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Case Report
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A Case of a Young Man with Severe Hypertension

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Abstract

Introduction: Secondary hypertension has many causes, and they vary by age. Coarctation of the Aorta (CoA) is the leading cause of secondary hypertension in adolescents, but it is easily missed.

Case Report: Here, we report a case of high blood pressure from CoA. The patient was a 17-year-old male who was admitted to our hospital because of his uncontrolled high blood pressure. Because of his typical signs (upper limb blood pressure higher than lower limb blood pressure, coarse murmurs audible in the chest, and weakened femoral artery pulsation), we screened for CoA specifically. This case was diagnosed as CoA based on Computed Tomography Angiography (CTA) of the thoracic aorta, and other differential diagnoses were ruled out. Stent implantation was carried out, and the patient's blood pressure was monitored continuously. Anti-hypertensive drugs were used according to circumstances. Normal blood pressure can be maintained by the administration of a small amount of antihypertensive drugs after surgery.

Conclusion: This report reminds us to pay close attention to the likelihood of CoA and other low-incidence diseases. Physicians should screen for the causes of secondary hypertension according to the patient's typical signs and symptoms to avoid overlooking the diagnosis of CoA. Patients get better treatment to maximize patient benefits.

Keywords: CoA; Secondary hypertension; Adolescents

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Introduction

Hypertension is an important risk factor for cardiovascular diseases and metabolic diseases. The number of adults with raised blood pressure is increasing globally [1]. Hypertension falls into two categories: Primary hypertension and secondary hypertension. Primary hypertension is very rare in adolescents. Secondary hypertension develops as a consequence of other diseases. The causes of secondary hypertension vary with age [2]. The etiologic factors of secondary hypertension in adolescents mostly include renal parenchymal disease, CoA, and monogenic disorders [3,4].

This work covers a patient with CoA who presented typical signs with severe hypertension diagnosed as CTA of the thoracic aorta. His typical signs included upper limb blood pressure higher than lower limb blood pressure, coarse vascular murmurs audible in the chest, and weakened femoral artery pulsation. This case gives us a most important warning, reminding us to screen for CoA in adolescents with typical signs. We want to emphasize the importance of typical signs for evaluation of CoA and to impress the diagnostic approach for secondary hypertension in adolescents.

Case Presentation

The patient was a 17-year-old male who was admitted to our hospital in May 2020 due to uncontrolled hypertension for 6 months and weakness of limbs for 20 days. Six months prior to admission, blood pressure of the patient was found to have increased to 200/120 mmHg during the physical examination. Since then, his blood pressure fluctuated between 180-200/100-120 mmHg. The patient did not present obvious symptoms like headache, dizziness, palpitation, arrhythmia, or hyperhidrosis. He bought some antihypertensive drugs (with unknown names, including diuretics) and kept his blood pressure at around 160/100 mmHg.

Twenty days ago, he was admitted to a local hospital due to limb weakness without other symptoms. His blood potassium was 2.51 mmol/L, brain Magnetic Resonance Imaging (MRI)

Table 1: Results of cortisol rhythm.

	8 am	4 pm	12 pm
ACTH (reference range 5–60 pg/ml)	38.97	23.06	9.92
Cortisol (reference range 8 am: 8.7–22.4 μg/dl 4 am <10 μg/dl)	12.85	7.15	1.13

ACTH: Adrenocorticotropic Hormone

Table 2: Results of aldosterone-to-renin ratio.

	Renin (pg/ml)	Aldosterone (pg/ml)	ARR (reference range 0–40)
Dorsal position	17.55	148.51	8.46
Vertical position	35.54	200.59	5.64

ARR: Aldosterone-to-Renin Ratio

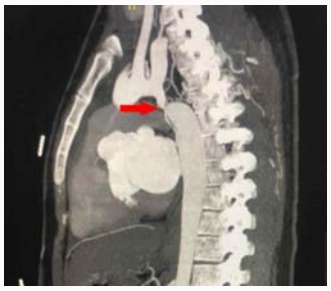

Figure 1: Thoracic aorta CTA shows severe aortic coarctation (arrow). The diameter of the ascending aorta is about 30 mm, and that of the descending aorta about 20 mm. Confined isthmus of the aorta was detected, and the narrowest point was about 3 mm in diameter.

showed cavernous hemangioma, and Magnetic Resonance Angiography (MRA) showed no abnormalities. After discontinuation of diuretic and potassium supplementation, serum potassium was normal. The blood pressure was controlled at about 160/100 mmHg after treatment with spironolactone, amlodipine, and benazepril. To further clarify the cause, the patient was admitted to our hospital. He had a history of hypospadias. His mother had been not exposed to known teratogens such as tobacco, drugs, radiation, or other toxins during her pregnancy. He had no history of smoking or drinking alcohol, and no family history of hypertension or cardiovascular disease.

