Cardiology in the Young

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Multiple myocardial bridges causing severe ischaemia in adolescent with pulmonary stenosis

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Brief Report

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Abstract

Myocardial bridges are often asymptomatic but may need therapy when causing ischaemia. They have rarely been reported in children or in association with CHD, where symptomatology may be mistakenly attributed to the CHD. We report a case of multiple myocardial bridges causing ischaemia in an adolescent with pulmonary stenosis and discuss management.

Case presentation

A 15-year-old girl with pulmonary stenosis after balloon dilation at 14 months of age developed recurrent chest pain on exertion, initially attributed to her mild residual pulmonary stenosis (gradient maximal 40, mean 23 mmHg) on echocardiogram. On cardiopulmonary exercise test, she had chest pain with ST depression in leads V3-V6 and decreased maximal oxygen consumption at 26 ml/kg/min (39 ml/kg/min 3 years earlier). Nuclear scintigraphy showed limited ischaemia of the anterior and apical left ventricular wall on exercise, while cardiac catheterisation showed mild residual pulmonary stenosis with 35 mmHg peak-to-peak gradient. Coronary angiography revealed a complex network of intramyocardial arteries in the mid interventricular septum perfusing the left anterior descending and its branches. Multiple myocardial bridges with severe systolic compression were observed (Fig 1), while the apical portion of the left anterior descending was also supplied by a mature collateral from the dominant right coronary artery (Fig 2). The patient improved with β-blocker therapy with atenolol 50 mg twice daily and low-dose aspirin 100 mg once daily and was followed in clinic twice a year. She remains stable and without symptoms for 4 years, developing asymptomatic ST changes on exercise test only for heart rates over 140/min.

Discussion

Myocardial bridges, coronary artery segments coursing into the myocardium, can be asymptomatic variants but may need therapy when causing ischaemia.[1] They almost exclusively affect the left anterior descending coronary artery and have rarely been reported in children[2] or in association with CHD.[2,3]

Ischaemia related to myocardial bridges may be due to direct compression during cardiac systole as well as endothelial injury of the vessel proximal to the myocardial bridge due to abnormal haemodynamics resulting in coronary atherosclerosis and stenosis.[1] The latter is probably the reason that myocardial bridges often become symptomatic later in life despite their presence since birth. Exercise-induced ischaemia is due to tachycardia, increasing myocardial oxygen requirement and decreasing diastolic coronary flow.[4] Myocardial bridges have been reported in myocardial diseases such as dilated, noncompaction[5] and more commonly hypertrophic cardiomyopathy.[6] Although most patients with myocardial bridges remain asymptomatic, symptoms like angina, exertional chest pain, or dyspnoea may develop,[7] and ventricular arrhythmia, myocardial infarction, syncope, and sudden death have been reported.[1]

Myocardial bridges in children have been described usually in the presence of ventricular hypertrophy and hypertrophic cardiomyopathy, while ventricular fibrillation, syncope, and cardiac arrest have been reported as presenting symptoms more often in children.[2,8] Myocardial bridging in association with structural CHD has been reported only in a neonate with coarctation, ventricular septal defect, and parachute mitral valve[2] and an adult with transposition of the great arteries after Mustard procedure.[3] Our case and these two reports prove that myocardial bridges can exist in patients with CHD and may account for their symptomatology. This fact is important as symptoms of myocardial bridges may be mistakenly attributed to residual CHD thus delaying diagnosis and, also, because young patients with significant structural heart disease and dysfunction may be more susceptible to life-threatening complications of myocardial bridges, such as arrhythmias and sudden death.

