

# PRIMARY HYPERALDOSTERONISM: CLINICAL AND THERAPEUTIC APPROACH OF A CENTER

Martins D<sup>1</sup>, Moreno C<sup>1</sup>, Baptista C<sup>1</sup>, Paiva I<sup>1</sup>, Guelho D<sup>1</sup>, Vicente N<sup>1</sup>, Cardoso LM<sup>1</sup>, Oliveira D<sup>1</sup>, Lages A<sup>1</sup>, Ventura M<sup>1</sup>, Carrilho F<sup>1</sup>  
<sup>1</sup>Endocrinology, Diabetes and Metabolism Department of Coimbra Hospital and University Centre, Portugal



**Introduction:** Primary hyperaldosteronism (PHA) is the main cause of secondary hypertension, with a prevalence estimated between 6-20% in patients with resistant hypertension. Clinical suspicion is critical, especially if aldosterone-to-renin ratio (ARR) >25, however the diagnosis is dependent on confirmatory evidence, including aldosterone suppression tests.

**Methods:** A retrospective study including 44 patients with suspected PHA, identified between 2010 and 2015 at the Endocrinology, Diabetes and Metabolism Department of Coimbra Hospital and University Centre, Portugal, was performed. Diagnostic criteria and treatment response were evaluated.

## Results

**N=44 patients**

Suspicion of secondary hypertension  
 Refractory hypertension  
 Hypertension with hypokalemia  
 Hypertension and adrenal mass

**N=16 patients**

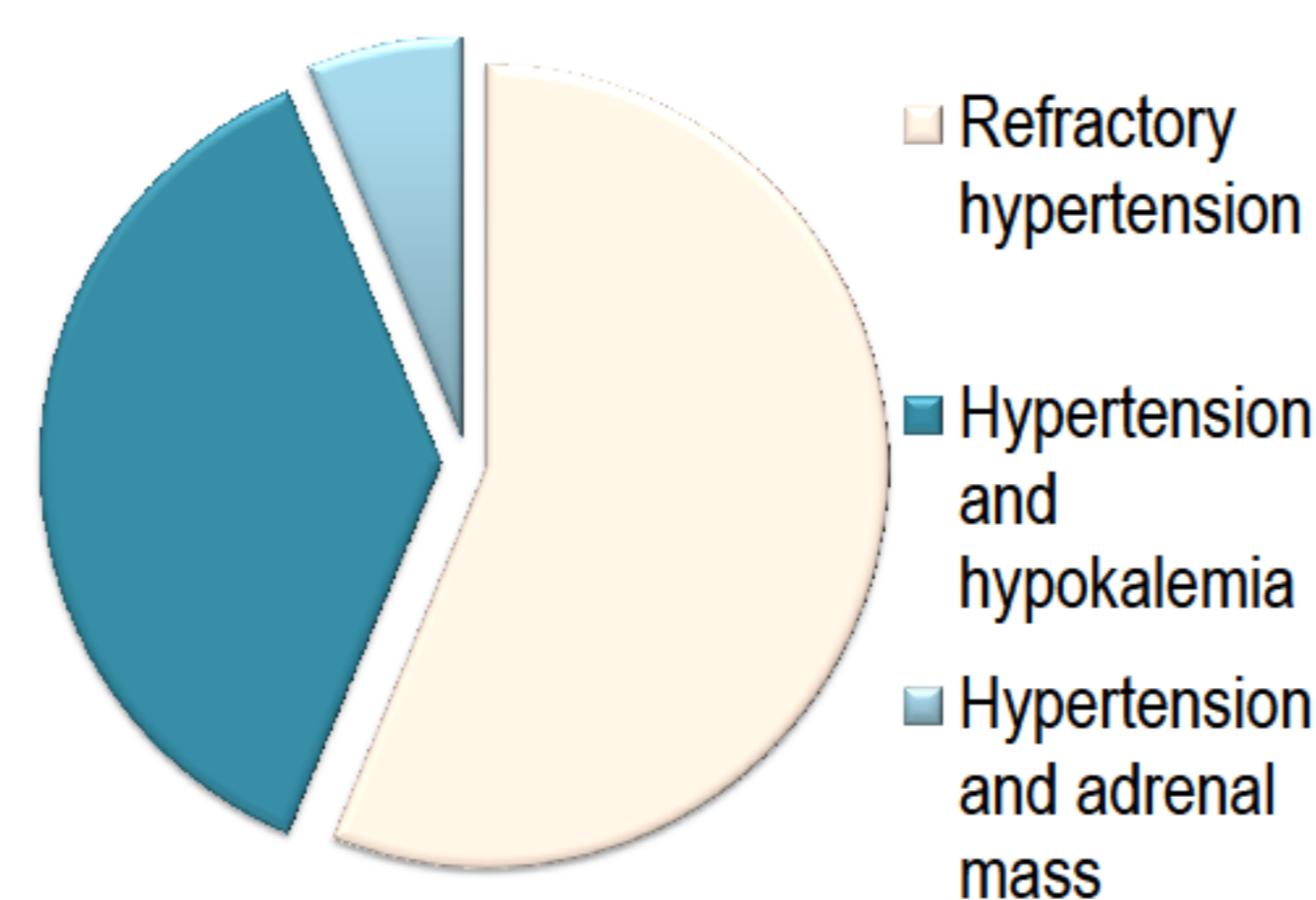
Men: n= 9 (56.25%); Women: n= 7 (43.75%)  
 Median age=60.5 years

