Journal of
Medical Case Reports

CASE REPORT

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Extreme compensatory dilatation of the splanchnic artery caused by congenital coarctation of the abdominal aorta: a case report

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Abstract

Background Coarctation of the aorta is a congenital cardiovascular anomaly characterized by aortic narrowing, typically presenting in early life. This report describes an exceptionally rare case of severe abdominal aortic coarctation with extensive collateral vessel formation in an asymptomatic 80-year-old male, underscoring the importance of recognizing delayed presentations and compensatory mechanisms. This report describes a case of severe abdominal aortic narrowing and significant compensatory dilatation of visceral arteries in an elderly male, along with a review of pertinent literature.

Case presentation An 80-year-old Han Chinese male with a 10-year history of hypertension controlled with medical management (maximum blood pressure 150/90 mmHg) was incidentally diagnosed with near-occlusion of the upper abdominal aorta during routine physical examination. Computed tomography angiography revealed severe stenosis of the abdominal aorta measuring 7 mm in diameter at its narrowest segment, accompanied by marked compensatory dilatation of the superior mesenteric, inferior mesenteric, and celiac arteries (maximum diameter: 1.4 cm), forming extensive collaterals. Physical examination showed preserved dorsalis pedis pulses and no symptoms of ischemia. Given his asymptomatic status, advanced age, and robust collateral circulation, conservative management with ongoing surveillance was pursued.

Conclusion This case illustrates the potential for advanced coarctation of the aorta to remain asymptomatic in elderly patients owing to compensatory vascular adaptation. It highlights the need for heightened clinical suspicion in patients with hypertension and individualized management strategies balancing risks of intervention against natural history.

Keywords Aortic coarctation, Congenital heart disease, Vascular dilation, Mortality rates, Case report

Introduction

Coarctation of the aorta (CoA) is a cardiovascular anomaly characterized by narrowing or occlusion of the aortic lumen, comprising approximately 6–8% of congenital heart diseases [1]. The diagnosis requires imaging confirmation of luminal narrowing (>50%) and collateral circulation. It can manifest at any age and may be congenital or acquired. Congenital CoA typically occurs near the aortic isthmus and is often associated with abnormal

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aortic arch development. Acquired CoA, however, is commonly caused by aortitis, severe aortic constriction, or traumatic aortic transection, primarily affecting the thoracic and abdominal aorta. According to Bouchart [2], mortality rates among patients with untreated congenital CoA reach 25%, 50%, and 90% by ages 20, 32, and 58 years respectively, with an average life expectancy of approximately 35 years. The exact cause of CoA is not fully understood, but it is believed to involve abnormal development of the fetal aorta during pregnancy. In some cases, there may be a genetic component or association with certain genetic syndromes such as Turner syndrome or bicuspid aortic valve. CoA can lead to significant cardiovascular problems, including hypertension, left ventricular hypertrophy, and even heart failure if untreated [3]. Historically, untreated CoA in severe cases, particularly in infants and young children, could lead to high mortality rates due to complications such as heart failure, hypertension, and endocarditis [4].

Diagnosis of CoA often involves clinical examination, imaging studies, and hemodynamic assessments. Notably, incidental findings on routine imaging, as in this case, may reveal asymptomatic CoA in elderly patients, underscoring the importance of comprehensive evaluation even in the absence of overt symptoms. Echocardiography is particularly useful for evaluating the transcoarctation gradient, detecting coexisting bicuspid aortic valve (present in up to 50% of cases), and assessing left ventricular hypertrophy, a critical marker of disease progression [5].

Treatment strategies are individualized on the basis of age, anatomical characteristics (e.g., type and extent of constriction), and comorbid conditions, ranging from conservative management to surgical or endovascular intervention [5, 6]. Surgical repair involves resecting the narrowed segment and reconnecting the healthy segments of the aorta [7]. In less severe cases or as a bridge to surgery, balloon angioplasty and stenting may be performed to widen the narrowed area and improve blood flow [8]. In this case, we present an 80-year-old patient with an extremely rare CoA. Owing to the absence of any clinical symptoms and excellent compensatory dilation of visceral arteries, conservative treatment offered greater benefits than surgery. Following comprehensive assessment, continued conservative management may be considered.

