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A Case Study on Severe Hypertension

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Abstract

Secondary hypertension has a wide range of causes, and these causes change with age. The most common cause of secondary hypertension in teenagers is Coarctation of the Aorta (CoA), still it's frequently overlooked. Then, we present a case of elevated blood pressure caused by CoA. The case, a manly case aged 17, was brought into our sanitarium due to unbridled high blood pressure. We especially estimated CoA. On the basis of a Thoracic aorta CTA and the rejection of other possible judgments, CoA was determined to be the opinion in this case. The case had a stent implanted while having their blood pressure continuously checked. Depending on the situation, anti-hypertensive specifics were utilized. After surgery, a little cure of antihypertensive drug might be given to keep blood pressure within normal range. This paper serves as a memorial to keep an eye out for conditions with low prevalence, similar to CoA. According to the case's typical signs and symptoms, croakers should test for secondary hypertension reasons to help missing the opinion of CoA. Cases admit better care to maximize their advantages. The study has main contributions such as- A significant threat factor for metabolic and cardiovascular diseases is hypertension. Primary hypertension and secondary hypertension are the two types of hypertensions. Compared to original hypertension, secondary hypertension is more dangerous. Here in this study, firstly there is an introduction regarding the case study i.e., of hypertension then there is case presentation which includes two tables about the case. Then comes the discussion of the topic which is explained by the figures. And finally, the conclusion shows that secondary hypertension is more dangerous.

Keywords

Secondary hypertension, Adolescents, CoA.

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1. Introduction

A significant threat factor for metabolic and cardiovascular diseases is hypertension. Encyclopedically, there are more grown-ups with high blood pressure[1]. Adolescents with primary hypertension are relatively uncommon. The development of secondary hypertension follows from former ails. Age affects the etiology of secondary hypertension [2]. Renal parenchymal complaint, CoA, and monogenic conditions are the main etiologic causes for secondary hypertension in teenagers.

This study focuses on a case with CoA who displayed typical symptoms and was linked as having CTA of the thoracic aorta due to high hypertension. His typical symptoms included weakened femoral roadway palpitation, coarse vascular murmurs audible in the casket, and upper branch blood pressure that was lesser than lower branch blood pressure. These flaunting common symptoms. We wish to stress the individual system for secondary hypertension in adolescents as well as the significance of typical suggestions for CoA webbing.

2. Case Presentation

A 17-year-old male patient was hospitalized to our hospital in May 2020 as a result of uncontrolled hypertension that had been present for six months and 20 days of limb weakness. During the physical examination, it was discovered that the patient's blood pressure had risen to 200/120 mmHg six months prior to admission.

His blood pressure has been ranging between 180 and 200 and 100 and 120 mmHg ever since. The patient did not exhibit any overt symptoms, such as hyperhidrosis, palpitations, arrhythmia, or headaches. He purchased several unidentified antihypertensive medications, such as diuretics, and an MRA scan produced no aberrant results. Serum potassium levels returned to normal when the use of diuretics and potassium supplements was stopped. Following treatment with spironolactone, amlodipine, and benazepril, the blood pressure was kept under control at about 160/100 mmHg. The patient was admitted to our hospital in order to determine the cause in more detail such as smoke, medications, radiation, or other poisons. He had never smoked or consumed alcohol, and neither did any members of his family have a history of hypertension or cardiovascular disease.

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

2.1. The physical examination of the patient revealed the following

2.1.1. Blood Pressure

Right upper limb 176/86 mmHg, right upper limb 114/80 mmHg, and right lower limb 108/79 mmHg. ABI values for the right and left ankles were 0.65 and 0.66, respectively. The thyroid gland did not grow, and there was no sign of a carotid artery murmur. II/VI systolic ejection murmur on the left upper sternal border was audible on auscultation. Weakened bilateral femoral artery pulsation. The physical examination at rest revealed no anomalies.

Blood catecholamine levels, metabolites, and urinary cortisol levels were all within normal ranges. Urinary Vanillyl mandelic Acid (VMA) levels were also within normal ranges at 6.2 and 21.62 ng/24 h, respectively. Two weeks after stopping the initial antihypertensive medication, the patient switched to terazosin for renin and aldosterone detection. Further research was done, and the findings are displayed.

2.1.2. Both Tables 1 and 2

An ultrasonography of both carotid arteries was normal. CTA of the thoracic aorta was carried out (Figure 1). With the use of the thoracic aorta CTA, the patient's CoA was identified. High-throughput sequencing was used to evaluate the complete exons of the genes; however no obvious pathogenicity variations connected to illness symptoms were discovered.

He was then moved to our cardiac surgery division for the placement of an aortic stent (Figure 2), and he recovered nicely. With a little dose of beta log, the patient's blood pressure may be kept under normal control.

The patient had stopped taking his antihypertensive drugs and was still maintaining a blood pressure of 120/80 mmHg at the follow-up appointment in December 2020.

2.2. Discussion

Even though it is not cost-effective, all hypertensive patients must have their secondary hypertension assessed on their initial hospital visit.

The cause of the patient's hypertension was determined after the patient was admitted to the hospital. His traits included the following.

- He was a teenager.
- Antihypertensive medications had a subpar therapeutic effect, and the blood pressure was markedly increased.
- Typical symptoms included weaker femoral artery pulsation, coarse murmurs audible in the chest, and upper limb blood pressure that was greater than lower limb blood pressure.

