Original Article

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Incidence of Acute Kidney Injury after Adrenalectomy in Patients with Primary Aldosteronism

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Background: Aldosterone-induced glomerular hyperfiltration can lead to masked preoperative renal dysfunction in primary aldosteronism (PA) patients. We evaluated whether PA patients had a higher prevalence of acute kidney injury (AKI) after unilateral adrenalectomy. In addition, we identified risk factors for AKI in these subjects.

Methods: This retrospective study included 107 PA patients, and 186 pheochromocytoma patients as a control group, all of whom underwent adrenalectomy between January 2006 and November 2017 at Yonsei University Severance Hospital. The primary outcome was AKI within 48 hours after adrenalectomy. Univariate and multivariate logistic regression analyses were performed to identify predictors of AKI after adrenalectomy.

Results: Overall incidence of AKI was 49/293 (16.7%). In PA patients, the incidence of AKI was 29/107 (27.1%). In contrast, incidence of AKI was 20/186 (10.7%) in pheochromocytoma patients. Univariate and multivariate logistic regression analysis both showed a higher risk of postoperative AKI in PA patients compared to pheochromocytoma patients. In addition, old age, diabetes, longer duration of hypertension, lower preoperative estimated glomerular filtration rate, high aldosterone-cortisol ratio (ACR) and lateralization index (LI) were identified as independent risk factors for postoperative AKI in PA patients after unilateral adrenalectomy.

Conclusion: Incidence and risk of postoperative AKI were significantly higher in PA patients after surgical treatment. High ACR on the tumor side and high LI were associated with higher risk of AKI in PA patients compared to pheochromocytoma patients.

Key Words: Primary aldosteronism, Adrenalectomy, Renal insufficiency, Pheochromocytoma

Introduction

Primary aldosteronism (PA) is one of the most frequent causes of secondary hypertension and is characterized by high blood pressure, hypokalemia, low plasma renin activity, and excessive aldosterone production by breaking away from the renin-angiotensin system[1,2]. PA has long been considered to be a relatively benign form of hypertension due to the suppression of the renin-angiotensin axis which plays an important role in cardiovascular remodeling and damage[3]. However, other studies have shown that long-term exposure to high levels of aldosterone is associated with tissue fibrosis, vascular remodeling, and endothelial dysfunction[4-7]. In addition, TAPAI Study Group et al. reported that even mild impairment of renal function may predict residual hypertension after unilat-

eral adrenalectomy in patients with aldosterone-producing adenoma[8].

Aldosterone-induced renal impairments in PA patients present clinically with increased urinary albumin excretion and estimated glomerular filtration rate (eGFR)[9]. Urinary albumin excretion and eGFR in PA patients decrease after adrenalectomy or starting treatment with mineralocorticoid receptor antagonists. This can be explained by aldosterone's effects on glomerular hyperfiltration, inflammation, fibrosis, mesangial cell proliferation, podocyte injury, and endothelial inflammation[10].

Since aldosterone-induced glomerular hyperfiltration can lead to masked preoperative renal dysfunction in PA patients, it is important to be able to predict which patients are at risk of adverse renal outcomes. Thus, we evaluated whether PA patients had a higher prevalence of renal impairment after unilateral adrenalectomy compared to pheochromocytoma patients, who represented secondary hypertension not caused by PA. In addition, we evaluated risk factors for renal impairment after adrenalectomy in PA patients.