Case presentation

The patient was an 80-year-old Han Chinese male with no significant clinical symptoms. Abdominal ultrasound revealed abdominal aortic stenosis. For further detailed examination, the patient was admitted to the hospital 3 days later. The patient has a 10-year history of hypertension, managed with oral amlodipine 5 mg daily. Upon admission, the blood pressure measurements were as follows: left upper limb 127/71 mmHg, right upper limb 131/81 mmHg, left lower limb 111/72 mmHg, and right lower limb 113/70 mmHg.

On cardiovascular examination, all peripheral pulses—including carotid, brachial, femoral, popliteal, posterior tibial, and dorsalis pedis—were palpable with good volume (+) and symmetrical on both sides. Precordial examination revealed normal heart sounds (S1 and S2) with no murmurs or gallops. The abdomen was flat, with no visible pulsations, masses, scars, or skin lesions. Examination of other systems was unremarkable.

Serological investigations revealed normal inflammatory markers (C-reactive protein, 2.99 mg/L; erythrocyte sedimentation rate (ESR), 10 mm/hour) and lipid profile (low density lipoprotein (LDL), 0.75 mmol/L). Autoantibodies (e.g., antinuclear antibody (ANA) and antineutrophil cytoplasmic antibody (ANCA)) were negative, excluding vasculitis. Atherosclerosis was excluded owing to absent calcification on imaging. Takayasu arteritis was ruled out by normal inflammatory markers (C-reactive protein (CRP), 2.99 mg/L; ESR, 10 mm/hour) and lipid profile (LDL 0.75 mmol/L), while giant cell arteritis was excluded given the absence of cranial symptoms [1, 5].

Thoracoabdominal computed tomography angiography (CTA) demonstrated atherosclerotic changes in the aorta and branches, with severe stenosis (7 mm lumen) of the upper abdominal aorta (Fig. 1A, B). Compensatory dilatation of the superior mesenteric (1.4 cm), inferior mesenteric, and celiac arteries was noted, forming anastomotic collaterals (Fig. 2). Echocardiography showed no left ventricular hypertrophy or valvular abnormalities. Video 1 and Video 2 show the three-dimensional reconstruction and cross-section of the thoracic and abdominal CTA respectively. Carotid Doppler ultrasound demonstrated no significant stenosis or plaque formation. Brain magnetic resonance imaging (MRI), performed to exclude silent cerebrovascular ischemia, revealed no acute or chronic infarcts. Lower extremity Doppler confirmed adequate perfusion.

Given the patient's asymptomatic status, preserved distal perfusion, and extensive collaterals, surgical or endovascular intervention was deemed high risk owing to potential hemodynamic disruption. Statin therapy was initiated for atherosclerosis management, and biannual clinical/imaging follow-up was recommended.

Discussion

Aortic coarctation is anatomically divided into pre-ductal and post-ductal [9]. In the precatheterization (infantile) type, the constriction is located before the ductus arteriosus and is often extensive, accounting for about 10%

