A multimodality approach to an elderly patient with aortic coarctation, patent ductus arteriosus, and bicuspid aortic valve

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A 66-year-old man was admitted to the hospital due to worsening of exercise tolerance, exertional dyspnea, and occasional unspecific chest pain. A medical history revealed coarctation of the aorta (CoA) and bicuspid aortic valve (BAV) diagnosed about 5 years earlier. Additionally, the patient suffered from multidrug hypertension, type 2 diabetes, hypothyroidism, and permanent atrial fibrillation. Auscultation on admission revealed loud systolic-diastolic murmur over the aortic valve and between the scapulas. Blood pressure was 135/70 mmHg. An electrocardiogram revealed atrial fibrillation and left anterior hemiblock. Laboratory parameters were within the reference ranges, except for slightly elevated troponin and brain natriuretic peptide levels. Transthoracic echocardiography showed the enlargement of the left atrium (59 mm), left and right ventricles (56 mm and 36 mm, respectively), as well as hypertrophy of the posterior wall (14 mm) and of the interventricular septum (14 mm). The aortic valve was bicuspid (FIGURE 1A) with calcification and a maximum gradient of about 30 mmHg and mild aortic regurgitation. Additionally, elevated right ventricular systolic pressure (80 mmHg), pericardial effusion (max. to 7 mm) in the diastole, small patent ductus arteriosus (PDA), and CoA with a systolic pressure gradient of about 81 mmHg were found. PDA was not described in previous transthoracic echocardiography. Ejection fraction was preserved.

The above anatomic findings were confirmed by cardiac magnetic resonance (CMR) imaging, which revealed CoA, PDA, and BAV (FIGURE 1B–1D). A 64-slice computed tomography confirmed the presence of PDA and the narrowing of the aorta (about 5-mm long) below the level of the left subclavian artery (a minimum lumen of 13 mm), with tortuous course (FIGURE 1E). Coronary angiography revealed 3-vessel disease with severe stenosis of the left anterior descending artery, left circumflex artery, and right coronary artery.

The patient did not agree to a cardiac surgery. Due to severity of the CoA and characteristics of the patient, he was referred for percutaneous coronary intervention (PCI; full revascularization) and endovascular dilation and stenting of the aorta. He consented only to PCI, and full revascularization with stent implantation was successfully performed. The patient is still alive.

Although PDA is frequently found in association with CoA, the presence of BAV in this situation is rare. Usually, we expect aortic stenosis due to degeneration or calcification as comorbidity in elderly patients. To our knowledge, coexistence of CoA with PDA and BAV in elderly patients has not been reported yet. There have been several case reports presenting coexistence of CoA with PDA or BAV with CoA in neonates or in young adults, sometimes with different congenital abnormalities. A common genetic background for these abnormalities had been proposed, but our patient did not undergo genetic screening because of his age and had no cardiac abnormalities in family history.

We concluded that transthoracic echocardiography and multislice computed tomography are useful for the assessment of CoA, PDA, and BAV, and CMR imaging may be used to confirm diagnosis. Echocardiography is an ideal initial imaging tool because it is simple, noninvasive, widely available, and cost-effective and reveals the anatomy, location, and hemodynamic consequences of congenital heart diseases. CMR is a complementary imaging technique that carries no
risk of radiation and provides detailed information on the anatomy and tissue characterization.

Our case shows a constellation of uncommon complex congenital cardiac anomalies revealed by a multimodality approach in adulthood (in a 65-year-old patient).

REFERENCES