Methods

1. Study participants

Between January 2006 and November 2017, 522 patients with adrenal tumors were admitted to Yonsei University Severance Hospital. We excluded patients diagnosed with Cushing syndrome (n=12), patients who did not undergo adrenalectomy (n=207), patients who underwent bilateral adrenalectomy (n=9), and patients who were aged under 18 years old (n=1). Finally, a total of 293 patients were selected (Fig. 1). Of these patients, 107 were diagnosed with PA by a saline loading test, which was then localized by adrenal or abdominopelvic computed tomography (CT)[11]. The remaining 186 patients were diagnosed with pheochromocytoma and set as a control group. Pheochromocytoma was confirmed by serum or urine catecholamine, metanephrine, and vanillylmandelic acid testing and localized by adrenal or abdominopelvic CT[12]. Patients with PA or pheochromocytoma underwent unilateral adrenalectomy between January 2006 and November 2017 at Yonsei University Severance Hospital. This study was conducted in accordance with the principles of the Declaration of Helsinki, and the study protocol was approved by the Institutional Review Board (IRB) of Yonsei University Health System's Clinical Trial Center (approval number: 4-2019-0922). Because this was a retrospective observational study, the informed consent requirement was waived.

2. Data collection

Demographic details including sex, age, body mass index (BMI), smoking status, history of hypertension, and history of diabetes mellitus (DM) were collected by retrospective review of electrical medical records from the day of adrenalectomy before the operation. BMI was calculated by dividing body weight (kg) by height squared (m^2). After 5 minutes of seated rest, blood pressure was measured using an electronic sphygmomanometer. After overnight fasting, venous samples were collected to determine blood urea nitrogen, creatinine, sodium, potassium, chloride, total CO_2, uric acid, and total cholesterol levels, as well as eGFR. Albuminuria was measured by dipstick. We used a creatinine measurement method which was calibrated to be traceable to isotope dilution mass spectrometry. The eGFRs were calculated using the Chronic Kidney Disease-Epidemiology Collaboration equation (CKD-EPI)[13]. In all PA patients, adrenal vein sampling (AVS) was performed to evaluate aldosterone and cortisol levels in each adrenal gland. We as-

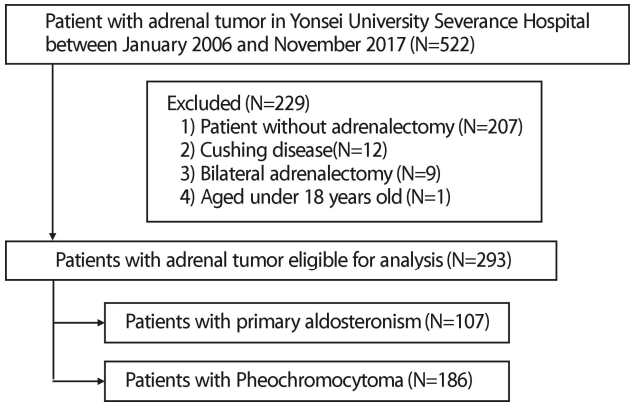

Fig. 1. Flow diagram of study design.

sessed peripheral cortisol, adrenal vein cortisol, inferior vena cava cortisol, peripheral aldosterone, adrenal vein aldosterone, and inferior vena cava aldosterone. The lateralization index (LI) was defined as the aldosterone-to-cortisol ratio of the adrenal vein on the tumor side divided by the aldosterone-to-cortisol ratio of the contralateral adrenal vein[14].

3. Statistical analysis

Continuous variables were expressed as means and standard deviations (SDs) or as medians with interquartile ranges (IQRs). Categorical variables were expressed as numbers and percentages. Comparisons between groups were made by way of analysis of variance or Student's *t*-test for continuous variables and by the chi-squared test or Fisher's exact test for categorical variables. The Kolmogorov-Smirnov test was performed to determine the normality of distribution of the parameters. If the resulting data did not show a normal distribution, the geometric mean±standard deviation was reported; the Mann-Whitney U test or Kruskal-Wallis test was used for multiple comparisons. Multivariate logistic regression analysis was performed to identify independent predictors of AKI after adrenalectomy. The results of the logistic regression analysis were presented as odds ratios (ORs) and 95%

