



Endovascular repair of severe aortic coarctation, transcatheter aortic valve replacement for severe aortic stenosis, and percutaneous coronary intervention in an elderly patient with long term follow-up

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To the best of our knowledge, there have not been any reports of total transcatheter approach including stenting of severe coarctation of the aorta (CoA), transcatheter aortic valve replacement (TAVR) for concomitant severe aortic valve stenosis, and percutaneous coronary intervention (PCI) to treat significant coronary artery disease in a single patient. We report a 70-year-old female, who presented with uncontrolled hypertension and acute decompensated heart failure (ADHF) and was found to have severe CoA, severe bicuspid aortic valve (BAV) stenosis, and significant proximal left anterior descending (LAD) coronary artery disease. In a multidisciplinary heart team meeting, we decided to perform an endovascular repair of both cardiac and vascular pathologies using a two-stage approach due to the significant comorbidities; mainly uncontrolled hypertension, type 2 diabetes mellitus, chronic obstructive pulmonary disease, and severe calcifications of the ascending aorta. The procedures were successfully performed and the patient was asymptomatic 30 months later at follow-up and was without any significant gradients across the coarctation or the aortic valve.

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Introduction

Coarctation of the aorta (CoA) is a circumscribed narrowing of the aorta located at the site of ductus arteriosus insertion distal to

the left subclavian artery (LSA). It is a relatively common congenital anomaly which accounts for approximately 5% to 8% of all congenital cardiovascular defects [1]. The clinical presentation

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varies from hypertension to symptoms of congestive heart failure (CHF) according to the time of presentation. The presence of refractory hypertension or significant CHF indicates the need for intervention. The available strategies of treatment include surgery and endovascular balloon dilatation with or without stent placement [1,2]. To the best of our knowledge, there have not been any reports of total transcatheter approach, stenting severe CoA, transcatheter aortic valve replacement (TAVR) for concomitant severe aortic valve stenosis, and percutaneous coronary intervention (PCI) to treat significant coronary artery disease (CAD).

Case report

History

A 70-year-old female with a 50-year history of uncontrolled hypertension despite a combination of antihypertensive agents including amlodipine 10 mg, spironolactone 25 mg, losartan 100 mg, and furosemide 40 mg, all used once daily. The patient was referred to our center as a case of acute decompensated heart failure (ADHF) for further workup. On admission, the patient was in severe respiratory distress consistent with the New York Heart Association (NYHA) class III–IV and was orthopneic but fully conscious with normal cognition. Physical examination showed a blood pressure of 153/93 mmHg, a heart rate of 117 beats per minute, a respiratory rate of 28 breaths per minute, and oxygen saturation of 94% on bi-level positive airway pressure ventilation. She had bilateral basal crepitation, bilateral scattered rhonchi, weak delayed femoral pulses, bilateral lower limb edema, raised jugular venous pressure, and loud second heart sound with ejection systolic murmur at the aortic area. Routine blood chemistry and urine analysis were unremarkable.

A chest X-ray revealed cardiomegaly, rib notching, and bilateral pulmonary congestion. Baseline electrocardiography showed left ventricular hypertrophy with a strain pattern. Transthoracic echocardiography showed left ventricular hypertrophy, basal infero-posterior, mid, and basal anterior wall hypokinesia with an ejection fraction of 30%, mild mitral regurgitation, severe aortic stenosis with peak pressure gradient of 118 mmHg and mean pressure gradient of 76 mmHg, and an estimated systolic pulmonary artery pressure of 50 mmHg.

A computed tomography angiogram (CTA) clearly diagnosed severe stenotic calcified concentric 11 mm long aortic coarctation located 16 mm

distal to the LSA (Fig. 1A). The diameter of the narrowest part was approximately 3 mm with significant prevertebral and intercostal collaterals. There was bicuspid aortic valve (BAV) type 1 with severe asymmetric diffuse leaflet calcifications, and marked calcifications of the ascending aorta and aortic arch.

