Published in final edited form as:

Ann Surg Oncol. 2013 March ; 20(3): 753–758. doi:10.1245/s10434-012-2670-2.

Adrenal Histologic Findings Show No Difference in Clinical Presentation and Outcome in Primary Hyperaldosteronism

Allison B. Weisbrod, MD, MPH¹, Richard C. Webb, MD¹, Aarti Mathur, MD¹, Stephanie Barak, MD², Smita Baid Abraham, MD³, Naris Nilubol, MD¹, Martha Quezado, MD², Constantine A. Stratakis, MD, D.Sc.⁴, and Electron Kebebew, MD¹

¹Endocrine Oncology Branch, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Bethesda, MD

²Laboratory of Pathology, Center for Cancer Research, National Cancer Institute, National Institutes of Health, Bethesda, MD

³Program of Reproductive and Adult Endocrinology, National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, MD

⁴Section on Endocrinology and Genetics, National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, MD

Abstract

Background—Primary hyperaldosteronism is most commonly due to a solitary cortical adenoma. Thus, some surgeons have suggested a subtotal adrenalectomy is a reasonable approach when a mass can be identified. On the other hand, adrenal vein sampling (AVS) is being used more frequently to distinguish patients with unilateral disease for adrenalectomy, even if a discrete mass is not identified on axial imaging. In these cases, surgical pathology may reveal a cortical adenoma, a cortical adenoma with hyperplasia, or cortical hyperplasia. The goal of this study was to compare the presentation and outcome among patients undergoing adrenalectomy and found to have different histologic features.

Methods—We performed a retrospective analysis of 136 patients with primary hyperaldosteronism. Ninety-five patients had an adrenalectomy for unilateral disease. The preoperative clinical and laboratory, and postoperative outcome of three aforementioned histologic groups were compared.

Results—Ninety-five patients underwent an adrenalectomy. We found no significant difference in age, gender, body mass index, duration of hypertension, number of antihypertensive medications, serum aldosterone level, serum renin level or adrenal vein sampling ratios among the 3 histologic categories. We also found no significant difference among the three categories in postoperative cure rate.

Conclusion—The rate of unilateral hyperplasia in patients with primary hyperaldosteronism (16%) is likely higher than previously reported which may be due to the increasing use of AVS. The clinical presentation and outcome of patients regardless of the histologic findings are similar. Our data also suggests that subtotal adrenalectomy would not be appropriate in patients with primary aldosteronism.

Keywords

Primary Hyperaldosteronism; Adrenal Vein Sampling; Aldosterone Producing Adenoma

Introduction

Primary hyperaldosteronism was first described by Jerome Conn as the association of an adrenal cortical adenoma with hypertension, hypokalemia and excessive urinary excretion of aldosterone.¹ Increased screening of hypertensive populations has identified primary hyperaldosteronism as the leading cause of secondary hypertension, with prevalence rates as high as 18% in patients with hypertension.²⁻³ If untreated, patients with primary hyperaldosteronism are at a greater risk for end organ damage resulting in heart and kidney dysfunction, and vasculature disease than those with hypertension attributed to other causes.^{2,4-6}

Once a diagnosis of primary hyperaldosteronism has been established, it is important to distinguish unilateral from bilateral adrenal disease. Unilateral disease has historically been synonymous with an aldosterone-producing adenoma, and may be treated surgically.⁷ On the other hand, bilateral adrenal disease is most consistent with the diagnosis of idiopathic hyperplasia, and is treated with mineralocorticoid receptor antagonists.⁷