Table 1. Baseline characteristics of patients

	Total (N=293)	PA (N=107)	Pheochromocytoma (N=186)	p-value
Age (years)	49.6±12.8	48.2±10.6	50.5±13.8	0.19
Male (n, %)	138 (47.1)	57 (53.2)	81 (43.5)	0.19
BMI (kg/m^2)	24.1±3.9	25.6±4.4	23.3±3.3	<0.001
Smoking (n, %)	82 (27.9)	33 (30.8)	49 (26.3)	0.32
DM (n, %)	74 (25.2)	16 (14.9)	58 (31.1)	0.003
Dyslipidemia (n, %)	64 (21.8)	26 (24.2)	38 (20.4)	0.37
Duration of hypertension (years)	1.0 (0.0-5.0)	4 (1.0-9.8)	0.4 (0.0-2.0)	<0.001
Number of anti-hypertensive medications	1.7±1.0	2.3±1.0	1.4±0.9	<0.001
Blood pressure (mmHg)				
Systolic	130.1±18.6	137.8±16.1	125.8±18.6	<0.001
Diastolic	81.5±13.3	87.6±13.7	78.0±11.8	<0.001
Creatinine (mg/dL)	0.8±0.2	0.9±0.3	0.8±0.2	<0.001
eGFR (mL/min/1.73 m^2)	93.5±19.2	87.7±21.3	96.8±17.2	<0.001
Uric acid (mg/dL)	4.7±1.4	5.1±1.6	4.4±1.3	<0.001
Total cholesterol (mg/dL)	159.5±38.1	162.8±31.9	157.6±40.5	0.24
Electrolytes (mmol/L)				
Sodium	140.6±2.5	140.5±2.8	140.6±2.4	0.77
Potassium	4.0±0.5	4.0±0.7	4.1±0.4	0.09
Chloride	104.5±3.2	103.0±2.3	105.2±3.3	<0.001
Total CO_2	24.7±3.2	25.8±3.4	24.2±3.0	<0.001
Albuminuria (n, %)	61 (21.8)	29 (27.1)	32 (17.2)	<0.001
Kidney size (cm)**				
Right	10.0±1.4	10.3±1.0	9.7±1.5	0.001
Left	10.5±1.1	10.7±1.0	10.4±1.2	0.05
Mean	10.3±1.0	10.5±0.9	10.1±1.0	<0.001

Note: Values for categorical variables are given as number (percentage); values for continuous variables, as mean±standard deviation or median (interquartile range). eGFR was calculated using the CKD-EPI equation.
**Kidney size was measured by computed tomography.
PA, primary aldosteronism; BMI, body mass index; DM, diabetes mellitus; eGFR, estimated glomerular filtration rate; CO_2, carbon dioxide; CKD, chronic kidney disease; CKD-EPI, chronic kidney disease-epidemiology collaboration.

confidence intervals (CIs). Statistical significance was defined as $p<0.05$. Data were analyzed using IBM SPSS statistical analysis software for Windows version 23.0 (IBM Corporation, Armonk, NY, USA) and SAS software version 9.4 (SAS Institute, Cary, NC, USA).

4. Primary outcomes

The primary outcome of interest in this study was AKI within 48 hours after adrenalectomy. Postoperative AKI was defined as an increase in serum creatinine level ≥0.3 mg/dL or a decrease in eGFR >25% from preoperative baseline values, in accordance with KDIGO (Kidney Disease Improving Global Outcomes) guidelines[15] and a previous study[16].

Results

1. Baseline characteristics

Baseline characteristics are shown in Table 1. The mean age of study participants was 49.6±12.8 years and 138 (47.1%) were men. Baseline BMI was 24.1±3.9 kg/m^2 and mean eGFR was 93.5±19.2 mL/min per 1.73 m^2. Of the 293 patients, 107 (35.9%) were diagnosed with PA and 186 (63.5%) were diagnosed with pheochromocytoma. Baseline BMI was significantly higher in PA patients (25.6 kg/m^2 vs. 23.3 kg/m^2, $p<0.001$). The duration of hypertension was significantly longer in PA patients (4 years vs. 0.4 years, $p<0.001$) and systolic blood pressure was significantly higher (137.8±16.1 mmHg vs. 125.8±18.6 mmHg, $p<0.001$). Baseline eGFR was significantly lower in PA patients (87.7±21.3 mL/min per 1.73 m^2 vs. 96.8±17.2 mL/min per 1.73 m^2, $p<0.001$) and albuminuria was more frequent (27.1% vs. 17.2%, $p<0.001$). In addition, mean kidney size was slightly larger in PA patients (10.5±0.9 cm vs. 10.1±1.0 cm, $p<0.001$).