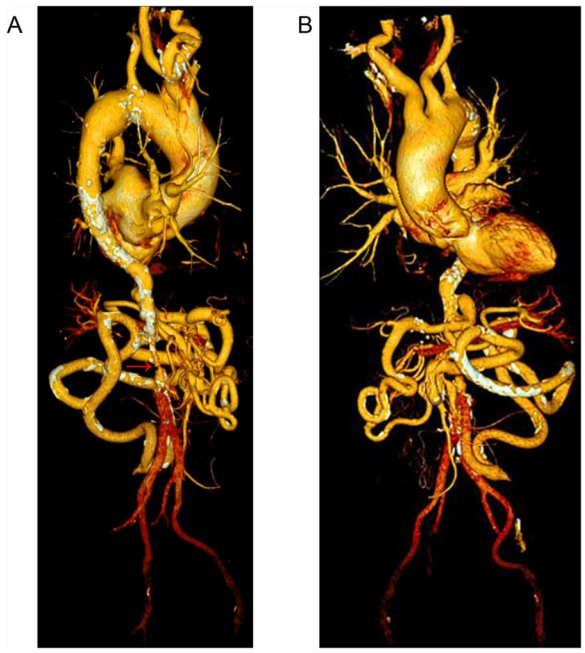

Fig. 1 Thoracoabdominal computed tomography angiography; **A** posterior view showing severe abdominal aortic stenosis (arrow) and dilated visceral arteries; **B** anteroposterior view highlighting collateral anastomoses

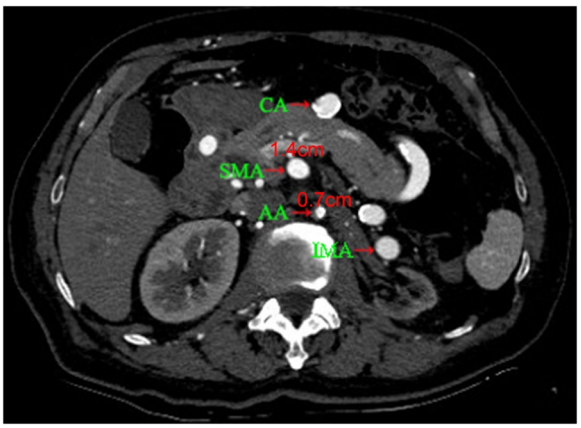

Fig. 2 Axial computed tomography angiography demonstrating near-occlusion of the abdominal aorta (arrow) and compensatory visceral artery dilatation

of cases, often involving the aortic arch and subclavian artery, with an unobstructed ductus arteriosus, and the thoracic and abdominal areas and lower extremities are mainly supplied by the ductus arteriosus, with little collateral circulation [10]. These patients have a poor prognosis and most die in infancy. In the post-ductal type, also called the adult type, the narrowing is distal to the ductus arteriosus or ligament and is often limited in extent, accounting for approximately 90% of cases [10]. The ductus arteriosus is mostly closed, with abundant collateral circulation and fewer combined cardiac malformations.

CoA symptoms vary with severity, age at diagnosis, and associated heart defects. Adults may present with headaches, leg fatigue, cold extremities, poor exercise tolerance, or nosebleeds. In rare cases, it may first manifest with severe complications such as aortic dissection, stroke, or heart failure. Up to 50% of mild cases remain asymptomatic until hypertension or related complications develop. Typical clinical signs include a blood pressure difference between the arms and legs (> 20 mmHg), brachiofemoral delay, and a systolic murmur heard below the left clavicle or between the scapulae. Uniquely, our case was detected at a later age, remained asymptomatic, and showed no typical clinical examination findings [11–13].

The mean age of death in untreated cases is 35 years, and 75% of patients die before 46 years. Leading causes of death are the following: congestive heart failure, bacterial endocarditis, spontaneous aortic aneurysm rupture, and intracranial hemorrhage [5]. Our patient's survival to 80 years likely reflects robust collateralization mitigating end-organ ischemia. Key differentials—atherosclerotic stenosis, Takayasu arteritis, and congenital mid-aortic syndrome—were excluded via imaging and serology [14].

Current guidelines advocate intervention for symptomatic CoA or hypertension refractory to medical therapy [5]. However, asymptomatic elderly patients with stable collaterals may face greater risks from intervention than disease progression. In this case, the decision to conservatively manage aligns with literature emphasizing individualized risk–benefit analysis [15].

