Cardiol Young 2006; 16: 179–181

ISSN 1047-9511
doi: 10.1017/S1047951106000138

Brief Report

Correction of coronary arterial anomalies at surgical repair of common arterial trunk with ischaemic left ventricular dysfunction

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Abstract Common arterial trunk is frequently associated with anomalies of coronary arterial origin and distribution. We describe two infants in whom coronary intervention was performed during the repair of common arterial trunk, with reversal of ischaemic left ventricular dysfunction. We discuss the surgical implications of recognition of the coronary arterial anomalies during the repair of common arterial trunk.

Keywords: Truncus arteriosus; ischaemic heart disease; heart failure

The incidence of coronary arterial anomalies in common arterial trunk, as found in clinical series, is estimated to vary from 5 to 18%.[1,2] We report two infants with severe ischaemic left ventricular dysfunction in this setting due to associated coronary arterial anomalies. Intervention was successfully accomplished during the surgical repair, with reversal of ischaemic changes and recovery of the left ventricular function.

Clinical summaries

First patient

A 7-week-old male infant with the diagnosis of common arterial trunk, with a confluent arterial segment feeding the pulmonary arteries, was admitted in heart failure. He weighted 3.7 kilograms. His chest radiograph showed cardiomegaly and pulmonary plethora, and the electrocardiogram showed depression of the ST segments, with inversion of the T waves, in leads I, AVL, V1-V6. The echocardiogram revealed a dilated and poorly functioning left ventricle, with fractional shortening of less than 10%, a dyskinetic ventricular septum, and hypokinesia of the apical and lateral walls. The truncal valve was tricuspid with mild regurgitation, the right and left pulmonary arteries were of good size, and the aortic arch was normal. Cardiac catheterization was undertaken to define the coronary arterial anatomy, and revealed stenosis of the orifice of the left coronary artery, with retrograde filling from the right coronary artery (Fig. 1)

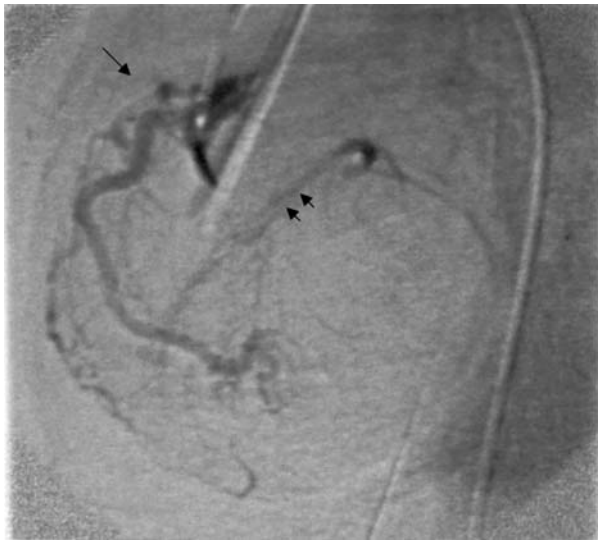

Figure 1.
Truncal root angiogram showing the dominant right coronary artery (single arrow), and stenosis of the origin of the left coronary artery, with retrograde filling of the artery from the right coronary artery (double arrows).

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Accepted for publication 5 October 2005

At operation the trifoliate nature of the truncal valve was confirmed. The stenotic orifice of the left coronary artery was situated between the zones of apposition of the non-coronary and left coronary arterial valvar leaflets. We also found an intra-mural course of the artery within the truncal wall.

A probe of 1 millimetre was passed into the artery, permitting unroofing of its intramural part course, following which the orifice was enlarged, securing the edges with 8/0 prolene sutures. This was followed by patch closure of the large sub-truncal ventricular septal defect through the right ventriculotomy, and insertion of a 12 millimetre pulmonary homograft to establish continuity from the right ventricle to the pulmonary arteries. A Shelhigh bovine pericardial patch was then used to repair the transverse aortotomy, taking care to avoid distortion of the previously intramural course of the coronary artery. The patient was separated from the cardiopulmonary bypass without difficulties. The postoperative course was uneventful, with delayed sternal closure being achieved on the second postoperative day. Five years after the operation, the patient is free from symptoms, with normal left ventricular function and no ischaemic changes on the electrocardiogram. Repeated catheterization at 4.5 years of age has revealed no obstruction to the left coronary arterial system, albeit with moderate obstruction of the conduit.

