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CASE REPORT

Accessory renal arteries - a source of hypertension: A case report

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Abstract

BACKGROUND

Secondary hypertension is a relatively rare condition most commonly caused by renovascular disease due to atherosclerotic vascular disease or fibromuscular dysplasia. Although accessory renal arteries are frequent, to date, only six cases of secondary hypertension determined by their existence have been reported.

CASE SUMMARY

We describe a case of a 39-year-old female who came to the emergency department with an urgent hypertensive crisis and hypertensive encephalopathy. Despite normal renal arteries, the computed tomography angiography revealed an inferior polar artery with 50% stenosis of its diameter. Conservative treatment with amlodipine, indapamide and perindopril was adopted, leading to blood

pressure control within one month.

CONCLUSION
To the best of our knowledge, there are controversies regarding accessory renal arteries as a potential etiology for secondary hypertension, but the seven similar cases already described, along with the current case, could reinforce the necessity of more studies concerning this subject.

Key Words: Accessory artery; Secondary hypertension; Renal artery stenosis; Renovascular disease; Risk factor; Young patients; Case report

Core Tip: Unfortunately, nowadays, there are still a lot of young patients with hypertension that receive medical treatment, without any further research of secondary causes of hypertension. Also, secondary hypertension caused by the presence of accessory renal arteries is a controversial subject in the medical literature because there are studies confirming this association and studies that have found no association.

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INTRODUCTION

Hypertension is a frequent medical condition that is increasingly observed around the world. In 5%-15% of cases, hypertension arises as a result of an identifiable cause – secondary hypertension. The most common pathological process leading to secondary hypertension is renovascular disease, which is the result of atherosclerotic renovascular disease or fibromuscular dysplasia in most of these cases. No practical benefits have been found from screening all hypertensive patients for secondary hypertension. However, looking for a potential cause of hypertension might be of great importance in patients younger than age 40 and in patients with severe (grade 3) hypertension or a hypertensive emergency [1]. We report a case of secondary hypertension caused by stenosis of an accessory left renal artery; a very rare condition in the medical literature.

CASE PRESENTATION

Chief complaints

A 39-year-old white female who resided in the countryside presented to the emergency department with vertigo and a fronto-occipital headache.

History of present illness

Her symptoms started 3 months prior and had progressively worsened.

History of past illness

The patient's medical history comprised severe hypertensive disease with a maximum systolic blood pressure of 230 mmHg, first diagnosed at the age of 20. She inconsistently took bisoprolol 5 mg per day, candesartan 32 mg per day and indapamide 1.5 mg per day, but she had interrupted her treatment 4 months prior.

Personal and family history

The patient denied smoking, consuming alcohol, and substance abuse and claimed that her mother also suffered from hypertension.

Physical examination

The physical examination revealed an overweight woman (BMI = 29 kg/m^2) with a right arm blood pressure (BP) of 280/140 mmHg and a left arm BP of 275/140 mmHg, normal breath sounds, normal respiratory rate, normal heart sounds, no vascular murmurs, normal symmetric peripheral pulse and

normal neurological examination results.

Laboratory examinations

Laboratory findings (creatinine, blood urea nitrogen, sodium, potassium, blood count, urinalysis, free triiodothyronine, thyroid-stimulating hormone) were unremarkable except for mild hypercholesterolemia.

Imaging examinations

Direct ophthalmoscopy performed in the ER revealed some flame-shaped hemorrhages and hard exudates at the inferior temporal arcade in the right eye and a macular hemorrhage and some flame-shaped bleeding along the temporal arcades in the left eye. Electrocardiography showed sinus rhythm, a heart rate of 90, left ventricular hypertrophy and negative T waves in DI and aVL. Further echocardiography in the ER displayed mild left ventricular hypertrophy, normal systolic and diastolic function, normal wall movement and the absence of hemodynamically significant valvulopathies.

