



Primary Hyperaldosteronism (Conn's Syndrome)

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Iuliana Bobanga, Cassandre Bénay,
and Vikram D. Krishnamurthy

Algorithmic Approach

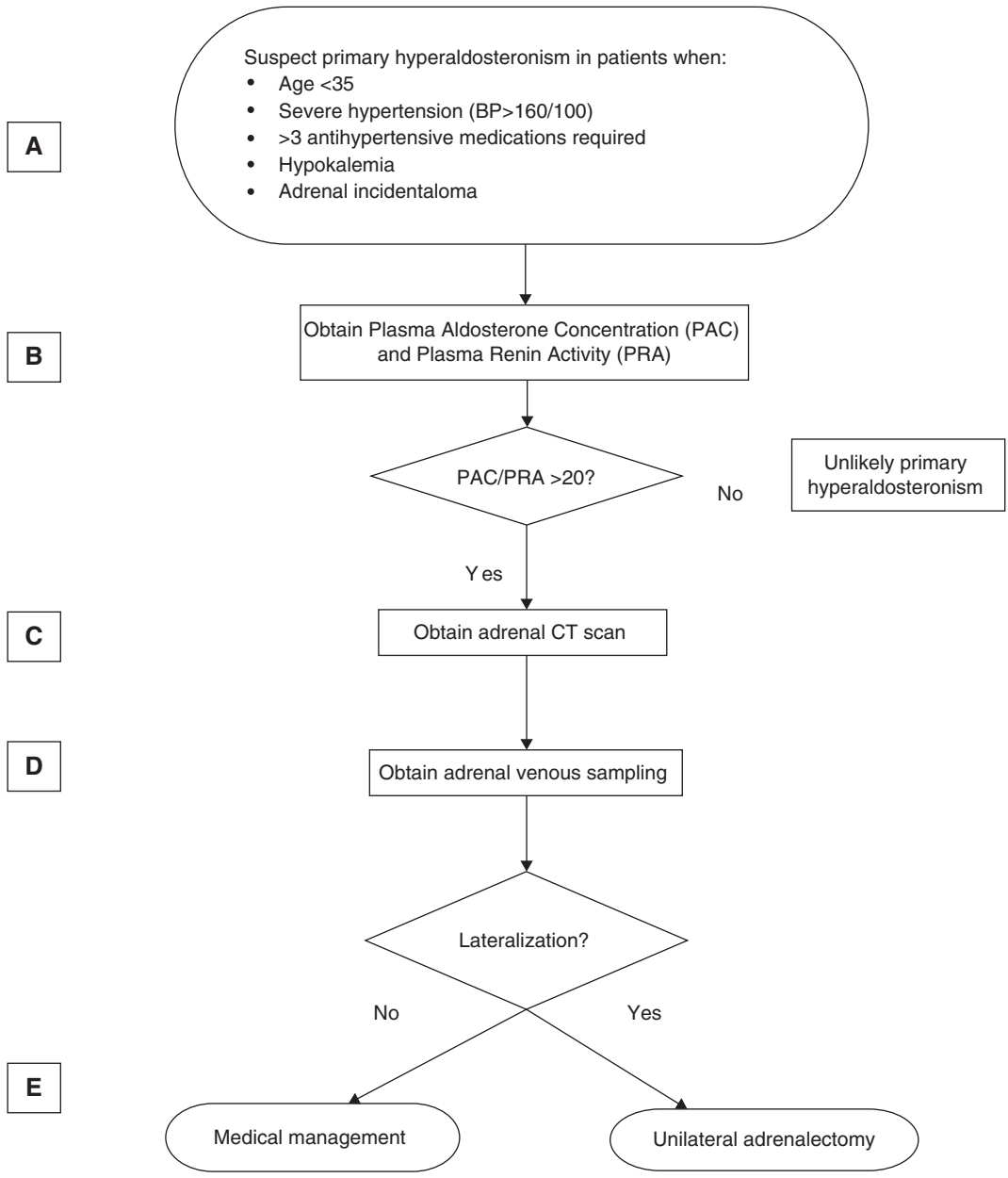
- A. Primary hyperaldosteronism is the most common cause of secondary hypertension and should be suspected in patients with hypertension that is diagnosed earlier than age 35, severe (BP < 160/100), persistent despite >3 medications, and present along with hypokalemia and/or an adrenal incidentaloma [1].
- B. The first diagnostic assessment in patients with Conn's syndrome requires demonstration of aldosterone excess along with suppressed plasma renin activity. Before measuring serum aldosterone levels and plasma renin activity, medications that can affect these measurements (e.g., angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, aldosterone receptor antagonists, and beta-blockers) are replaced with others (e.g., calcium channel blockers or alpha-adrenergic blockers) for several weeks. The aldosterone-to-renin ratio (ARR) is then calculated, with a ratio >20–40:1 along with an aldosterone level >15 ng/dL being suggestive of primary hyperaldosteronism (furthermore, an ARR >35 is 100% sensitive and 92% specific). If the ARR is equivocal but the diagnosis is still suspected, confirmatory testing may be performed (e.g., oral sodium loading, saline infusion, or captopril challenge testing) [1, 2].
- C. After establishing the diagnosis of hyperaldosteronism by biochemical studies, the next step is localizing the production to either a unilateral adenoma or bilateral hyperplasia. The imaging modality of choice is an “adrenal protocol” thin-cut computed tomography (CT) scan, which can demonstrate normal adrenal glands, thickened glands, or a unilateral adrenal mass [3]. An aldosterone-producing adenoma should be suspected in younger patients with hypertension whose CT scan reveals a unilateral adrenal mass and a contralateral normal adrenal gland.
- D. The laterality of aldosterone hypersecretion is then confirmed by bilateral adrenal venous sampling (AVS), which is the gold standard for distinguishing between unilateral and bilateral disease. The acquisition of AVS in every patient with primary hyperaldosteronism is controversial; however, many centers perform this routinely. Reliance on CT findings alone is questionable for two reasons: (1) aldosteronomas are typically small and may be missed (false negatives) and (2) non-aldosterone-secreting/nonfunctional adrenal adenomas increase with age (false positives). During AVS, aldosterone levels are obtained from the SVC, IVC, and adrenal veins. Cortisol levels are also obtained, with an adrenal vein to IVC cortisol ratio >2:1 confirming successful

I. Bobanga · C. Bénay · V. D. Krishnamurthy (✉)
Department of Endocrine Surgery, The Cleveland
Clinic, Cleveland, OH, USA
e-mail: krishnv3@ccf.org

cannulation of the adrenal veins. The aldosterone-to-cortisol ratio between both adrenal veins is then compared. A ratio of more than 4:1 lateralizes unilateral hypersecretion and identifies the gland to be removed [4, 5].

E. Hyperaldosteronism is typically cured by laparoscopic adrenalectomy; however, blood pressure may remain elevated secondary to

the vascular damage caused by the prior long-standing, severe hypertension. Factors predictive of the resolution of hypertension include female sex, body mass index (BMI) ≤ 25 , hypertension lasting ≤ 6 years, prescription of ≤ 2 antihypertensive medications, and decline in plasma aldosterone by 10 ng/dL on postoperative day 1 [6, 7].



Algorithm 111.1

References

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