Adrenal venous sampling (AVS) was first introduced in the 1960s to assist in the localization of unilateral adenoma, as the majority of aldosterone-producing adenomas (APAs) are less than 15mm.⁸ This technique was soon replaced by computed tomography (CT) scan as imaging resolution improved.⁹ However, recent publications have highlighted the inaccuracy of CT scan, citing that up to 68% of patients would have been inappropriately managed without AVS, thus advocating for use of AVS as a gold standard test before offering patients adrenalectomy.¹⁰⁻¹² Even with improved diagnostic efforts, untreated post-operative normotension is only achieved in 42% of operative candidates but most studies observe greater than 90% of their surgical cohort have improved blood pressure on a reduced number of anti-hypertensive medications.¹³⁻¹⁹ Several pre-operative indicators have been associated with improved outcomes (blood pressure control), including age < 40, female gender, body mass index (BMI) < 25, duration of hypertension < 6 years and the need for < 2 anti-hypertensive medications.¹⁸ There is limited data on whether the cause of unilateral primary hyperaldosteronism (adrenal histologic features showing unilateral hyperplasia and/or cortical adenoma) is associated with a different presentation and outcome in patients undergoing adrenalectomy.²⁰ Thus, the goal of this study was to determine the prevalence of unilateral adrenal histologic findings and any association with presentation and outcome in patients undergoing unilateral adrenalectomy.

Materials and Methods

Patients

One hundred thirty-six patients with primary aldosteronism were identified who were enrolled in prospective clinical protocols approved by the Office of Human Subject Research at the National Institutes of Health (NIH) Clinical Center. Medical records were retrospectively reviewed for all patients who met criteria for surgical intervention and underwent unilateral adrenalectomy. In order to meet surgical criteria at the NIH, a patient must have: (1) biochemical testing consistent with primary hyperaldosteronism (elevated serum aldosterone level with suppressed plasma renin, and elevated aldosterone-to-renin ratio >20), (2) secondary confirmation of the diagnosis with sodium chloride loading test, a captopril test, or a posture test, and (3) unilateral disease by AVS in all patients. No co-

secretors were included. Patient demographics, clinical information, preoperative and postoperative data was collected.

Adrenal Venous Sampling

Adrenal venous sampling was performed in all patients with primary hyperaldosteronism, as previously described, regardless of CT scan findings.⁹ Bilateral adrenal veins were simultaneously catheterized through access of the ipsilateral femoral veins. For right adrenal vein sampling, either a 4 french Mickaelsson, a Simmons 1 or a Cobra 2 catheter was used. For left adrenal vein sampling, a 4 french Simmon 2 or 3 catheter or a sub 3 french microcather was used. Samples from the right iliac vein was used as the peripheral venous samples to compare adrenal vein levels for cortisol and aldosterone. For all time points, a blood sample was collected from each adrenal vein and the right iliac vein. Two baseline levels were drawn prior to an intravenous bolus of 0.25mg of adrenocorticotropin hormone (ACTH). An ACTH infusion was then started (0.25mg in 250mL normal saline) at 150mL/hr. Additional samples were drawn at 5, 10 and 15 minutes after infusion of ACTH.

An aldosterone-to-cortisol (AC) ratio was calculated for each sample. If the AC ratio was at least four times greater on one side compared to both contralateral adrenal and peripheral ratios, a diagnosis of unilateral hyperfunctioning adrenal gland was made and the patient was offered an adrenalectomy.

Histopathologic Classification

Surgical pathology reports were reviewed to categorize patients by histopathologic diagnosis. Available tissue sections of unilateral hyperplasia were additionally reviewed by a pathologist at the NIH for confirmation of the histologic diagnosis. At our institution, adrenocortical hyperplasia is defined as an increase in the number of adrenocortical cells above the expected component of the zona glomerulosa (expected 5–10% of total cortex) without the presence of a cortical adenoma and the uninvolved adrenal tissues not being atrophied.

Outcomes

Clinical outcome by different histopathologic diagnosis was measured by change in number of antihypertensive medications as well as change in mean arterial pressure MAP both at time of discharge (post-operative value).

Statistical Methods

Statistical significance was defined as $p < 0.05$. Intra-group associations were analyzed using the Wilcoxon signed-rank test. The Kruskal-Wallis test was used to determine inter-group associations. Cure rates amongst groups were compared using a 3x2 Fisher's exact test. Wilcoxon signed-rank tests and Kruskal-Wallis tests were performed using StatView software (SAS Institute, Inc., Cary, NC). Fisher's exact test was performed using In-Silico Fisher's exact test (http://in-silico.net/statistics/fisher_exact_test/2x3, Fairbanks, AK).