FURTHER DIAGNOSTIC WORK-UP

Suspecting a secondary hypertensive disease, we admitted the patient to the Internal Medicine Department to further seek the etiology and to stabilize her blood pressure. Abdominal echography and Doppler renal artery ultrasonography revealed a left kidney considerably smaller than the right kidney (100/53 mm *vs* 118/50 mm), a left renal sinus with a duplex collecting system, and normal flow in the extraparenchymal renal arteries. Additional computed tomography (CT) angiography revealed an aberrant extrarenal artery originating from the abdominal aorta and supplying the inferior pole of the left kidney (Figure 1). This structure presented stenosis of approximately 50% of the diameter at its origin in the abdominal aorta (Figure 2). To demonstrate whether the newfound renal artery stenosis was responsible for the patient's high blood pressure values, we measured the level of plasma renin. Laboratory tests showed extremely high hyperreninemia (1300 µUI/mL; normal range, 4.4-46.1 µUI/mL), which confirmed our theory.

DIFFERENTIAL DIAGNOSIS

Regarding the differential diagnosis for renovascular hypertension in our patient, we considered the following potential causes of secondary hypertension: (1) Pheochromocytoma: Patients usually present with flushing, headache, tachycardia, and episodic uncontrolled hypertension, symptoms that were not present in our patient. Additionally, the CT scan did not reveal any suprarenal masses; (2) Primary hyperaldosteronism: Patients present with persistent hypokalemia and metabolic alkalosis. Our patient had normal blood potassium levels; (3) Obstructive sleep apnea: This is usually seen in obese males with an increased neck circumference and a history of snoring, which was not applicable in our case; (4) Coarctation of the aorta: Patients usually have a systolic murmur, radio-femoral delay, and upper extremity hypertension, which were not present in our patient. Additionally, the CT scan did not reveal narrowing in the aorta; and (5) Cushing syndrome: This is associated with discriminatory physical features such as moon facies, buffalo hump, proximal myopathy, glucose intolerance, abdominal striae, and central obesity, which our patient did not show at clinical examination.

FINAL DIAGNOSIS

Severe secondary hypertension caused by stenosis of an accessory left renal artery.

TREATMENT

Considering the patient's urgent hypertensive crisis and hypertensive encephalopathy, the patient immediately received intravenous furosemide and oral nifedipine and clonidine before undergoing further investigation for a diagnosis, and her blood pressure dropped to 160/80 mmHg. Regarding renal artery stenosis, its permeability, and the absence of pathological changes in the left and right renal arteries, we decided to adopt conservative management in this case.

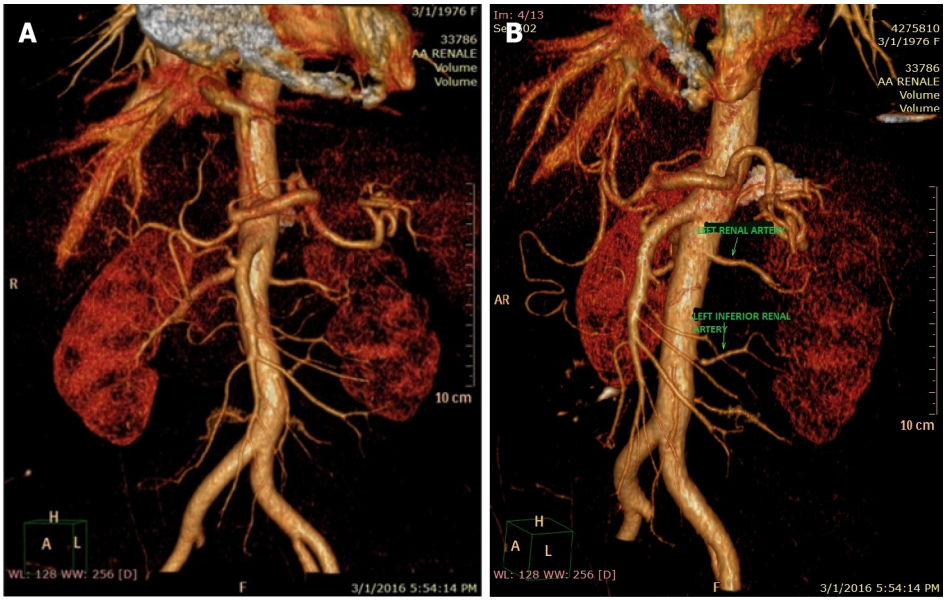

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Figure 1 3D Renal Computed Tomography image showing two left renal arteries. A: 3D reconstruction of the descending aorta; B: Two left renal arteries supplying the left kidney.

