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# Pheochromocytoma with the Renovascular Hyperreninemia Attendant on Renal Artery Stenosis

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**ABSTRACT**

A 68-year-old female, diagnosed with essential hypertension, demonstrated a muscular depression caused by hypokalemia. Endocrinological data revealed elevated plasma rennin activity (PRA), plasma aldosterone concentration (PAC), and cathecholamine, and the computed tomography (CT) presented a 6 cm mass above the left kidney. I-metaiodobenzylguanidine (MIBG) scintigraphy showed an uptake in accord with the tumor and I-adosterol scintigraphy showed no abnormal accumulation. Magnetic resonance angiography (MRA) demonstrated left renal artery stenosis (RAS). Peak systolic velocity in the left renal artery measured by a duplex ultrasound was elevated. We considered that the left RAS accompanied by compression caused hyperreninemia. The tumor strongly adhered to the left renal artery and vein, so we performed a laparoscopic left nephroadrenalectomy. Two weeks after the operation, PRA, PAC, and cathecholamine were almost normalized. We present a rare case of pheochromocytoma with the renovascular hyperreninemia attendant on RAS diagnosed by MRA and a duplex ultrasound.

**KEYWORDS**: Pheochromocytoma; Renovascular hyperreninemia; Renal artery stenosis

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## INTRODUCTION

The coexistence of renal artery stenosis (RAS) and pheochromocytoma was reported in 1958 [1]. The reports concerning pheochromocytoma with RAS have been seen afterwards, and the diagnosis of RAS was performed by catheter angiography [2]. However, catheter angiography is an invasive test and has the possibility of postangiographic dissection or occlusion [3]. Recently, the utility of other noninvasive methods to diagnose RAS have been reported [4-6]. We report the pheochromocytoma with the renovascular hyperreninemia attendant on RAS diagnosed by using magenetic resonance angiography (MRA) and a duplex ultrasound preoperatively.

## CASE REPORT

A 68-year-old female, diagnosed with essential hypertension, demonstrated muscular depression caused by hypokalemia at the outpatient clinic. Endocrinological data revealed elevated PRA, PAC, and cathecholamine, and the CT presented a 6 cm mass above the left kidney (Figure 1). Both PRA and PAC values, measured 2 or more times on a similar condition, were also elevated. The patient was referred to the Juntendo Shizuoka Hospital. She had had a history of headaches associated with palpitations and diaphoresis.

In the pooled urine, a total catecholamine and the noradrenaline were elevated. A 123I-metaiodobenzylguanidine (MIBG)




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case report



Figure 1. CT scan showing a 6 cm mass above the left kidney
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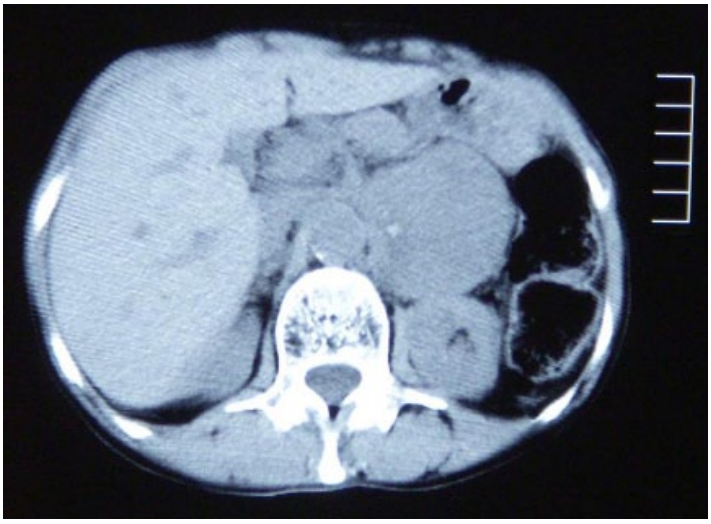

Figure 2. MRI on colonal section demonstrated left RAS caused by tumor compression
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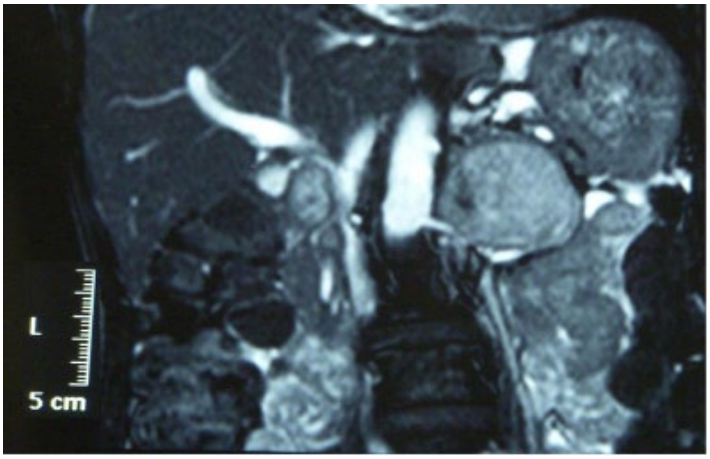


scintigraphy showed an uptake in accord with the tumor, though a 131I-adosterol scintigraphy showed no abnormal accumulation. She was preoperatively diagnosed with a pheochromocytoma arising from the adrenal glands or retroperitoneum.

The abdominal magnetic resonance imaging system (MRI) (Figure 2) and magentic resonance angiography (MRA) (Figure 3) were performed and demonstrated left RAS caused by tumor compression.