The patient physical examination showed the following: blood pressure: Left upper limb 163/82 mmHg, left lower limb 108/79 mmHg, right upper limb 176/86 mmHg, and right lower limb 114/80 mmHg. Ankle Braehial index (ABI) was 0.65 on the right side and 0.66 on the left. No murmur was heard in the carotid artery, and no enlargement of the thyroid gland was detected. Upon auscultation, II/VI systolic ejection murmur on the left upper sternal border was heard. Bilateral femoral artery pulsation was weakened. No abnormalities were found in the rest physical examination.

An extensive workup for full blood count, urine test, urea, liver function, thyroid function, systemic immunity tests, and electrolyte levels were normal. Urinary cortisol: 31.62 (normal: 21 μg/24 h to 110 μg/24 h), urinary Vanillylmandelic Acid (VMA): 6.2 (normal: <13.6 mg/24 h), blood catecholamine's, and metabolites were normal. The patient stopped taking the original antihypertensive drugs and changed to terazosin for renin and aldosterone detection 2 weeks later. Further investigations were carried out, and results are shown in Table 1 and Table 2. Bilateral carotid artery ultrasound was normal. Adrenal and renal artery CTA: No abnormalities were observed in bilateral adrenal glands, and no renal artery stenosis was observed. Echocardiography showed left ventricular hypertrophy, aortic

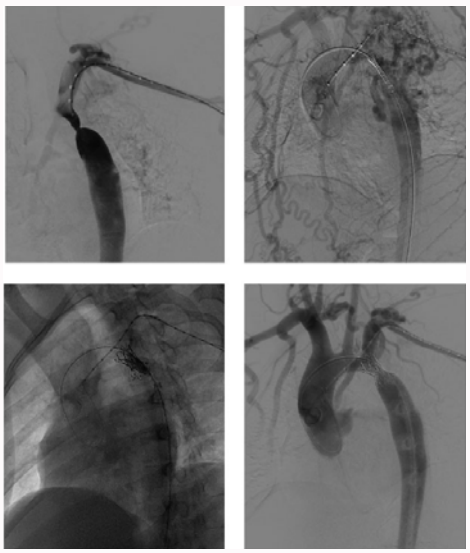

Figure 2: Stent implantation of CoA. A) Cardioangiography shows the severe coarctation (arrow). B, C) Deployment of a stent followed by repeat balloon dilatation. D) The final placement of the stent (arrow).

regurgitation (mild), and tricuspid regurgitation (mild). Thoracic aorta CTA was performed (Figure 1). The patient was diagnosed with CoA with the thoracic aorta CTA. We performed full-exon gene testing using high-throughput sequencing, but found no clear pathogenicity variants associated with disease phenotypes.

He was then transferred to our cardiac surgery department for aortic stent implantation (Figure 2), and he did well after the operation. The patient's blood pressure could be controlled normally with a small dose of beta log.

At the follow-up in December 2020, the patient had discontinued his antihypertensive medications and maintained his blood pressure at 120/80 mmHg.

Discussion

All hypertensive patients must be evaluated for secondary hypertension during their first visit to a hospital, but it is not cost-effective; however, there are some characteristics that may indicate patients more likely to have secondary hypertension. According to 2018 ESC/ESH guidelines for the management of arterial hypertension (4) and clinical experience, screening should be considered in these hypertensive patients: (1) Age at the onset of hypertension <40 years, and lack of other risk factors such as family history and obesity, (2) pre-adolescent children with elevated blood pressure, (3) resistant hypertension (blood pressure >140/90 mmHg after treatment with the best-tolerated doses of three or more drugs, which should include a diuretic, an ACE inhibitor or an ARB, and a CCB), (4) patients with severe hypertension (blood pressure >180/110 mmHg) or a hypertension emergency, (5) target organ damage (such as left ventricular hypertrophy and hypertensive retinopathy), (6) acute worsening hypertension in patients with previously documented chronically stable normotension, (7) clinical features suggestive of obstructive sleep apnea, and (8) symptoms suggestive of pheochromocytoma or family history of pheochromocytoma.