Results

Ninety-five of the 136 patients underwent surgical intervention for unilateral primary hyperaldosteronism with histologic examination. Sixty-six patients had an aldosterone-producing cortical adenoma. Fourteen patients demonstrated an aldosterone-producing adenoma with diffuse adrenal hyperplasia. Histopathology of the remaining 15 patients showed diffuse cortical hyperplasia without evidence of a cortical adenoma (Figure 1). For these 15 patients, the diagnosis manifested as multinodular hyperplasia in 10 patients and non-nodular hyperplasia in 5 patients. There was no significant difference between these

groups in terms of age at diagnosis, gender distribution, body mass index (BMI), duration of hypertension, number of preoperative antihypertensive medications, or preoperative aldosterone or renin level. Additionally, there was no significant difference in the clinical and laboratory features associated with patient outcome by histologic groups (Table 1). Patients' clinical outcomes were followed for a median of 3 months, with a range of 0.1 to 87 months.

All three histologic categories showed significant post-surgical improvement in the number of anti-hypertensive medications required to control blood pressure at discharge ($p < 0.05$, Table 2). Additionally, all three histologic categories showed significant improvement in MAP at the time of discharge ($p < 0.05$, Table 3).

Discussion

Our results suggest that the clinical presentation and outcome of patients with unilateral primary hyperaldosteronism are similar regardless of the histopathologic diagnosis. A solitary cortical adenoma, previously believed to account for the vast majority of histologic diagnoses of surgical primary hyperaldosteronism, was not as prevalent in our series. This is likely due to the use of AVS, rather than radiography, for evidence of unilateral disease. Furthermore, all three histologic categories demonstrated similar postoperative cure rates and similar quantitative improvements in number of antihypertensive medications and MAP.

Of the 95 patients in our cohort treated for unilateral primary hyperaldosteronism, 69% were due to a solitary cortical adenoma, 15% were due to cortical adenoma with cortical hyperplasia, and 16% were due to cortical hyperplasia. Unilateral adrenal hyperplasia has been described as a rare disease throughout the literature; however, the frequency is rarely cited. Since Ross published the initial description of unilateral cortical hyperplasia, the majority of publications addressing unilateral adrenal hyperplasia were single case reports or small case series, with less than 30 cases described by 2005.^{12,21–41} A prospective study in Japan is the only large scale population-based trial we were able to identify that attempted to describe the prevalence of unilateral adrenal hyperplasia, finding this disease process in 0.1% of 1,020 hypertensive patients.⁴² Based on this, it has been estimated that unilateral adrenal hyperplasia accounts for less than 1% of all patients with primary hyperaldosteronism.³⁹

Since 2005, we identified six studies which assess patient outcomes based on histopathologic diagnosis of unilateral primary hyperaldosteronism. Unilateral adrenal hyperplasia accounted for 10 to 36% of the cohort in these series.^{7,20,43–46} In four of these studies, the diagnosis of unilateral adrenal hyperplasia was based exclusively on CT scan or CT scan in combination with other methods for lateralization technique; a tool which has since been described as inaccurate for this purpose.^{9,11–12} Murashima and colleagues used AVS exclusively in their series of 52 patients, while Quillo and colleagues used AVS for all patients except women less than 40 years of age with a clearly identifiable adrenal mass on imaging. The incidence of unilateral adrenal hyperplasia in these studies were 17% and 14%, respectively,^{20,43} which is similar to our finding of a 16% rate of unilateral adrenal hyperplasia.

The prevalence of cortical adenoma within cortical hyperplasia is poorly described in the literature. Murashima and colleagues identified an adenoma within hyperplasia in 3 of 52 patients (6%) while Proye and colleagues identified this diagnosis in 24 of 100 (24%).^{43,45} Our analysis identified a prevalence of 15%.

All histologic categories in our cohort achieved statistically significant improvement in the number of required post-operative antihypertensive medications as well as post-operative

MAP. Several investigators have observed clinical improvement in patients who had a histologic diagnosis consistent with a solitary cortical adenoma.^{7,44,46} However, in studies in which AVS was used to select patients for adrenalectomy, no significant difference in outcome, based on histologic diagnosis was observed.^{20,43,45}

In conclusion, as AVS is becoming widely used to select patients with primary hyperaldosteronism for adrenalectomy, there is a higher likelihood that these patients will have histologic findings showing higher rates of non-solitary cortical adenoma. We observed 16% of patients with primary hyperaldosteronism had unilateral adrenal cortical hyperplasia without cortical adenoma. The clinical presentation and outcome of patients regardless of the histologic findings are similar. As such, subtotal adrenalectomy may not be appropriate in patients with primary hyperaldosteronism, since cortical hyperplasia may account for increased hormone production in patients with cortical adenoma.