**28 patients excluded**

- 15 patients: ARR < 25
- 11 patients: negative infusion saline test (IST)
- 2 patients: negative captopril test

### 1) Clinical presentation

Clinical presentation	n	%
Refractory hypertension	9	56.25
Hypertension and hypokalemia	6	37.5
Hypertension and adrenal mass	1	6.25



### 2) Hypertension (HT) - characterization

Median duration: 9.00 years

Stage of HT	I	14	87.5
	II	1	6.3
	III	1	6.3
	IV	0	0

Median number of antihypertensive drugs  
 N= 1.75 (min 1; máx 3)

Associated Diseases	N	%
Cardiovascular Stroke	0	0
<b>EAM</b>	<b>1</b>	<b>6.25</b>
AF	0	0
CHF	0	0
Chronic renal disease	0	0
Hypertensive retinopathy	0	0

ARBs	2 (12.5%)
ACE inhibitors	1 (6.3%)
<b>Calcium channel blockers</b>	<b>11 (68.8%)</b>
Beta-blockers	3 (18.8%)
<b>Thiazide diuretics</b>	<b>4 (25%)</b>
Spironolactone	1 (6.3%)
Antiadrenergics	2 (12.5%)

### 3) Analytical evaluation

Parameter	Result*	VR
Plasmatic renin $\mu\text{UI/mL}$	1.65	7-76
Plasmatic aldosterone $\text{pg/mL}$	213.00	40-310
<b>ARR</b>	<b>131.44</b>	-
Potassium $\text{mmol/L}$	3.45	3.50-5.10