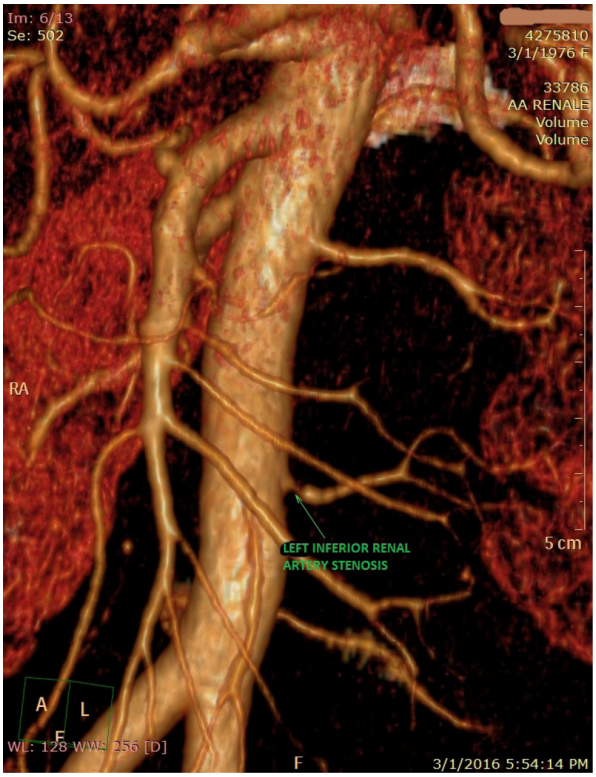

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Figure 2 3D image of renal computed tomography with contrast showing the area of ostial stenosis of the left accessory renal artery.

OUTCOME AND FOLLOW-UP

Consequently, the patient was discharged home with a recommendation for a low-sodium diet and medical treatment, including perindopril 10 mg per day, amlodipine 10 mg per day and indapamide 1.5 mg per day. One month later, she maintained adequate BP control (120/60 mmHg), a normal heart rate (72 bpm) and normal laboratory findings (serum creatinine, blood urea nitrogen, sodium and potassium) without pathological changes.

DISCUSSION

An accessory renal artery is a vestige from the intrauterine development period, with a prevalence ranging from 24% to 42%[2]. In a cadaver dissection study, the incidence of an accessory renal artery was slightly higher in males. Additionally, a higher frequency of these structures was observed on the right side than on the left side, and there was a higher probability of encountering a superior polar artery than an inferior one in cases with a single accessory renal artery[3-4]. Our female patient was born with a rare anatomical variation – an inferior left polar renal artery. The chief pathophysiological mechanism underlying renovascular hypertension involves the hypersecretion of renin, which accelerates the conversion of angiotensin I to angiotensin II and enhances the adrenal release of aldosterone. The result is profound angiotensin-mediated vasoconstriction and aldosterone-induced sodium and water retention. The ensuing cascade of events varies, depending on the presence of a functioning contralateral kidney[5].

When two kidneys are present, aldosterone-mediated sodium and water retention is handled properly by the nonstenotic kidney, precluding volume from contributing to angiotensin II–mediated hypertension. In contrast, a solitary ischemic kidney has little or no capacity for sodium and water excretion, allowing volume to play an additive role in hypertension[6].

When the contralateral kidney is functional, such as in the case of our patient, volume expansion is avoided, and renin levels remain high. The two kidneys are in opposition; the stenotic kidney avidly retains sodium and produces excess renin in response to renal ischemia, while the nonstenotic kidney excretes sodium and water to maintain euvolemia, and the renin production decreases. The end result is systemic hypertension that is mediated by both renin and angiotensin[7].

In this way, the patient presented with secondary hypertension at a very young age.

Not adhering to the treatment was an aggravating factor for her disease, resulting in complications in other organs, such as hypertensive retinopathy. Considering the permeability of the left inferior polar artery, our first therapeutic option was conservative management of the disease to avoid surgery and its possible complications, such as vascular injuries to a functional accessory renal artery, a situation that might result in complications such as kidney failure[8]. During our research, we identified only 6 similar cases. They are summarized in Table 1[9-12].

As might be observed in the Table, like our case, the majority of the patients with secondary hypertension associated with the presence of an accessory renal artery were females under 45 years of age. Additionally, the accessory renal artery was more frequently located on the left side.