2. Hormonal indexes in patients with PA

In PA patients, median (IQRs) plasma renin activity was 0.2 (0.1-0.3) ng/mL/hr and median plasma aldosterone concentration was 309.2 (184.5-437.0) pg/mL. The aldosterone-to-renin ratio was 154.6 (68.8-430.7) ng/dL per ng/ml/hr. The median LI was 8.25 (2.9-15.8) (Table 2).

3. Incidence of postoperative AKI

In both PA and pheochromocytoma patients, postoperative eGFR was significantly lower than preoperative eGFR (postoperative eGFR: 73.0±1.97 mL/min per 1.73 m^2 in PA patients and 85.1±2.07 mL/min per 1.73 m^2 in pheochromocytoma patients) (Fig. 2). However, the incidence of postoperative AKI was 29/107 (27.1%) in PA

Table 2. Hormonal indexes in patients with PA

	PA (N=107)
Peripheral	
Plasma renin activity (ng/mL/hr)	0.2 (0.1-0.3)
Plasma aldosterone concentration (pg/mL)	309.2 (184.5-437.0)
Aldosterone-to-renin ratio (ng/dL per ng/mL/hr)	154.6 (68.8-430.7)
Adrenal vein sampling	
Aldosterone, tumor side (pg/mL)	19,902.5 (10,327.8-36,361.5)
Aldosterone, contralateral (pg/mL)	2,490.6 (1,600.0-6,133.3)
Cortisol, tumor side (μg/dL)	417.2 (221.2-600.0)
Cortisol, contralateral (μg/dL)	432.1 (202.5-602.5)
Aldosterone-to-cortisol ratio, tumor side	4.7×10^{-3} (2.6×10^{-3}-11.5×10^{-3})
Aldosterone-to-cortisol ratio, contralateral	5.7×10^{-4} (5.2×10^{-4}–11.4×10^{-4})
Lateralization index	8.25 (2.9-15.8)

Note: All data in this table are expressed as median (interquartile range).
PA, primary aldosteronism.

patients but 20/186 (10.7%) in pheochromocytoma patients. The incidence of postoperative AKI was significantly higher in PA patients than in pheochromocytoma patients (p<0.001).

4. Predictors of postoperative AKI

First, risk factors for AKI after adrenalectomy were determined by univariate logistic regression analysis. According to univariate logistic regression analysis, PA patients had a significantly higher risk of AKI compared to pheochromocytoma patients (OR 1.88, 95% CI 1.02-5.14, p=0.04). Older age (OR 1.05, 95% CI 1.02-1.20, p<0.001), previous history of diabetes (OR 2.14, 95% CI 1.36-5.56, p=0.02), and longer duration of hypertension (OR 1.33, 95% CI 1.04-2.22, p=0.03) were also significantly associated with postoperative AKI. In addition, larger kidney size increased the risk of AKI (OR 1.28, 95% CI 1.04-1.66, p=0.05) (Table 3). Multivariate logistic regression analysis revealed that patients with PA were still at increased risk of postoperative AKI even after adjustment for confounding factors such as age, sex, DM, eGFR, systolic blood pressure, BMI, duration of hypertension, albuminuria, and mean kidney size (OR 4.32, 95% CI 1.33-11.42, p=0.01). In addition, lower eGFR was associated with an increased risk of postoperative AKI (OR 0.91, 95% CI 0.88-0.96, p=0.002). We further evaluated the risk factors in PA patients according to AVS values. Among the AVS values, high aldosterone-cortisol ratio (ACR) (OR 2.02, 95% CI 1.22-3.61, p=0.03) and high LI (OR 3.47, 95% CI 1.65-7.42, p=0.003) may be independently associated with AKI after adrenalectomy in PA