References

1. Conn JW, Louis LH. Primary aldosteronism: a new clinical entity. *Trans Assoc Am Physicians*. 1955; 68:215–231. discussion, 231-213. [PubMed: 13299331]
2. Young WF. Primary aldosteronism: renaissance of a syndrome. *Clin Endocrinol (Oxf)*. 2007 May; 66(5):607–618. [PubMed: 17492946]
3. Schwartz GL, Turner ST. Screening for primary aldosteronism in essential hypertension: diagnostic accuracy of the ratio of plasma aldosterone concentration to plasma renin activity. *Clin Chem*. 2005 Feb; 51(2):386–394. [PubMed: 15681560]
4. Stowasser M, Sharman J, Leano R, et al. Evidence for abnormal left ventricular structure and function in normotensive individuals with familial hyperaldosteronism type I. *J Clin Endocrinol Metab*. 2005 Sep; 90(9):5070–5076. [PubMed: 15941863]
5. Milliez P, Girerd X, Plouin PF, Blacher J, Safar ME, Mourad JJ. Evidence for an increased rate of cardiovascular events in patients with primary aldosteronism. *J Am Coll Cardiol*. 2005 Apr 19; 45(8):1243–1248. [PubMed: 15837256]
6. Herrada AA, Campino C, Amador CA, Michea LF, Fardella CE, Kalergis AM. Aldosterone as a modulator of immunity: implications in the organ damage. *J Hypertens*. 2011 Sep; 29(9):1684–1692. [PubMed: 21826023]
7. Tresallet C, Salepcioglu H, Godiris-Petit G, Hoang C, Girerd X, Menegaux F. Clinical outcome after laparoscopic adrenalectomy for primary hyperaldosteronism: the role of pathology. *Surgery*. 2010 Jul; 148(1):129–134. [PubMed: 20083287]
8. Webb R, Mathur A, Chang R, et al. What is the Best Criterion for the Interpretation of Adrenal Vein Sample Results in Patients with Primary Hyperaldosteronism? *Ann Surg Oncol*. Nov 3.2011
9. Mathur A, Kemp CD, Dutta U, et al. Consequences of adrenal venous sampling in primary hyperaldosteronism and predictors of unilateral adrenal disease. *J Am Coll Surg*. 2010 Sep; 211(3):384–390. [PubMed: 20800196]
10. Lonborg J, Mathur M, Grieve SM, et al. Constrictive pericarditis diagnosed by cardiac magnetic resonance. *J Am Coll Cardiol*. 2010 Nov 9.56(20):e39. [PubMed: 21050969]
11. Young WF, Stanson AW, Thompson GB, Grant CS, Farley DR, van Heerden JA. Role for adrenal venous sampling in primary aldosteronism. *Surgery*. 2004 Dec; 136(6):1227–1235. [PubMed: 15657580]
12. Magill SB, Raff H, Shaker JL, et al. Comparison of adrenal vein sampling and computed tomography in the differentiation of primary aldosteronism. *J Clin Endocrinol Metab*. 2001 Mar; 86(3):1066–1071. [PubMed: 11238487]
13. Steichen O, Zinzindohoue F, Plouin PF, Amar L. Outcomes of adrenalectomy in patients with unilateral primary aldosteronism: a review. *Horm Metab Res*. 2012 Mar; 44(3):221–227. [PubMed: 22395801]
14. Sawka AM, Young WF, Thompson GB, et al. Primary aldosteronism: factors associated with normalization of blood pressure after surgery. *Ann Intern Med*. 2001 Aug 21; 135(4):258–261. [PubMed: 11511140]