Aortography confirmed the diagnosis of severe calcified juxtaductal CoA. There was a 70-mmHg peak to peak gradient across the coarctation, a 90 mmHg gradient across the aortic valve, and the mean left ventricular pressure was 290/26 mmHg. Coronary evaluation demonstrated a significant proximal left anterior descending (LAD) lesion with a fractional flow reserve (FFR) value of 0.78 (Fig. 1B). A significant dilatation of the left internal mammary artery was also noted.

The presence of a high gradient across the coarctation together with the presence of hypertension necessitates a curative treatment. A multidisciplinary heart team of interventional cardiologists, cardiac anesthesiologists, and vascular and cardiac surgeons decided to perform endovascular repair of both cardiac and vascular pathologies using a two-stage approach due to the significant comorbidities, mainly uncontrolled hypertension, type 2 diabetes mellitus, chronic obstructive pulmonary disease, and also the presence of severe calcifications of the ascending aorta.

Procedure

The first stage was performed under conscious sedation with a surgical team on standby. The right common femoral artery (CFA) was chosen as the main access. The stenosis was crossed safely; aortography in anteroposterior (AP) and lateral projections confirmed the critical stenosis (Fig. 1C). A 14F sheath was advanced across the coarctation segment and direct implantation of a covered Cheatham-platinum stent (CP Numed, Canada) sized 39 mm and mounted on Cristal balloon sized 20/40 mm was accomplished. There were good wall apposition and brisk forward flow with no residual gradient across the coarctation segment (Fig. 1D).

One week later and under conscious sedation, TAVR was performed through right CFA access. We put access in the contralateral side as a vascular access bailout. A 14F e-sheath was used for valve deployment. We used a balloon expandable valve (SAPIEN 3 Edwards Life Sciences Inc., Irvine, CA, USA). Valve sizing was conducted according to the computed tomography (CT) measurements that showed an annulus area of