Secondary hypertension caused by the presence of accessory renal arteries is a controversial subject in the medical literature because there are studies confirming this association and studies that have found no association[13-15]. Thus, although renal arteries are related to higher blood pressure in middle-aged patients with primary hypertension, a recent retrospective study concluded that an accessory renal artery is an independent risk factor for developing increased blood pressure[16]. Not only did our patient have an accessory artery, but the artery had a 50% stenosis at its origin from the aorta causing a significant hypersecretion of renin, which led to severe and uncontrolled hypertension.

We consider that the difficulty of this case was choosing the optimal therapeutic intervention for our patient – pharmacological treatment vs percutaneous revascularization. Usually, revascularization is reserved for patients with hypertension refractory to antihypertensive medications, progressive worsening of renal function, or a degree of renal artery stenosis greater than 80% to 85%[17,18].

CONCLUSION

An increasing number of studies have proven that accessory renal arteries are common in the general population. This case, along with the few similar cases already published in the medical literature of an accessory renal artery as the cause of secondary hypertension, proves that every clinician should be aware of this potential etiology when treating a patient with secondary hypertension, especially when significant asymmetry in kidney size is seen on ultrasound or other imaging scans. Furthermore, these cases reinforce the necessity for future multicenter studies on this subject.

Table 1 A summary of demographic, radiographic, and clinical information from a review of six previously published cases of secondary hypertension due to accessory renal artery pathology

Ref.	Clinic	Imaging	Treatment	Follow-up
[13]	5-year-old boy with severe hypertension (BP 190/130 mmHg) partially uncontrolled with propranolol, diuretics and spironolactone	Arteriogram: an elongated, nonstenotic aberrant artery arising from the common iliac artery feeding the lower pole of the right kidney	Partial nephrectomy and resection of the aberrant artery at its origin	One month later: BP 120/70 mmHg without any medication
[13]	16-year-old girl with severe hypertension (BP 220/115 mmHg) partially controlled with metoprolol 100 mg/day and hydrochlorothiazide 50 mg/day)	Arteriogram: a nonstenotic aberrant artery arising from the lower aorta supplying the lower pole of the left kidney	Medical treatment with captopril, diuretics and a ß-blocker	Generally, well controlled under medical treatment;Lost to follow-up at 19 years old
[15]	29-year-old patient with hypertension uncontrolled with amlodipine 10 mg and atenolol 50 mg daily (BP 160/100 mmHg)	Digital subtraction angiography: left accessory renal artery entrapped by the diaphragmatic crus with 90% stenosis of the proximal ostial segment	Medical treatment	Close monitoring of the patient's BP and consideration of further invasive and aggressive treatment in case of prolonged uncontrolled hypertension
[12]	21-year-old female with severe hypertension (BP 220/142 mmHg) without relevant previous medical history	Renal magnetic resonance angiography: bilateral accessory renal arteries were seen superior to the main renal arteries; Renal angiography: no stenosis in the main or accessory arteries bilaterally	Medical treatment with spironolactone 75 mg and amlodipine 10 mg daily	BP control achieved with medication
[12]	41-year-old woman with history of hypertension for 3 years partially controlled with amlodipine 5 mg daily (BP 145/100 mmHg)	Renal magnetic resonance angiography: bilateral small accessory left renal arteries supplying the upper pole of the kidney; Renal angiography: no stenosis in the accessory arteries	Medical treatment with spironolactone 50 mg and oral potassium chloride 1.2 mg daily	BP control achieved with medication
[14]	31-year-old female with reported history of elevated blood over the past 7 years (BP 150/100 mmHg)	Renal ultrasound: left accessory renal artery; Renal CT: ostial stenosis of the left accessory renal artery	Medical treatment with amlodipine 10 mg and lisinopril 5 mg	BP control was achieved with lisinopril 10 mg, and amlodipine was discontinued

BMI: Body mass index; BP: Blood pressure; CT: Computed tomography; ER: Emergency room.

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FOOTNOTES

Author contributions: Rusu A and Minca D designed and performed the research; Guluta EC and Minca A analyzed the data and wrote the paper; Calinoiu A and Gheorghita V cared for the patient, designed and analyzed the research, and helped with writing the paper; Minca DG and Negreanu L participated in revising the manuscript; Tomescu L performed imaging studies; all authors have read and approved the final manuscript.

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