

Late diagnosis of congenital aortic coarctation associated with CAD in adults

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Introduction

Coarctation of the aorta accounts for 5–8% of congenital heart disease^{1,2} and is treated usually in childhood. It is not rare for aortic coarctation to be associated with coronary artery disease³ in patients past 35years of age. Some patients with aortic coarctation remain asymptomatic up to 30–40years of age when the occurrence of coronary artery disease increases. When both pathologies coexist surgical management becomes particularly difficult.

This is a 33years male patient with a history of HTN since early adulthood and recent onset type II DM.

He presented to his local area hospital with a new onset exertional retro-sternal chest pain radiating to the back and left shoulder, and relieved by rest. Accordingly he was referred to a cardiologist who initially treated him medically, and tried to control his high B.P, which was quite high at that time, however despite being compliant on 3 antihypertensive medications, he continued to suffer from these pains, that significantly limited his activities, and his BP remained uncontrolled.

Finally he was admitted with a severe chest pain at rest on 11/12/2012, without significant ECG changes, but with marked Troponin elevation, and he was diagnosed as NSTEMI. Because of recurrent chest pains, he was transferred to our institute for coronary angiography, which was done on 15/1/2013 through the Rt radial approach, which is the standard approach in our cath lab, however the, 0.035 guide wire went through a strange tortuous curve from the Rt SCA. We tried to use an RJ4 diagnostic catheter to manipulate the guide wire into the ascending AO, but it didn't work. So a small hand injection was done in this tortuous vessel after the catheter was advanced downward and it showed a large tortuous vessel that came off the huge RIMA.

AT that moment we suspected the presence of coarctation of AO, and we decided to go through the femoral approach and to introduce a pig tail catheter, which was advanced up to the thoracic descending AO but the guide wire could not be advanced further, so the wire was withdrawn and 50 cc of iso-osmolar contrast was injected, and revealed a severe discrete post Lt subclavian artery COA, with failure of opacification of the the arch and descending AO and despite several trials using a terumo hydrophilic, and PTCA floppy wires, we failed to cross the tight discrete COA narrowing.

His ECHO showed a normal LV size and function without hypertrophy, and the Descending thoracic AO showed a tight COA distal to Left SCA, with a PG of 35mmHg and diastolic tail. Finally we decided to do CT angiography to confirm the diagnosis of coarctation, and also to assess the coronary arteries, and it showed a severe discrete post-SCA COA and severe 3 vessel CAD in the form of totally occluded LAD, bifurcating with a big D1 which showed a severe ostial lesion, moderate OM1 lesion and tight mid RCA lesion.

We had a long discussion about the management plan of this patient, as being a diabetic patient with 3 vessel disease, we know that there is an accumulating data favoring the long term results of CABG Vs PCI in such patients, but the presence of COA in our case complicates the decision, and finally we decided to do staged percutaneous approach starting first with fixing his coronaries followed by COA stenting in a later session.

Coronary angiography was done on 3/3/2013, through left radial approach, due to straighter course towards the ascending AO., confirmed the CT findings, and he had 2 DES, for LAD and D1 using crush technique, and another 1 DES for the RCA, and he was discharged after a couple of days.

In the next follow up visit, he reported great improvement, so the next step was to treat his COA which was done under G.A on 31/3/2013. Angiography showed a tight COA with a diameter of 4mm, and a peak to peak gradient of 60mmHg. During rapid ventricular pacing, the COA segment was stented using a 15*35mm CP covered stent with resolving of the gradient between ascending and descending AO.