Figure 3. MRA on axial section demonstrated left RAS caused by tumor compression
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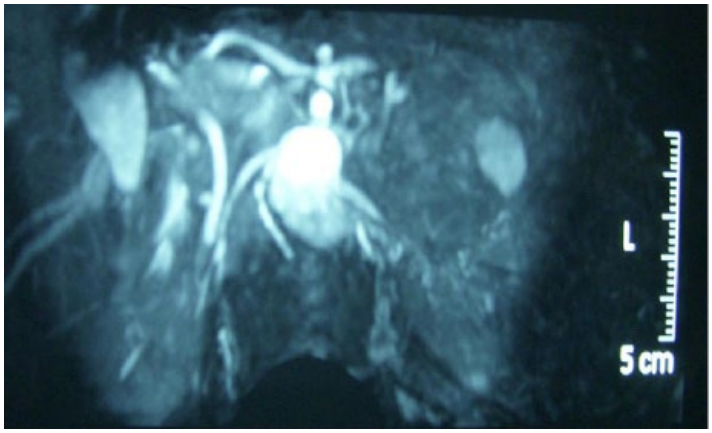


The renal-aortic ratio (RAR) was calculated by dividing the peak systolic velocity in the renal artery by that in the aorta using a Doppler ultrasound [4]. The right and left peak systolic velocity in the renal artery was 30.3 cm/s and 101.1 cm/s. Peak systolic velocity in the aorta was 45.6 cm/s. The right and left RAR were 0.66 and 2.22, respectively.

We considered that the left RAS accompanied by tumor compression caused hyperreninemia. A tumor strongly adhered to the left renal artery and vein, so we performed a laparoscopic left nephroadrenalectomy. The systolic blood pressure that rose up to 180 mm Hg during the procedure returned to a normal value when temporarily interrupting the operation. The pathological finding was a paraganglioma arising from the left renal hilus. In addition, a slight arteriosclerotic change was identified in a left renal artery.

Two weeks after the operation, PRA, PAC, and cathecholamine were almost normalized.

## DISCUSSION

Nephroadrenalectomy might not be common in the case that pathological finding is a benign condition. However, we selected the nephroadrenalectomy because the malignant pheochromocytoma had been strongly doubted by the adhesion and vascularization of the tumor during the procedure.

After laparoscopic surgery for the pheochromocytoma, the case that the local recurrence occurred by sowing in 3 to 4 years was reported [7]. The sowing caused by the tumor capsule injury must be avoided [8].

Not pushing the tumor strongly and keeping the lower pneumoperitoneum pressure within the possible range prevents a hypertensive crisis. Matsuda et al. reported that blood pressure variation in the laparoscopic surgery was lower compared to the conventional open adrenalectomy [9]. We consider laparoscopic surgery appropriate for the pheochromocytoma.

### *Causes of the hyperreninemia in pheochromocytoma*

Several investigators reported the pheochromocytoma with hyperreninemia [2]. RAS, which was on the same side as the tumor, had accompanied by tumor compression [10]. However, the case that RAS did not present during the operation or the catheter angiography, and the case for whom RAS improved by administering the hypotensive drug, were reported [11-14]. The vasoconstrictive effect on an excessive catecholamine was considered the cause of RAS in such a case [11, 15]. PRA does not rise in the noradrenaline secretion type but rises in the adrenaline secretion type or the dopamine secretion type [16]. This corresponds with the use of the secretion of the renin through β receptor. On the other hand, Brewster et al. reported that the vasoconstrictive effect was strong in the tumor outside the adrenal gland of the noradrenaline domination type [11]. PRA increased by renal ischemia resulting from vasoconstrictive action of noradrenaline secreted from the tumors [17].

Other considerable mechanisms for hyperreninemia include redistribution of intrarenal blood flow, concomitant renal artery lesions, including atherosclerosis, fibromusclar hyperplasia, and transient vasospasm during cathecholamine crisis [18].

In our case, administering the hypotensive drug decreased PRA slightly. Two main causes were considered for the hyperreninemia. One is the vasoconstrictive effect of the noradrenaline, and the other is RAS attendant on tumor compression.

### *Diagnosis of the RAS*

For the diagnosis of RAS, CT angiography (CTA), MRA, a Doppler ultrasound, and catheter angiography, including digital subtraction angiography (DSA), have been used. In the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, duplex ultrasonography, CTA, and MRA are recommended screening tests to establish the diagnosis of RAS [19]. However, the precision in CTA and MRA decreases in the case that the kidney function is deceased, though the sensitivity and specificity are high. Captopril renal scintigraphy is not recommended. Catheter angiography is the gold standard when the clinical index of suspicion is high and the results of noninvasive tests are inconclusive. However, we cannot distinguish whether the patient with RAS has a functional problem with catheter angiography. The Doppler ultrasound enables us to evaluate, functionally, the difference in pressure, in addition to the degree of RAS. The difficulty in this examination is that precision depends on the capability of the investigator.

In consideration for her kidney function, we performed the non-enhanced MRA and a Doppler ultrasound.

In conclusion, several factors may affect the hyperreninemia in this case. Noninvasive methods without the catheter angiography were useful for the diagnosis of RAS. The Doppler ultrasound helps us to evaluate functionality. Performing the Doppler ultrasound, in addition to CTA or MRA, might be effective for the objective diagnosis of RAS.

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