**14 patients with ARR > 25 and positive aldosterone suppression test**

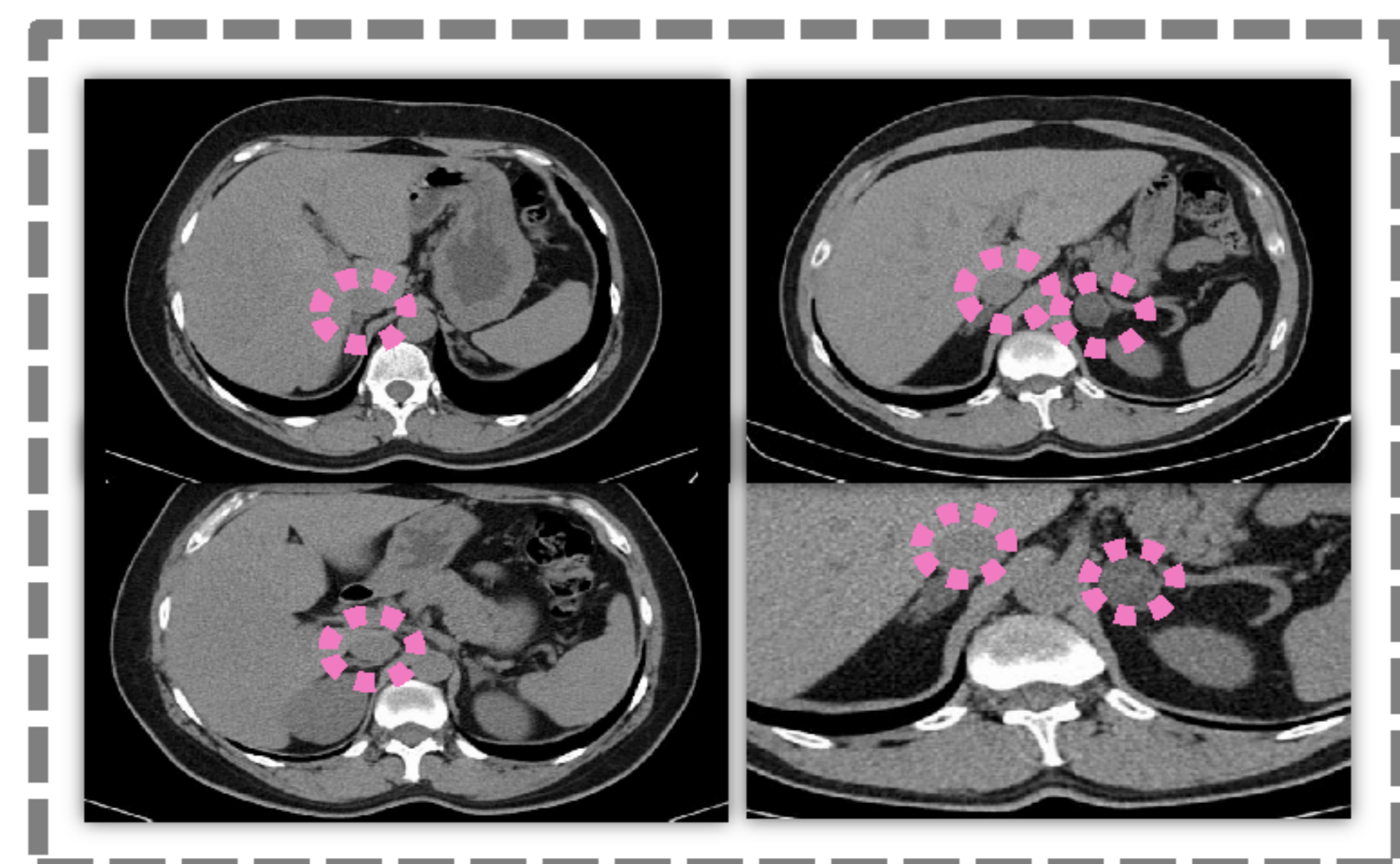
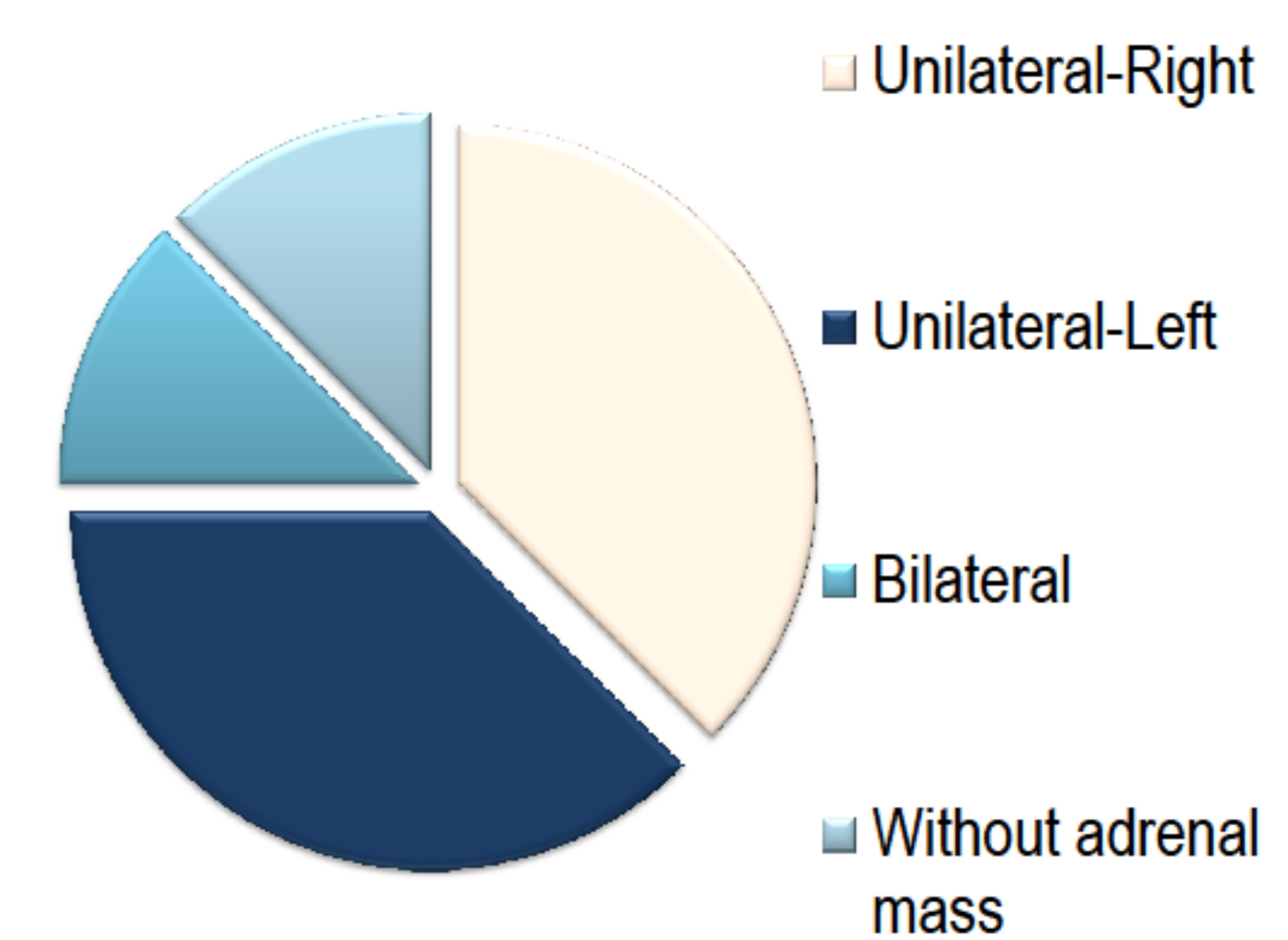
**2 patients with ARR < 25 and positive aldosterone suppression test**