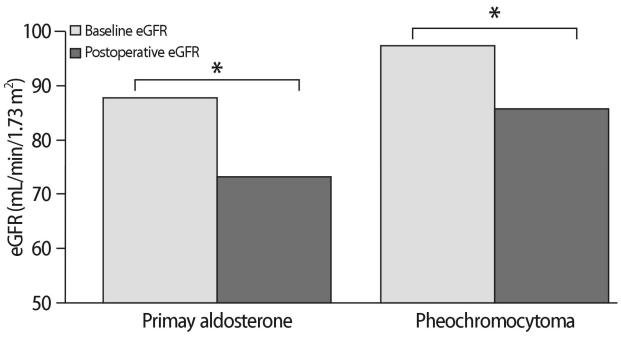

Fig. 2. Comparison of baseline and postoperative eGFR in PA and pheochromocytoma patients. Note: Baseline and postoperative eGFR were compared using the Student's t-test. (**A**) Primary aldosteronism, (**B**) Pheochromocytoma. *p-value=0.001. PA, primary aldosteronism; eGFR, estimated glomerular filtration rate.

Table 3. Logistic regression analyses for risk of postoperative AKI

	Univariate analysis		Multivariate analysis	
	OR (95% CI)	p-value	OR (95% CI)	p-value
PA (vs. pheochromocytoma)	1.88 (1.02-5.14)	0.04	4.32 (1.33-11.42)	0.01
Age (years)	1.05 (1.02-1.20)	<0.001	1.14 (1.04-1.26)	0.04
Male (vs. female)	1.02 (0.44-2.46)	0.88	0.60 (0.46-1.87)	0.49
DM (vs. non-DM)	2.14 (1.36-5.56)	0.02	5.74 (2.84-8.96)	<0.001
eGFR (mL/min/1.73 m^2)	1.04 (0.92-1.06)	0.64	0.91 (0.88-0.96)	0.002
SBP (mmHg)	1.03 (0.98-1.04)	0.42	1.02 (0.95-1.06)	0.66
BMI (kg/m^2)	1.02 (0.94-1.13)	0.89	1.06 (0.92-1.14)	0.82
Duration of HTN (years)*	1.33 (1.04-2.22)	0.03	1.12 (1.04-1.16)	0.02
Number of anti-hypertensive medications	1.24 (0.91-1.82)	0.46	1.29 (0.92-1.91)	0.45
Serum uric acid (mg/dL)	0.84 (0.67-1.24)	0.39	0.86 (0.70-1.44)	0.72
Serum total CO_2 (mmol/L)	1.02 (0.88-1.12)	0.52	0.99 (0.89-1.16)	0.80
Albuminuria (vs. no albuminuria)	1.32 (0.81-2.21)	0.38	1.58 (0.94-2.46)	0.06
Kidney size, mean (cm)	1.28 (1.04-1.66)	0.05	1.46 (1.16-1.90)	0.04

Note: Multivariate logistic regression analysis was performed after adjustment for confounding factors including age, sex, DM, eGFR, SBP, BMI, duration of HTN, albuminuria, and mean kidney size.
*Variable was log transformed.
AKI, acute kidney injury; OR, odds ratio; CI, confidence interval; PA, primary aldosteronism; DM, diabetes mellitus; eGFR, estimated glomerular filtration rate; SBP, systolic blood pressure; BMI, body mass index; HTN, hypertension; CO_2, carbon dioxide.