Second patient

A 4-months-old female infant weighing 3.3 kilograms was readmitted with severe heart failure. She had been diagnosed soon after birth to have duodenal atresia and common arterial trunk with confluent origin of the pulmonary arteries. The initial echocardiogram had shown a dysplastic and moderately regurgitant truncal valve, with anomalous origin of the left anterior descending coronary artery from the right coronary artery. Duodenostomy was performed at the age of 5 days. A palliative band was placed round the confluent segment of the pulmonary arteries at the age of 4 weeks because of persistent cardiac failure and failure to thrive.

The electrocardiogram on the current admission revealed left ventricular enlargement and anteroseptal ischaemic changes. The left ventricle was found to be dilated, with severely impaired function and septal hypokinesia on the echocardiogram. In addition there was severe truncal valvar regurgitation.

Intra-operatively, the truncal valve was seen to have four leaflets. The anomalous origin of the left anterior descending artery from the right coronary artery was confirmed, and its aberrant course crossing the right ventricular infundibulum was noted. The circumflex branch of the left coronary artery originated separately between the posteromedial and posterolateral leaflets of the truncal valve, but exited via an intramural course.

During correction, the ventricular septal defect was closed using a bovine pericardial patch placed through the truncal valve, and the right and left pulmonary arteries were reconstructed. The orifice of the truncal valve could not be divided due to anomalous course of the left anterior descending coronary artery across the right ventricular outflow tract. An aortic homograft of 13 millimetres dimensions was used to replace the truncal valve. The right coronary artery and the intramural segment of the left circumflex coronary artery were separately transferred to the neo-aortic root. Continuity between the right ventricle and the pulmonary arteries was established using an 11 millimetre Shelhigh conduit. The patient was separated from the cardiopulmonary bypass without difficulties. The post operative course was complicated by reduced ventricular function requiring inotropic support for 12 days, intestinal malfunction needing parenteral nutrition for a period of four weeks, and sepsis necessitating broad spectrum antibiotic therapy. The total stay on the intensive care unit was 19 days, and the patient was hospitalized for 39 days. One year after the operation, the patient is free of symptoms, with good left ventricular function and no ischaemic changes on the electrocardiogram.

Discussion

There are considerable variations in the origin and distribution of the coronary arteries in patients having a common arterial trunk.[3–5] If unrecognised, these anomalies contribute to peri-operative mortality and late sudden death following successful repair.

In one of the largest autopsy series, Suzuki et al.[3] studied 84 specimens of common arterial trunk, and found a high incidence of abnormalities in both the origin and the epicardial distribution of the coronary arteries. An abnormal location relative to the valvar sinuses was found in four-fifths, the arteries originating at the margins of the sinus, or at the upper margin of a zone of apposition between valvar leaflets. Dual orifice of the right or the left coronary arteries, common sinusal origin of the two coronary arteries, and atresia of the orifice of the left coronary artery, were also noted. In one-tenth of hearts, there was a single coronary artery. Origin of either the left anterior descending, or the left circumflex artery, from the right coronary artery, and absence or hypoplasia of the left circumflex artery, were other anomalies noted.

Stenosis of the origin of a coronary artery can be due to small size, its slit-like shape, or location of the orifice above or in the zone of apposition between the valvar leaflets, this arrangement causing functional

https://doi.org/10.1017/S1047951106000138 Published online by Cambridge University Press

obstruction. Low diastolic pressure due to aorto-pulmonary run-off, and truncal valvar regurgitation, can further compromise coronary arterial perfusion in this setting. This can lead to left ventricular ischaemia, infarction, and sudden death.[6,7] An aberrant course of an artery across the right ventricular outflow area exposes it to damage during placement of the conduit usually required for repair, and can cause post-operative death.[3,6,8] An intramural course is prone for distortion during reconstruction of the neo-aortic root after explanting the pulmonary arteries. It is also a potential risk factor for transfer of the coronary arteries during truncal root replacement.

Signs of myocardial ischaemia on serial electrocardiograms, and left ventricular dysfunction on serial echocardiographic examinations, are the pointers to coronary arterial anomalies. Careful evaluation of the coronary arterial anatomy in this scene helps to plan surgical interventions for the relief of orificial stenosis, and permits modification of the surgical strategy to avoid any damage to the anomalous coronary arteries during the surgical repair.

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