15. Lumachi F, Ermani M, Basso SM, Armanini D, Iacobone M, Favia G. Long-term results of adrenalectomy in patients with aldosterone-producing adenomas: multivariate analysis of factors affecting unresolved hypertension and review of the literature. *Am Surg*. 2005 Oct; 71(10):864–869. [PubMed: 16468537]
16. Satoh F, Abe T, Tanemoto M, et al. Localization of aldosterone-producing adrenocortical adenomas: significance of adrenal venous sampling. *Hypertens Res*. 2007 Nov; 30(11):1083–1095. [PubMed: 18250558]
17. Pang TC, Bambach C, Monaghan JC, et al. Outcomes of laparoscopic adrenalectomy for hyperaldosteronism. *ANZ J Surg*. 2007 Sep; 77(9):768–773. [PubMed: 17685956]
18. Zarnegar R, Lee J, Brunaud L, et al. Good blood pressure control on antihypertensives, not only response to spironolactone, predicts improved outcome after adrenalectomy for aldosteronoma. *Surgery*. 2007 Dec; 142(6):921–929. discussion 921–929. [PubMed: 18063077]
19. Zarnegar R, Young WF Jr, Lee J, et al. The aldosteronoma resolution score: predicting complete resolution of hypertension after adrenalectomy for aldosteronoma. *Ann Surg*. 2008 Mar; 247(3): 511–518. [PubMed: 18376197]
20. Quillo AR, Grant CS, Thompson GB, Farley DR, Richards ML, Young WF. Primary aldosteronism: results of adrenalectomy for nonsingle adenoma. *J Am Coll Surg*. 2011 Jul; 213(1): 106–112. discussion 112–103. [PubMed: 21489832]
21. Dye NV, Litton NJ, Varma M, Isley WL. Unilateral adrenal hyperplasia as a cause of primary aldosteronism. *South Med J*. 1989 Jan; 82(1):82–86. [PubMed: 2643170]
22. Ganguly A, Yum MN, Pratt JH, et al. Unilateral hypersecretion of aldosterone associated with adrenal hyperplasia as a cause of primary aldosteronism. *Clin Exp Hypertens A*. 1983; 5(10): 1635–1658. [PubMed: 6365363]
23. Banks WA, Kastin AJ, Biglieri EG, Ruiz AE. Primary adrenal hyperplasia: a new subset of primary hyperaldosteronism. *J Clin Endocrinol Metab*. 1984 May; 58(5):783–785. [PubMed: 6707184]
24. Ross EJ. Conn's syndrome due to adrenal hyperplasia with hypertrophy of zona glomerulosa, relieved by unilateral adrenalectomy. *Am J Med*. 1965 Dec; 39(6):994–1002. [PubMed: 5853475]
25. Sasagawa I, Nakada T, Hashimoto T, et al. Unilateral diffuse adrenal hyperplasia masquerading as aldosterone-producing adenoma in primary hyperaldosteronism. *Urol Int*. 1993; 50(4):218–222. [PubMed: 8506594]
26. Ganguly A, Bergstein J, Grim CE, Yum MN, Weinberger MH. Childhood primary aldosteronism due to an adrenal adenoma: preoperative localization by adrenal vein catheterization. *Pediatrics*. 1980 Mar; 65(3):605–609. [PubMed: 7360550]
27. Otsuka F, Otsuka-Misunaga F, Koyama S, et al. Hormonal characteristics of primary aldosteronism due to unilateral adrenal hyperplasia. *J Endocrinol Invest*. 1998 Sep; 21(8):531–536. [PubMed: 9801995]
28. Haenel, LCt; Hermayer, KL. A case of unilateral adrenal hyperplasia: the diagnostic dilemma of hyperaldosteronism. *Endocr Pract*. 2000 Mar-Apr; 6(2):153–158. [PubMed: 11421533]
29. Morioka M, Kobayashi T, Sone A, Furukawa Y, Tanaka H. Primary aldosteronism due to unilateral adrenal hyperplasia: report of two cases and review of the literature. *Endocr J*. 2000 Aug; 47(4):443–449. [PubMed: 11075725]
30. Oberfield SE, Levine LS, Firpo A, et al. Primary hyperaldosteronism in childhood due to unilateral macronodular hyperplasia. Case report. *Hypertension*. 1984 Jan-Feb; 6(1):75–84. [PubMed: 6319281]
31. Pignatelli D, Falcao H, Coimbra-Peixoto A, Cruz F. Unilateral adrenal hyperplasia. *South Med J*. 1994 Jun; 87(6):664–667. [PubMed: 8202780]
32. Mendlowitz M. A case of predominantly unilateral pseudoprimary hyperaldosteronism. *Mt Sinai J Med*. 1982 Jan-Feb; 49(1):76–77. [PubMed: 6979689]
33. Goh BK, Tan YH, Chang KT, Eng PH, Yip SK, Cheng CW. Primary hyperaldosteronism secondary to unilateral adrenal hyperplasia: an unusual cause of surgically correctable hypertension. A review of 30 cases. *World J Surg*. 2007 Jan; 31(1):72–79. [PubMed: 17180480]
34. Weinberger MH, Grim CE, Hollifield JW, et al. Primary aldosteronism: diagnosis, localization, and treatment. *Ann Intern Med*. 1979 Mar; 90(3):386–395. [PubMed: 218482]