Table 4. Relationships between hormonal indexes and postoperative AKI in patients with PA

Variables	OR (95% CI)	p-value
Peripheral		
Plasma renin activity (ng/mL/hr)	1.36 (0.82-1.62)	0.74
Plasma aldosterone concentration (pg/mL)	1.48 (0.74-3.32)	0.42
Aldosterone-to-renin ratio	1.22 (0.71-1.84)	0.72
Adrenal vein sampling		
Aldosterone, tumor side (pg/mL)	1.82 (1.01-3.12)	0.05
Aldosterone, contralateral (pg/mL)	0.63 (0.43-1.18)	0.12
Aldosterone-to-cortisol ratio, tumor side	2.02 (1.22-3.61)	0.03
Aldosterone-to-cortisol ratio, contralateral	0.64 (0.41-1.52)	0.42
Lateralization index	3.47 (1.65-7.42)	0.003

Note: The relationship between each hormonal activity index and AKI development was analyzed by multivariate logistic regression analysis with the following covariates: age, sex, DM, eGFR, SBP, BMI, duration of HTN, albuminuria, and mean kidney size. *All hormonal activity indexes were log transformed.
AKI, acute kidney injury; PA, primary aldosteronism; OR, odds ratio; CI, confidence interval; DM, diabetes mellitus; eGFR, estimated glomerular filtration rate; SBP, systolic blood pressure; BMI, body mass index; HTN, hypertension.

patients (Table 4).

Discussion

In this study, we demonstrated that AKI occurred in PA patients after unilateral adrenalectomy. The risk of AKI in PA patients was significantly higher than in pheochromocytoma patients. In addition, higher tumor-side ACR and LI were independent risk factors for AKI after unilateral adrenalectomy in PA patients. Furthermore, we identified old age, previous history of diabetes, longer duration of hypertension, and lower preoperative eGFR as risk factors for postoperative AKI.

Renal function impairment is often observed in PA patients after adrenalectomy. A previous study reported that nearly 40% of PA patients showed postoperative eGFR decline after unilateral adrenalectomy. On the other hand, renal function impairment was not seen in the control group who underwent the same surgical treatment[16]. This suggests that unilateral adrenalectomy was not the cause of postoperative renal function decline. In our study, the incidence of postoperative AKI in PA patients was 27.1%. This was relatively lower than in the previous report. This difference in the incidence of AKI between studies could have been due to differences in sample size, follow-up duration, and baseline characteristics of the study groups. In the previous study, PA patients and pheochromocytoma patients were followed up for 6 months after unilateral adrenalectomy. The eGFR decline after unilateral adrenalectomy in PA patients continued for up to 1 month, stabilizing afterwards[17]. The primary outcome in our study was creatinine decline within 48 hours post-adrenalectomy. With longer follow-up duration, further decline in eGFR would be observed, leading to more recorded AKI events. Although lower than in the previous study, the incidence of AKI in our study was significantly higher in PA patients than in pheochromocytoma patients with the same surgical treatment. Thus, these findings imply that a decrease in renal function after unilateral adrenalectomy occurs mainly in PA patients rather than in pheochromocytoma patients, possibly due to physiologic changes before and after surgery.

Physiologic changes in PA patients after adrenalectomy are well documented. Normally, aldosterone is a key hormone in electrolyte and water homeostasis[1]. Patients with PA are exposed to uncontrolled high aldosterone levels due to pathologic adrenocortical lesions[2,18]. Excess aldosterone stimulates sodium and water reabsorption, resulting in extracellular fluid volume expansion, causing secondary hypertension[19,20]. This condition increases renal perfusion, which leads to glomerular hyperfiltration[21]. Thus, it is hard to evaluate actual renal function in PA patients before treatment because renal dysfunction in PA patients is usually masked by increased eGFR, due to the

hyperfiltration effect of aldosterone[22,23]. When PA patients are treated surgically or medically, their actual renal function is revealed[8,18,24]. This is consistent with the changes that were observed in our study.