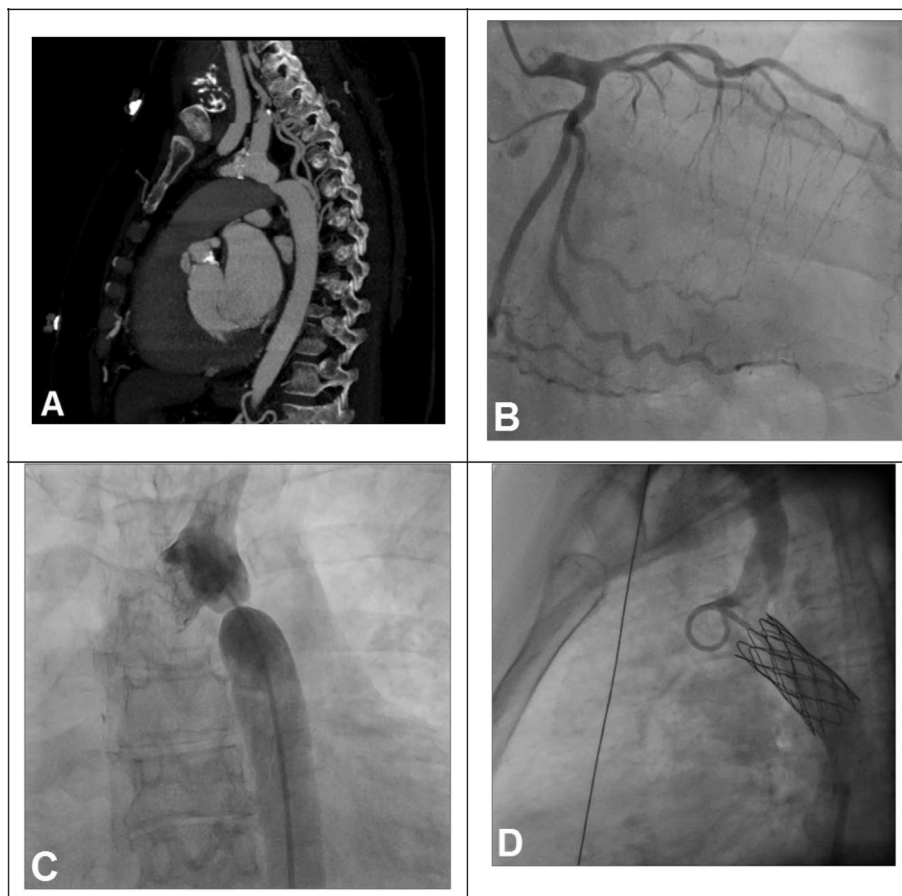


Figure 1. (A) Computed tomography angiogram sagittal view showing severe CoA distal to the left subclavian artery; (B) proximal LAD significant stenosis with an FFR value of 0.78; (C) lateral projection aortography showing severe CoA, shelf-like with subtotal obstruction and a good landing segment after the left subclavian artery; (D) cheatham-platinum covered stent is deployed in the CoA through a 14F sheath. CoA = coarctation of the aorta; FFR = fractional flow reserve; LAD = left anterior descending.

425 mm², coronary origin height of 12 mm and 14 mm for the left and right coronary arteries respectively. The valve sizing chart showed –3% for 23-mm valve versus +23% for 26-mm valve, and as the patient had a high risk of annular rupture because of heavy calcifications involving the bicuspid valve leaflets, annulus, ascending aorta, and sinutubular junction, we chose the smaller valve. The valve assembly was prepared according to the protocol and aligned to the annulus in its coplanar projection (Fig. 2A). After valve assessment, the left main artery was cannulated, LAD was wired, and direct stenting to the proximal LAD was performed (Fig. 2B and C). Unfortunately, an edge dissection needed another distal stent with good final results (Fig. 2C). The groin was controlled using Proglides in the right CFA and angioseal in the left CFA.

Follow up

The postprocedural course was uneventful. The patient was in sinus rhythm, normal intraventric-

ular conduction, and there were no neurological complications. A chest X-ray showed both the aortic valve and the CoA stent in place (Fig. 2D). Following the intervention, the patient has been asymptomatic, fully ambulated on the 2nd day after the procedure with a well-controlled blood pressure; mean of 133/69 mmHg, with amlodipine 5 mg per day, bisoprolol fumarate 5 mg per day, and olmesartan 20 mg per day. The patient was safely discharged on the 3rd day after the procedure. At 30 months follow up, the patient was asymptomatic and echocardiography showed stable aortic valve with normal function and no gradient across the coarctation stent.

Discussion

CoA is a common congenital heart anomaly that frequently affects males more than females and usually manifests in childhood. In adults, CoA is uncommon and most commonly represents cases of recoarctation after surgical repair or rarely