After the patient was admitted to hospital, the cause of hypertension was clarified. His characteristics were as follows: 1. He was an adolescent. 2. Blood pressure was severely elevated, and

the therapeutic effect of antihypertensive drugs was poor. 3. Typical signs were as follows: Upper limb blood pressure was higher than lower limb blood pressure, coarse murmurs audible in the chest, and weakened femoral artery pulsation. 4. He had no history of high-salt diet, obesity, smoking, alcohol consumption, or mental stress.

Typical causes of secondary hypertension vary according to age. In adolescents (12 to 18 years), the usual causes are renal parenchymal disease, CoA, and monogenic disorders. According to the patients' medical history, symptoms, signs, urine tests, blood tests, the adrenal glands CT, and kidney artery CTA, obstructive sleep apnea, renal parenchymal disease, renovascular disease, primary aldosteronism, pheochromocytoma, Cushing's syndrome, thyroid disease, drugs, and single-gene disorders were ruled out. The patient's upper limb blood pressure was higher than the lower limb blood pressure, coarse vascular murmurs could be heard in his chest, and the femoral artery pulsation was weakened. CoA was highly suspected. The CTA of the thoracic aorta was further improved, which confirmed the secondary hypertension caused by narrowing of the main artery isthmus.

CoA accounts for 4% to 8% of all congenital heart defects (5), which can be a simple aortic coarctation or amalgamative other heart malformation, such as ventricular septal defect, aortic valve malformation, or mitral valve malformation [6]. The etiology of CoA can be divided into acquired and congenital, but most of them are congenital. Because the patient was young, CoA and hypospadias were present at the same time, whether there was gene deletion or mutation that caused these two deformities, but unfortunately, the pathogenic gene was not found in full-exon gene testing.

The prognosis of CoA was poor without treatment. Of those surviving the serious hazards of the first two years, 25% die before the age of 20 years, 50% by the age of 32 years, 75% by the age of 46 years, and 90% by the age of 58 years. The arithmetical mean of the ages of death is 34 years. Causes of death include congestive heart failure, aortic rupture, bacterial endocarditis, and intracranial hemorrhage [7].

Adolescents with CoA, unlike adults with CoA [8,9], often have no obvious self-conscious symptoms. Hypertension has been observed during the physical examination, with upper limb blood pressure higher than lower limb pressure, and femoral artery pulsation weakened or disappeared. In this case, CoA is highly suggestive. Echocardiography has high sensitivity and specificity in the diagnosis of CoA. Echocardiography can be used to identify the site of the narrowest point, degree, and length, and the presence of any other cardiac vascular malformation, which is the preferred method of diagnosis [10]. However, in this case, echocardiography did not indicate CoA. The missed diagnosis of CoA by ultrasound is usually caused by neglecting the exploration of suprasternal fossa section. For this reason, the suprasternal fossa section should be taken as a routine probe section, especially for hypertensive patients under the age of 40 years. Cardiac enhanced CT and MRI can clearly show the anatomical structure of CoA [11], which can be used for diagnosis, surgical treatment, and long-term postoperative follow-up [10]. Cardioangiography is the traditional gold standard for evaluating CoA, but it is rarely used for a diagnosis now. Cardioangiography is usually performed in patients with complex intracardiac malformations, and simultaneous stent implantation or balloon dilatation is considered.

Patients with CoA may choose surgical treatment, stenting, or balloon dilation depending on their condition. Surgical data from the STS-CHS database from 2006 to 2010 show that the overall early postoperative mortality rate of CoA was 2.4%, among which the early postoperative mortality rate of CoA was 1% for simple CoA, 2.5% for those with ventricular septal defects, and 4.8% for those with other deformities [12]. Six months after aortic stent implantation, the patient was discontinued from antihypertensive medication and his blood pressure was normal. Long-term follow-up should be conducted after the operation, including resting blood pressure, difference of upper and lower limb blood pressure, 24-h blood pressure monitoring, cardiac ultrasound, cardiac CT, MRI, and cardiac catheterization.

Conclusion

Secondary hypertension is more harmful than primary hypertension. Early identification and early treatment are particularly important. CoA is an extremely important cause of secondary hypertension in adolescents. Screening the causes of hypertension according to the characteristic symptoms and signs can reduce the patient's economic burden, shorten the diagnosis and treatment time, and offer the patient more benefits.

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Author Contributions

LG and FqL wrote the manuscript. LG, FqL, CcF and XlZ performed the medical care of the patient. LJ contributed to the design of the study and the modifying of the manuscript. All authors read and approved the final manuscript.

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