We also identified risk factors for AKI in PA patients after unilateral adrenalectomy. Old age, previous history of diabetes, longer duration of hypertension, and lower preoperative eGFR were identified as risk factors for postoperative AKI. Old age, history of diabetes, and longer duration of hypertension are well-known risk factors for renal impairment[25-29]. However, previous studies reported that higher preoperative eGFR increased the risk of postoperative AKI, while lower preoperative eGFR was associated with the development of CKD in PA patients after adrenalectomy[16]. Our results differ somewhat from these previous reports, possibly due to the following reason. PA patients in our study had a significantly longer duration of hypertension compared to pheochromocytoma patients. Chronic hypertension and long-term aldosterone exposure can cause structural kidney damage such as tubulointerstitial fibrosis[30-32]. Thus, PA patients in this study may have had subclinical renal impairments that were concealed by the effect of aldosterone, leading to lower eGFR compared to pheochromocytoma patients who had a shorter duration of hypertension. As a result, lower eGFR caused by long-term hypertension was associated with postoperative AKI which was revealed after adrenalectomy when the aldosterone level dropped.

Long-term aldosterone exposure promotes endothelial dysfunction, as well as cardiovascular and renal inflammation and fibrosis[4,33-36]. According to multivariate logistic regression analysis for the evaluation of risk factors for renal impairment after adrenalectomy, higher ACR on the tumor side and higher LI were associated with an increased risk of renal impairment in PA patients compared to in pheochromocytoma patients. ACR and LI are both calculated using cortisol levels collected by AVS to negate the dilution of aldosterone caused by tributaries and malposition of the catheter when carrying out AVS[37,38]. Thereby, ACR and LI reflect more accurate assessments of aldosterone levels than the actual aldosterone levels. This is why aldosterone level was not examined as a risk factor for renal impairment, whereas ACR and LI were examined. Therefore, it can be said that higher aldosterone levels, when corrected according to cortisol levels, resulted in increased risk of AKI. This result is compatible with the results of previous studies.

The strengths of our study are as follows. First, previous studies about renal dysfunction in PA patients mostly compared PA patients to essential hypertension patients to evaluate the effect of aldosterone, since both patient groups had hypertension[39-41]. In contrast, our study compared PA patients with pheochromocytoma patients in which both groups underwent unilateral adrenalectomy. By comparing PA and pheochromocytoma patients, we compared the effect of aldosterone on renal function since PA and pheochromocytoma induce hypertension by different mechanisms. Second, we used AVS to assess hormone levels. Most previous studies used samples from peripheral blood vessels, which do not reflect actual aldosterone levels. By using AVS, we more accurately measured aldosterone levels in PA patients. Thus, we identified that higher ACR and LI were independent risk factors for AKI after unilateral adrenalectomy in these patients.

There are some limitations to this study. First, we only included PA patients who underwent unilateral adrenalectomy with AVS. Thus, the study population was relatively small. Second, aldosterone-induced renal impairment presents clinically with urinary albumin excretion. In this study, urine albumin measurement was performed by urine dipstick and quantitative measurement was not performed. Third, postoperative AKI was defined as an increase in serum creatinine $\geq$0.3 mg/dL or a decrease in eGFR >25% from baseline. However, urine output was not available as a definition for AKI. In addition, surgery involves bleeding, hypotension, and use of intermittent nephrotoxic agents which may affect renal function postoperatively. We did not consider perioperative complications. Finally, we did not evaluate long-term renal outcomes in PA patients who underwent adrenalectomy. A further study should be performed to evaluate long-term renal damage using quantitative tests for urinary albumin or protein excretion and following the AKI or CKD criteria.

In conclusion, the incidence and risk of AKI were significantly higher in PA patients after unilateral adrenalec-

tomy. Old age, previous history of diabetes, longer duration of hypertension, and lower preoperative eGFR were risk factors for postoperative AKI. In addition, higher tumor-side ACR and LI were risk factor for AKI in PA patients. Considering that AKI is a potent risk factor for CKD, proper management and regular follow-up should be performed on PA patients with unilateral adrenalectomy. Further studies are needed to evaluate the association between postoperative AKI and development of CKD.

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Conflict of interest

The authors declare that they have no conflicts of interest. This material has not been published previously and will not be submitted for publication elsewhere.

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