35. Kem DC, Tang K, Hanson CS, et al. The prediction of anatomical morphology of primary aldosteronism using serum 18-hydroxycorticosterone levels. *J Clin Endocrinol Metab*. 1985 Jan; 60(1):67–73. [PubMed: 3964794]
36. Rao A, Melby JC. Idiopathic hyperplasia of the adrenal gland behaving like an aldosterone producing adenoma. *J Endocrinol Invest*. 1997 Jan; 20(1):29–31. [PubMed: 9075069]
37. Chen LG, Lee TI, Lin HD, Wang HC, Liu WY, Tang KT. Primary aldosteronism due to unilateral adrenal hyperplasia: a case report. *Zhonghua Yi Xue Za Zhi (Taipei)*. 1997 Feb; 59(2):114–120. [PubMed: 9175301]
38. Mansoor GA, Malchoff CD, Arici MH, Karimeddini MK, Whalen GF. Unilateral adrenal hyperplasia causing primary aldosteronism: limitations of I-131 norcholesterol scanning. *Am J Hypertens*. 2002 May; 15(5):459–464. [PubMed: 12022249]
39. Katayama Y, Takata N, Tamura T, et al. A case of primary aldosteronism due to unilateral adrenal hyperplasia. *Hypertens Res*. 2005 Apr; 28(4):379–384. [PubMed: 16138569]
40. Omura M, Sasano H, Fujiwara T, Yamaguchi K, Nishikawa T. Unique cases of unilateral hyperaldosteronemia due to multiple adrenocortical micronodules, which can only be detected by selective adrenal venous sampling. *Metabolism*. 2002 Mar; 51(3):350–355. [PubMed: 11887172]
41. Hirono Y, Doi M, Yoshimoto T, et al. A case with primary aldosteronism due to unilateral multiple adrenocortical micronodules. *Endocr J*. 2005 Aug; 52(4):435–439. [PubMed: 16127211]
42. Omura M, Saito J, Yamaguchi K, Kakuta Y, Nishikawa T. Prospective study on the prevalence of secondary hypertension among hypertensive patients visiting a general outpatient clinic in Japan. *Hypertens Res*. 2004 Mar; 27(3):193–202. [PubMed: 15080378]
43. Murashima M, Trerotola SO, Fraker DL, Han D, Townsend RR, Cohen DL. Adrenal venous sampling for primary aldosteronism and clinical outcomes after unilateral adrenalectomy: a single-center experience. *J Clin Hypertens (Greenwich)*. 2009 Jun; 11(6):316–323. [PubMed: 19527322]
44. Walz MK, Gwosdz R, Levin SL, et al. Retroperitoneoscopic adrenalectomy in Conn's syndrome caused by adrenal adenomas or nodular hyperplasia. *World J Surg*. 2008 May; 32(5):847–853. [PubMed: 18343972]
45. Proye CA, Mulliez EA, Carnaille BM, et al. Essential hypertension: first reason for persistent hypertension after unilateral adrenalectomy for primary aldosteronism? *Surgery*. 1998 Dec; 124(6):1128–1133. [PubMed: 9854594]
46. Celen O, O'Brien MJ, Melby JC, Beazley RM. Factors influencing outcome of surgery for primary aldosteronism. *Arch Surg*. 1996 Jun; 131(6):646–650. [PubMed: 8645073]

Figure 1.