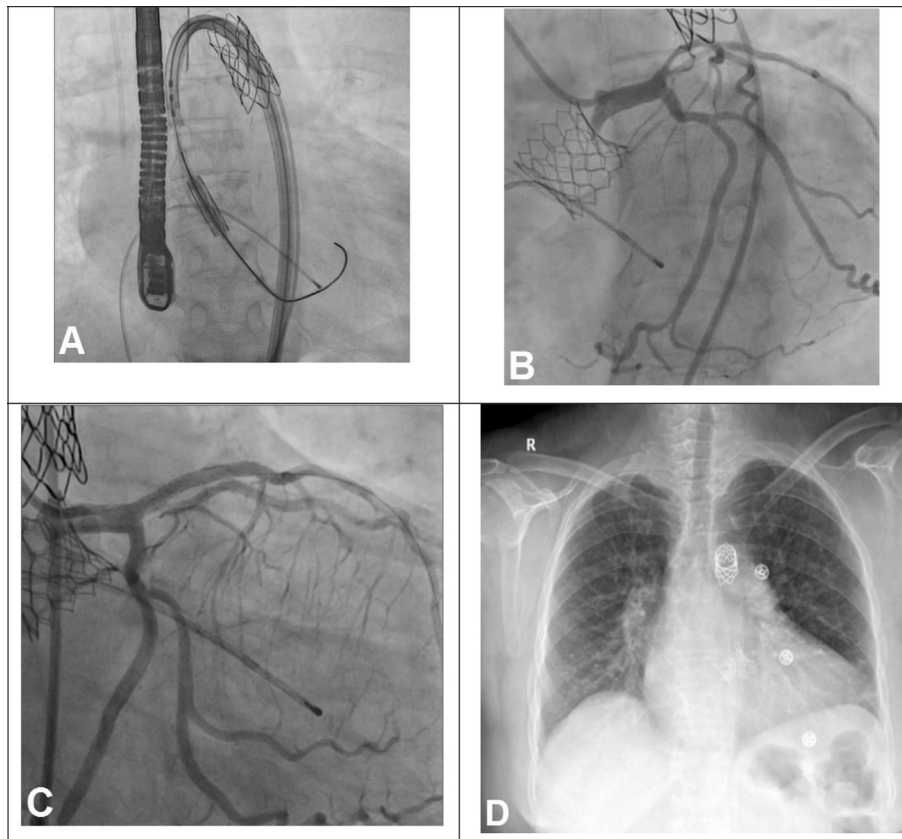


Figure 2. (A) The valve assembly crossed the CoA stent and delivered across the valve before final deployment; (B) coronary angiography before stenting with both a TAVR valve and a CoA stent; (C) final coronary angiography showing the proper positioning of the LAD stents, the TAVR valve, and a CoA stent; (D) chest x-ray in posteroanterior view showing both the aortic valve and the CoA stent in place. CoA = coarctation of the aorta; LAD = left anterior descending; TAVR = transcatheter aortic valve replacement.

missed native coarctation [3]. The natural history of untreated CoA is the development of refractory hypertension, dissection/rupture of the aorta, aortic endarteritis, premature CAD and CHF. The life expectancy of patients with untreated CoA is 35 years, with a mortality rate ranging from 75% to 90% by the age of 50 years [4,5]. CoA in adulthood is usually associated with other lesions including BAV, subaortic stenosis, mitral valve pathologies, and intracranial aneurysm [6]. BAV is a congenital anomaly which occurs in approximately 1% to 2% of the general population and >50% of resected aortic valves during surgical aortic valve replacement procedure have been found to be bicuspid [7]. Over 33% of patients with BAV will develop complications, with some studies suggesting that the morbidity and mortality rates associated with BAV are greater than that of all other congenital heart defects combined [8].

Our patient was a 70-year-old female who presented with ADHF. She had a long history of uncontrolled hypertension despite optimal anti-hypertensive medications. CTA was sufficient for

the diagnosis of severe CoA located 16 mm distal to LSA and calcified BAV with severe stenosis. The current guidelines recommend interventional repair when the peak-to-peak coarctation gradient is ≥ 20 mmHg, or when peak-to-peak coarctation gradient is < 20 mmHg with the presence of anatomic imaging evidence of significant coarctation, and with radiological evidence of significant collateral flow [5]. Because our patient was hypertensive with signs of ADHF, a curative intervention was indicated.

Generally, an invasive surgical approach carries higher risks especially in elderly patients with comorbidities. Recently, minimally invasive strategies are considered to be therapeutic options in high risk patients [8,9]. In our patient, the coexistence of critical CoA, severe BAV stenosis, and significant LAD lesion, in addition to her advanced age, chronic obstructive pulmonary disease, and severely calcified aortic wall made her of high surgical risk. However, the vessel wall is less compliant in elderly people and the risk of restenosis becomes higher, therefore, we aimed to

perform an endovascular stenting of the CoA with covered Cheatham-platinum (CP) stent; initially to avoid rupture of the calcified segment and subsequently to avoid aneurysmal formation and to prolong vessel patency [10].

Bartoccioni et al. [11], reported a similar case of a 55-year-old woman with CoA associated with aortic valve stenosis and ischemic cardiomyopathy who was managed by a single-stage surgical approach of these pathologies. Another case was described by Novosel et al. [3] of an elderly woman with aortic coarctation combined with aortic valve stenosis and mitral regurgitation who underwent a two-stage approach that combined a surgical correction of intracardiac pathologies in the first stage, followed by repair of the aortic coarctation by percutaneous placement of an Advanta V12 large-diameter stent graft.

Conclusion

To the best of our knowledge and after reviewing the literature, this is the first report of total endovascular repair of severe CoA associated with severe BAV treated by TAVR and critical proximal LAD stenosis treated by PCI in a high risk elderly patient. Although this technique had excellent procedural results with accepted short, intermediate, and long term outcomes; further studies are needed to evaluate the effect of CAD on the long-term outcomes after TAVR and the optimum timing of performing PCI in this group of patients.

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