With CoA, the choice of treatment depends on the patient's age, type of constriction, extent of constriction, and comorbid malformations [5, 6]. Surgery is the most effective treatment for patients with CoA comorbid cardiovascular malformations, aiming at removing the diseased stenotic segments, re-establishing normal blood flow channels in the aorta, and restoring blood pressure and circulatory function to normal [8]. With the updating of new stent materials such as growth stents, overlay stents, and biodegradable stents and the improvement of the safety of stent implantation, the interventional therapy is increasingly being applied to adolescents and adults. However, in this case, the patient did not have obvious chest tightness, abdominal pain, visceral arteries, and lower limb arterial ischemia manifestations. The following were observed: blood pressure control was good; the dorsalis pedis arteries of both lower limbs were present (+); thoracic and abdominal CTA suggested that the superior mesenteric and inferior mesenteric arteries, abdominal trunks, and their branches were relatively

coarsened; the superior mesenteric artery could be seen to emit two thick branches; pre-take bypass, the maximum diameter of the tube was about 1.4 cm, anastomosing to the celiac trunk and inferior mesenteric artery, respectively, suggesting open and compensatory tortuous dilatation of collateral vessels. If surgical treatment had been performed, either open surgery or minimally invasive intervention, this may have lead to changes in the hemodynamics of the visceral arteries, or even a reduction in blood supply, resulting in visceral artery ischemia. Moreover, the 80-year-old patient may not have been able to withstand the blow of the operation. After multidisciplinary assessment, continued follow-up may be considered. In the present case, the patient was 80 years old, which is exceptionally rare. Owing to the patient's lack of obvious symptoms and the lack of timely physical examination, aortic narrowing, compensatory thickening of the visceral arteries, anastomosis of the superior mesenteric, celiac, and inferior mesenteric arteries, opening of the collateral vessels, and compensatory tortuous dilatation were detected at a late stage.

CoA often combines with other cardiovascular malformations, and some cases of simple CoA may have no obvious symptoms in the early stage, which is easy to be overlooked and missed, and, if left untreated, the patient will usually die of hypertension with complications such as cardiac failure, aortic dissection, and intracranial hemorrhage. Clinically, patients diagnosed with CoA should be examined more systematically and comprehensively to avoid missing the diagnosis of cardiac and other systemic comorbidities, so as to improve the prognosis and reduce the morbidity and mortality rate.

Limitations

The absence of long-term follow-up data precludes definitive conclusions about natural history. In addition, genetic testing was not performed to exclude syndromic associations.

Clinical messages

1. CoA should be considered in elderly patients with hypertension with unexplained vascular findings.
2. Compensatory collaterals may delay diagnosis and symptom onset.
3. Conservative management is viable in select asymptomatic cases.
4. Multimodal imaging is critical for anatomical and hemodynamic assessment.
5. Age and collateral adequacy are pivotal in therapeutic decision-making.

Conclusion

This case highlights the clinical significance of severe abdominal aortic narrowing and compensatory dilatation of visceral arteries in an elderly male patient. The findings underscore the importance of early diagnosis and appropriate management strategies in such cases to optimize patient outcomes and reduce associated morbidity and mortality. Further studies are warranted to explore additional management options and long-term outcomes in similar patients.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13256-025-05282-3.

Supplementary Material 1: Video 1. Chest and abdominal CTA reconstruction video.

Supplementary Material 2: Video 2. Thoracic and abdominal CTA cross-section video.

Acknowledgements

Not applicable.

Author contributions

All authors contributed to the conception, writing, and editing of the case report. All authors agree to be accountable for all aspects of it.

Funding

This work was funded by the Natural Science Foundation of Jiangxi Province (20232BAB216010 and 20242BAB25433 to Zhiwei Zhong), Youth Fund of Education Department of Jiangxi Province (GJJ210251 to Zhiwei Zhong), National Natural Incubation Program of the Second Affiliated Hospital of Nanchang University (2022YNFY12013 to Zhiwei Zhong), and Jiangxi Province Health Science and Technology Plan Project (202510325 to Zhiwei Zhong).

Availability of data and materials

The authors of this manuscript are willing to provide any additional information regarding the case report.

Declarations

Ethics approval and consent to participate

Written informed consent was obtained from the patient for publication of the details of her medical case and any accompanying images. The research was conducted ethically in accordance with the World Medical Association Declaration of Helsinki.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests

No potential conflict of interest relevant to this article was reported.

Received: 12 August 2024 Accepted: 28 April 2025
Published online: 12 May 2025

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