Photo micrograph image of representative cases of adrenocortical hyperplasia of the zona glomerulosa of the adrenal cortex. **A)** Normal adrenal with 5–10% component of zona glomerulosa composed of smaller cells with less cytoplasm than the other cortical cells (see arrow). Hematoxylin and Eosin stain, 20X. **B)&C)** Two cases of adrenocortical hyperplasia showing hyperplastic zona glomerulosa, demonstrating an increase in number of cells. Hematoxylin and Eosin stain, 20X. In the setting of aldosterone excess, the most common findings are hyperplasia of the zona glomerulosa (see arrows) and micronodules.

Table 1

Clinical characteristics of study cohort

	Adenoma	Adenoma & Hyperplasia	Hyperplasia	Total	p value
Number (%)	66 (69%)	14 (15%)	15 (16%)	95	NA
Age at diagnosis Mean $\pm$ SD Number 40 years	49 $\pm$ 10.8 11	51.3 $\pm$ 10.4 1	54.2 $\pm$ 9.6 2	50.3 $\pm$ 10.3 14	0.19 0.82
Gender Male Female	38 28	11 3	11 4	60 35	0.26
Body Mass Index (Mean $\pm$ SD)	30.3 $\pm$ 5.8	32.4 $\pm$ 7.3	31.2 $\pm$ 5.1	30.7 $\pm$ 5.9	0.57
Duration of hypertension (years) $\pm$ SD Patients with hypertension 6 years	13.7 $\pm$ 9.6 16	10.0 $\pm$ 5.2 4	16.3 $\pm$ 11.3 4	13.6 $\pm$ 9.5 24	0.35 0.94
Antihypertensive Medications $\pm$ SD Patients with 2 medications	2.7 $\pm$ 1.2 32	3.2 $\pm$ 1.4 5	3.0 $\pm$ 1.4 5	2.8 $\pm$ 1.3 42	0.30 0.51
Aldosterone, ng/dL $\pm$ SD	39.5 $\pm$ 32.9	35.5 $\pm$ 18.0	48.7 $\pm$ 33.4	40.5 $\pm$ 31.2	0.52
Renin, ng/dL $\pm$ SD	0.9 $\pm$ 2.9	0.7 $\pm$ 0.4	0.6 $\pm$ 0.2	0.8 $\pm$ 2.4	0.33

Table 2

Change in number of anti-hypertensive medications.

	Number of Preoperative Anti-Hypertensive Medications	Number of Postoperative Anti-Hypertensive Medications	p value
Adenoma	2.7 ± 1.2	1.2 ± 1.1	<0.0001
(n=66)	(n=64)	(n=60)	
Adenoma & Hyperplasia	3.2 ± 1.4	2.0 ± 1.8	0.0277
(n=14)	(n=14)	(n=10)	
Hyperplasia	3.0 ± 1.4	2.0 ± 1.5	0.0121
(n=15)	(n=15)	(n=15)	
Total	2.8 ± 1.3	1.5 ± 1.3	<0.0001
(n=95)	(n=93)	(n=85)	

Data are presented as mean ± standard deviation

Table 3

Change in mean arterial blood pressure.

	Pre-Operative Mean Arterial Pressure	Post-Operative Mean Arterial Pressure	p-value
Adenoma	113.1 ± 15.6	102.7 ± 12.1	0.0002
(n=66)	(n=65)	(n=55)	
Adenoma & Hyperplasia	113.2 ± 12.0	103.1 ± 11.2	0.0262
(n=14)	(n=14)	(n=12)	
Hyperplasia	113.1 ± 10.2	106.1 ± 8.8	0.0090
(n=15)	(n=15)	(n=15)	
Total	113.1 ± 14.7	103.4 ± 11.4	<0.0001
(n=95)	(n=94)	(n=82)	

Data are presented as mean ± standard deviation