recent years have improved knowledge of PA prevalence in hypertension.

Gordon et al. in 1994 reported 8.5–13% prevalence of PA in 199 patients with hypertension and normal kalemia, depending on whether diagnosis was founded on elevated aldosterone-renin ratio (ARR) on two measurements confirmed by fludrocortisone test, or on a single measurement without confirmation [1]. Other authors subsequently reported prevalence of 6.1–18% in tertiary care centers, depending on the study population and hormonal diagnosis procedure (with or without confirmation) [2–6]. In the light of these findings, the Endocrine Society estimated PA prevalence in hypertension at ≥ 10% [7]. In 2012, Hannemann et al., in an epidemiological study of 2444 hypertensive patients in Germany, screening for PA by ARR elevation without confirmation test, reported prevalence of 7% [8].

There are two forms of PA, with differing prevalence in hypertension: idiopathic PA was more frequent (6.4%) than Conn's adenoma (4.8%) in the PAPY study [9].

The relation between hypertension and PA principally concerns the hypertensive action of aldosterone, as clearly shown in a large-scale epidemiological study based on the Framingham cohort [10]: in 1,688 subjects with normal blood pressure (BP), the risk of developing hypertension within a 4-year period was 14.8%, but this risk was multiplied by 1.61 in the upper quartile for aldosteronemia; ARR elevation, assayed in day-clinic in 3326 subjects with normal BP, was associated with a significant 1.16-fold extra risk (16%) of developing hypertension over a mean 3 years' follow-up [11].

3. PA detection in high-prevalence populations

R1.1: PA should be screened for in patients with severe hypertension (grade 3, systolic BP ≥ 180 mmHg and/or diastolic BP ≥ 110 mmHg)
(Strong; evidence ++)

Prevalence of PA increases with the grade of hypertension, as demonstrated in several studies conducted in hypertension patients [3,8,9]. In the PAPY study, Rossi et al. reported 6.6% PA prevalence in grade-1 hypertension, 15.5% in grade 2 and 19% in grade 3 [9]. A similar correlation of PA prevalence (18.3%) with the severity of hypertension was found by Hannemann et al., screening for PA on ARR [8]. Conversely, PA was rare in grade-1 hypertension, in which systematic PA screening would not be indicated, according to the Japan Endocrine Society [12].

4. Resistant hypertension

R1.2: PA should be screened for in patients with resistant hypertension (≥ 140/90 mmHg, despite adherence to lifestyle modifications and administration at optimal dose of ≥ 3 anti-hypertension drugs including 1 thiazide diuretic).
(Strong; evidence ++)

Just as prevalence of PA increases with hypertension grade, prevalence is high in resistant hypertension: doubled on average, to around 20%, depending on the report. Douma et al. reported PA in 11.3% (on salt loading test) and 20.9% (on ARR and elevated aldosteronemia without confirmation test) of patients with resistant hypertension in a cohort of 1616 [13]. Calhoun et al. reported 30% prevalence, but defined PA in terms of urinary aldosterone and plasma renin activity [14]. More recently, a German epidemiological study reported lower prevalence, at 11.9% [8].

5. Hypertension with hypokalemia

R1.3: PA should be screened for in patients with hypertension associated with permanent or intermittent hypokalemia (< 3.5 mmol/l), whether spontaneous or diuretic-induced, without obvious digestive potassium loss.
(Strong; evidence ++)

R1.4: Normal kalemia (≥ 3.5 to ≤ 5.0 mmol/l) in hypertension does not rule out PA, which should be screened for in case of normal kalemia associated with another indication for PA screening.
(Strong; evidence ++)

Until the present decade, hypertension associated with hypokalemia (< 3.5 mmol/l) was a prerequisite for PA screening [15]. This attitude was challenged in the light of high PA prevalence without hypokalemia, whether spontaneous or induced by a potassium-reducing diuretic. Mulatero et al. showed that only a low proportion (9–37%, depending on the center) of PA patients were hypokalemic [16]. Similar proportions were also reported elsewhere [4,5,8].

Nevertheless, hypertensive patients with PA show lower kalemia. Seiler et al. reported mean kalemia of 3.74 mmol/l