Discussion

Patients with congenital Aortic coarctation and associated coronary artery disease pose unique therapeutic challenges and there are few reports in the literature addressing this issue. In our case, the initial question was whether to proceed with surgical repair of COA, and CABG, or a percutaneous stenting approach for both, or a hybrid approach of COA stenting followed by CABG. A single stage surgical repair of aortic coarctation and coronary artery bypass grafting has been reported in the literature⁴ however, this approach might expose the patient for higher risk. The most feared complication of aortic surgery is paraplegia and risk of spinal cord injury. The risk of these complications increases with prolonged aortic cross-clamp time. Other surgical difficulties include need for extensive mobilization of the aorta, control of collateral blood vessels, and damage to the recurrent laryngeal or phrenic nerves.⁵ A repair of aortic coarctation through a left thoracotomy after coronary artery bypass grafting can reduce the risk of complications in patients with late presentation of coarctation.

Another dilemma is conventional CABG under cardiopulmonary bypass through a median sternotomy, as the associated coarctation may make cardiopulmonary bypass difficult, especially in a patient with pre-subclavian coarctation. Even though cardiopulmonary bypass has been used in an adult with untreated coarctation,⁶ important questions need to be addressed regarding the site of placement of the arterial return cannula.

However despite these concerns there some case reports in the literature. One of these reports is a series of 3 patients, with a mean age of 60 years, 2 were males and one female patient, but because the primary problem in all 3 patients was angina, and 2 patients had atypical forms of aortic obstruction, the first patient had a near interruption of the pre-subclavian transverse arch, and the third patient had a long-segment aortic obstruction, possibly due to aortitis where stents have not been documented to be safe in these settings, they decided to tackle the coronary artery disease first with off pump CABG as none of the three patients had a coronary anatomy that was suitable for angioplasty.

Given the circumstances, and despite the high risk of the procedure was remarkably well tolerated, with surprisingly little morbidity. Also there is one case report of staged surgical approach for a 37 years patient, who had CABG first followed by repair of coarctation after 8 weeks, although the procedure was uneventful, the patient was exposed to 2 major procedure in less than 2 months, making it difficult to be applied on a large scale, especially in older patients.⁷

One of the options we considered in our patient was to repair the coarctation first and then do the CABG in the usual manner. However, in the setting of unstable angina with critical coronary blocks, intra-operative ischemia was a real danger. Hence, this approach was not favored as we felt that in untreated coarctation, it is not advisable to sacrifice the internal mammary artery, which is an important source of collateral flow to the distal aorta. Also during the subsequent coarctation repair, clamping of the left subclavian artery would be hazardous with a left internal mammary artery graft in place.

Aortic coarctation (CoA) is reported to predispose to coronary artery disease (CAD). However, our clinical observations do not support this premise. Our objectives were to describe the prevalence of CAD among adults with CoA and to determine whether CoA is an independent predictor of CAD or premature CAD.

Methods and results

The study population was derived from the Quebec Congenital Heart Disease Database. We compared patients with CoA and those with a ventricular septal defect, who are not known to be at increased risk of CAD. The prevalence of CAD in patients with CoA compared with those with ventricular septal defect was determined. We then used a nested case-control design to determine whether CoA independently predicted for the development of CAD. Of 756 patients with CoA who were alive in 2005, 37 had a history of CAD compared with 224

of 6481 patients with ventricular septal defect (4.9% versus 3.5%; $P=0.04$). Male sex (odds ratio OR, 2.13; 95% confidence interval CI, 1.62-2.80), hypertension (OR, 1.95; 95% CI, 1.44-2.64), diabetes mellitus (OR, 1.68; 95% CI, 1.09-2.58), age (OR per 10-year increase, 2.28; 95% CI, 2.09-2.48), and hyperlipidemia (OR, 11.58; 95% CI, 5.75-23.3) all independently predicted for the development of CAD. CoA did not independently predict for the development of CAD (OR, 1.04; 95% CI, 0.68-1.57) or premature CAD (OR for CoA versus ventricular septal defect, 1.44; 95% CI, 0.79-2.64) after adjustment for other factors.

Conclusion

Although traditional cardiovascular risk factors independently predicted for the development of CAD, the diagnosis of CoA alone did not. Our findings suggest that cardiovascular outcomes of these patients may be improved with tight risk factor control.

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Conflicts of interest

Author declares there are no conflicts of interest.

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