\*Median

### 3) Radiological evaluation

Localization	n	%
<b>Unilateral adrenal lesion</b>	Right	6 <b>37.5</b>
	Left	6 <b>37.5</b>
Bilateral	2	12.5
Without adrenal lesion	2	12.5

Average size: **1.44±0.88cm**



### 4) Treatment

#### Surgical

**8 patients**

Adrenocortical adenoma (n=7)  
 Adrenal hyperplasia (n=1)

❖ Weiss Criteria: 0-1

#### Medical

**8 patients**

Bilateral adrenal lesions (n=2)  
 Non surgical conditions (n=1)  
 Refused surgery (n=5)

4 patients maintained medical therapy, with dose reduction

3 patients initiated therapy with espironolactone; 2 with eplerenone

### 5) Follow-up (6 months)

Parameter	Before treatment	After treatment	p
<b>Systolic BP<sup>1</sup> (mmHg)</b>	143.00	133.00	<b>0.008*</b>
<b>Diastolic BP (mmHg)</b>	82.00	80.00	0.197*
<b>Nr of antihypertensive drugs</b>	2.00	0.75	<b>0.004*</b>
<b>Plasmatic Aldosterone</b>	213.00	148.00	0.317*
<b>Plasmatic Renin</b>	1.65	20.00	<b>0.003*</b>

\*p<0.05;Friedman Test  
<sup>1</sup>Blood Pressure

Parameter	Medical treatment	Surgical treatment	p
<b>Systolic BP (mmHg)</b>	140.00	120.00	<b>0.021**</b>
<b>Diastolic BP (mmHg)</b>	80.00	80.00	0.953**
<b>Nr of antihypertensive drugs</b>	1.00	~0.00	0.397**
<b>Plasmatic Aldosterone</b>	205.50	132.60	<b>0.086**</b>
<b>Plasmatic Renin</b>	14.60	17.00	0.462**

\*\*p<0.05; Mann Whitney Test

**Conclusions:** On follow-up at 6 months, it was found a significant overall reduction in the levels of systolic BP and the number of antihypertensive drugs. Surgical treatment produced a more significant reduction in the levels of systolic BP compared with medical treatment alone and the reduction of serum aldosterone levels was also higher in the group undergoing surgery. Further studies are needed in order to clarify the ideal cut-off of ARR, as well as the benefits of medical vs surgical treatment.

**Bibliography:** Funder JW *et al.*, Case Detection, Diagnosis, and Treatment of Patients with Primary Aldosteronism: An Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab. September 2008, 93(9):3266–3281; Rossi GP *et al.*, A Prospective Study of the Prevalence of Primary Aldosteronism in 1,125 Hypertensive Patients. Journal of the American College of Cardiology, Vol. 48, No. 11, December 5, 2006:2293–300; Kline GA *et al.*, Medical or Surgical Therapy for Primary Hyperaldosteronism: Post-treatment Follow-up as a Surrogate Measure of Comparative Outcomes. Ann Surg Oncol, 2013, 20:2274-2278; Wu Vin-Cent *et al.*, Diagnosis and Management of Primary Aldosteronism. Acta Nephrologica, 2012, 26(3): 111-120; Muth A *et al.*, Systematic review of surgery and outcomes in patients with primary aldosteronism. BJS 2015, 102: 307-317; Jansen PM *et al.*, Test characteristics of aldosterone-to-renin ratio as screening test for primary aldosteronism. Journal of Hypertension, 2013, 31:000-000