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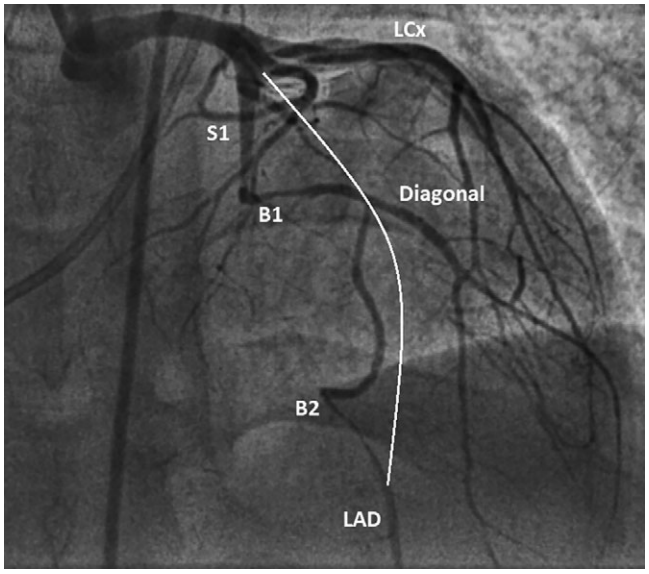

Figure 1. Left coronary artery angiogram in right anterior oblique projection with cranial angulation. The white line represents the normal left anterior descending course. B1: bridge 1, B2: bridge 2, diagonal: first diagonal artery, LAD: left anterior descending artery, LCx: left circumflex artery, S1: first septal.

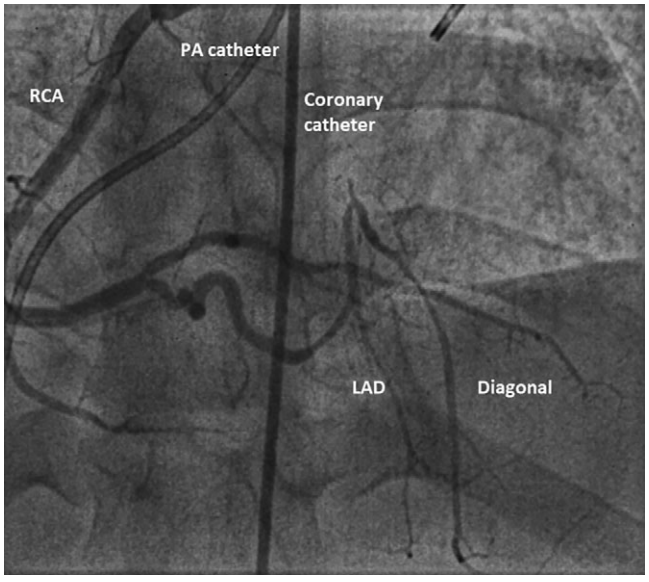

Figure 2. Right coronary artery angiogram in antero-posterior projection with cranial angulation. Diagonal: first diagonal artery, LAD: left anterior descending artery, PA catheter: pulmonary artery catheter, RCA: right coronary artery.

Myocardial bridges are usually visualised with invasive coronary angiography showing the milking or squeezing of the affected coronary artery, but can also be depicted and possibly quantified using intracoronary ultrasound, MRI, and CT.[3] Multidetector CT offers increased detection rates of myocardial bridging due to its higher resolution as well as the direct depiction of the myocardial bridge muscle.[1] Exercise stress testing, nuclear scintigraphy, coronary flow reserve evaluation by echocardiography, fractional flow reserve measurement, and myocardial perfusion imaging may be helpful in assessing the clinical relevance of myocardial bridging in affected patients.[9]

No treatment is required for asymptomatic patients with incidental diagnosis of myocardial bridging as this condition usually has a benign course. Symptomatic patients with myocardial bridges generally respond well to β-blockers as ischaemia usually manifests during tachycardia and stress, while nitrates should be avoided as they may exacerbate symptoms. Patients refractory to medical therapy have been successfully treated with percutaneous coronary intervention with the risks of stent fracture and coronary perforation, as well as with surgical myotomy and unroofing,[1] a procedure that has been reported successful even in children.[8,10]

In conclusion, myocardial bridging may exist in patients with CHD, as this case illustrates. Physicians should be aware of this possibility in the differential diagnosis of symptoms in CHD patients so that appropriate diagnostic and therapeutic strategies can be applied in this population.

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Conflicts of interest. None.

Ethical standards. No human or animal experimentation was involved.

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