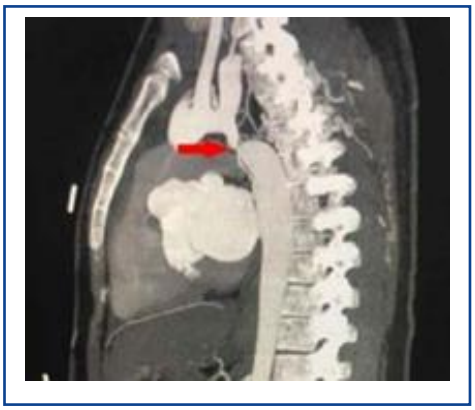

Figure 1. Cardiac angiography shows severe pain (arrow).

For the ascending aorta,
Has a diameter of about 30 mm, and for the descending aorta,
Has a diameter of about 20 mm. The isthmus of aortic stenosis is checked, the narrowest part of which is about 3 mm in diameter.

According to age, several causes of secondary hypertension are typical. The pulse was weaker. CoA was seriously questioned. The thoracic aorta's CTA was further enhanced, which supported the finding that the main artery isthmus narrowing was the primary source of secondary hypertension.

CoA, which can be a straightforward aortic coarctation or an amalgam of various heart malformations including a ventricular septal defect, an aortic valve malformation, or a mitral valve malformation[6], makes up 4% to 8% of all congenital heart problems[5]. The causes of CoA can be either congenital or acquired, however congenital causes account because of their youth, regardless of the gene deletion or mutation that resulted in these two malformations, full-exon gene testing failed to identify the pathogenic gene.

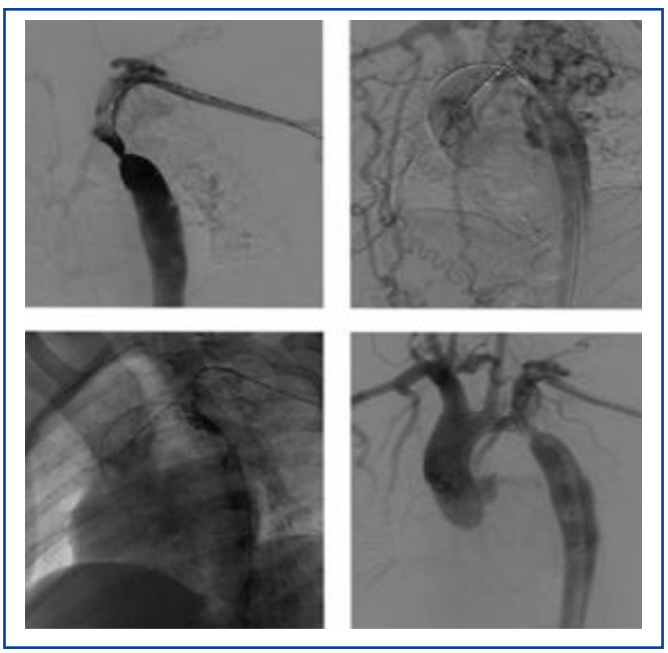

Figure 2. CoA's stent implantation.

A) Cardio angiography shows severe coarctation (arrow). B, C) Delivery of stent and the balloon inflates back. D) The stent's final placement (arrow).

Without treatment, the prognosis for CoA was not good. After surviving the significant risks of the first two years, 25% of survivors pass away before the age of 20, 50% before the age of 32, 75% before the age of 46, and 90% before the age of 58. The mathematical average of Death ages is 34 years. Congestive cardiac failure, aortic rupture, bacterial endocarditis, and cerebral haemorrhage are among the causes of mortality[7].Contrary to adults with CoA[8,9], adolescents with CoA frequently lack overt signs of self-consciousness. Physical examination results revealed hypertension, with upper limb blood pressure higher than lower limb blood pressure and weaker or absent femoral artery pulse. CoA is highly indicative in this situation.

High specificity and sensitivity of echocardiography In the CoA diagnosis. The primary method of diagnosis is echocardiography, which can be utilised to pinpoint the location, degree, and length of the narrowest point as well as the existence of any additional cardiac vascular malformations[10]. However, in this instance, CoA was not detected by echocardiography. The investigation of the suprasternal fossa portion is frequently neglected, which results in the CoA ultrasonography diagnosis being overlooked. This is why the suprasternal fossa section should be performed frequently, especially on hypertension patients who are less than 40 years old.

The original gold standard for assessing CoA is cardioangiography, although it's now hardly utilised to make an opinion. Cases with complicated intracardiac anomalies generally suffer cardio angiography, with concurrent stent placement or balloon dilatation being an option.

Depending on their condition, patients with CoA may opt for surgical therapy, stenting, or balloon dilatation. From 2006 to 2010, according to surgical data from the STS-CHS database, the early postoperative mortality rate for CoA was 2.4% overall, 1% for simple CoA, 5% and 4th percentile of patients with ventricular septal defect and 4.8% of patients with other malformations[12]. The patient stopped using his antihypertensive medication six months after getting an aortic stent, and his blood pressure was back to normal.

After the procedure, there should be a long-term follow-up with measurements of resting blood pressure, the difference between the blood pressure in the upper and lower extremities, 24-hour blood pressure monitoring, cardiac ultrasonography, cardiac CT, MRI, and cardiac catheterization.

3. Conclusions and Future Scope

Compared to original hypertension, secondary hypertension is more dangerous. Particularly crucial are early detection and treatment. Adolescent secondary hypertension has a major contributor: CoA. The financial burden on the patient, the length of the diagnostic and treatment process, and the benefits to the patient can all be decreased.

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Conflict of Interest

Regarding this research, I disclosed no potential conflicts of interest.

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