Role of Cardiovascular Magnetic Resonance Imaging in Postoperative Follow-Up After the Arterial Switch Operation for Transposition of the Great Arteries

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Cardiovascular magnetic resonance (MR) plays a major role in the evaluation of patients with transposition of the great arteries, especially during follow-up after surgical intervention such as the arterial switch operation (ASO). It is recommended in recent guidelines that cardiovascular MR should be integrated in the routine evaluation of all postoperative patients with transposition of the great arteries with the frequency dependent on nature of the operation, patient status, and other available clinical data. In some cases, cardiac MR should be considered the primary method for routine noninvasive evaluation with annual or biennial studies.1

During the ASO, the great arteries are transected, switched, and reanastomosed to the correct ventricle, resulting in a neoaoortic root and neopulmonary root with relocation of the pulmonary branches anteriorly to the neoaoorta by using the Lecompte manoeuvre, mobilization, and implantation of both coronary arteries in the neoaorta.

Several sequelae may occur after ASO that may require long-term follow-up by imaging. First, neoaoaric root dilatation and subsequent aortic valve regurgitation are widely recognized long-term complications after the ASO and are typically well tolerated hemodynamically. Maladaptation of the former pulmonary artery wall, scarring around surgical suture lines, intrinsic aortic wall abnormalities, and Gothic shape of the aortic arch may promote neoaoaric wall stiffening and dilatation, resulting in aortic insufficiency because of loss of coaptation of aortic valve leaflets, contributing to left ventricular hypertrophy and dysfunction.2 Cardiovascular MR studies have shown that enlargement of the neoaoaric root is frequently present after arterial switch surgery in conjunction with reduced elasticity of the proximal aorta, mostly minor degrees of aortic regurgitation and reduced left ventricular systolic function.3 In addition, early aortic stiffening in patients after ASO has been recognized as a contributing factor to left atrial enlargement and left ventricular diastolic dysfunction.4 Other factors may also contribute to early left ventricular dysfunction after ASO, including hypoplastic left anterior descending coronary artery, decreased coronary blood flow reserve, perfusion defects, and altered myocardial architecture.5 Coronary abnormalities may be found in asymptomatic subjects after ASO in 3% to 7% of the population. MR coronary angiography provides accurate information on coronary anatomy, ostial stenosis, and proximal coronary artery kinking in patients after ASO.6 Furthermore, late gadolinium enhancement is a useful MR technique to identify myocardial infarcts and viability when coronary occlusion has occurred. Moreover, late gadolinium enhancement may be combined with stress perfusion MR to detect coronary involvement after the ASO in one comprehensive protocol, although the yield seems to be low for detecting myocardial ischemia, and it has been suggested that cardiac MR imaging is not indicated routinely to evaluate possible myocardial ischemic damage in asymptomatic and clinically stable adult patients after the ASO.7,8

Other vascular sequelae after ASO involve the neopulmonary root and pulmonary artery branches. Pulmonary branch stenosis is seen in two third of patients after the ASO and is the most common reason for reintervention. Abnormal pulmonary artery stretch, decreased pulmonary artery distensibility because of scar, and overt stenosis of the neopulmonary root and branch pulmonary arteries may result in right ventricular hypertrophy, right ventricular relaxation disturbances, and right ventricular dilatation.9 Smaller size and reduced distensibility of the neopulmonary root has been recognized as an important and common determinant of reduced exercise capacity after the ASO at long-term follow-up.10

Both flow velocities and volume flow in the proximal aorta and pulmonary artery can be reliably measured using MR velocity-encoded techniques. Measurements are commonly obtained with 2-dimensional phase-contrast MR perpendicular to the vessel of interest with appropriate selection of velocity settings, thin slices, high temporal resolution, and preferable correction for through-plane motion. Great vessel flow and differential flow distribution to the left and right pulmonary artery branch and regurgitant flow can be monitored precisely by these techniques. For example, MR velocity mapping is the preferred modality to measure pulmonary regurgitation after Fallot surgery and to select patients for intervention. In ASO patients, both pulmonary flow and aortic regurgitation are accurately quantified by using MR velocity mapping. In addition, the great vessel dimensions and flow data can be used to assess local vascular distensibility and regional pulse wave velocity. Pulse wave velocity is measured at 2
locations in the large vessel of interest. The distance between the 2 locations (in meters) along the vessel of interest is divided by the difference in arrival time (in seconds) to calculate the pulse wave velocity (expressed in meters per second) as a marker of vessel stiffness. Distensibility is measured from the local vessel area change between systole and diastole corrected for pulse pressure. Mathematically, pulse wave velocity and distensibility are inversely related to one another.

The proximal aorta and aortic arch act as a buffer for left ventricular systolic load, modulating ventricular–aortic coupling between the left ventricle and elastic proximal aorta. Aortic stiffening in ASO patients contributes to the occurrence of aortic regurgitation and left ventricular dysfunction. Many possible contributing factors to aortic stiffening in ASO patients have been suggested, including abnormal aortico-pulmonary septation, damage to the vasa vasorum, surgical manipulation, inherited intrinsic wall abnormalities, and adaptation of the former pulmonary arterial wall to higher systemic pressures. Similarly, the ventricular–pulmonary interaction between the right ventricle and pulmonary vasculature can be evaluated by measuring pulmonary artery pulse wave velocity in conjunction with right ventricle structure and function. A new application available from MR velocity mapping is measurement of vortical blood flow along the main pulmonary artery by using 3-dimensional phase-contrast MR to estimate pulmonary arterial pressure noninvasively.

As discussed above, cardiovascular MR provides comprehensive evaluation of the postoperative cardiovascular sequelae after the ASO. Interestingly, there is increasing interest to explore the possible role of cardiovascular function in relationship to neurodevelopmental impairment in adolescents with congenital heart disease. MR imaging is not only well suited to assess cardiovascular function but also to investigate brain gray and white matter structures in the follow-up of congenital heart disease. For example, it has been shown that in children with dextro-transposition of the great arteries reduced white matter microstructure is associated with cognitive performance. Perioperative white matter injury because of hypoxia has been implicated as a dominant mechanism in causing structural changes in the brain in patients with congenital heart disease. However, it is now more widely recognized that there may also be a link between cardiovascular function and brain structure, leading to cognitive decline. Aortic stiffening is particularly deleterious to high flow/low impedance organs like the brain and kidneys. In the presence of aortic stiffening, microvascular brain disease may result from harmful vascular pulsatility penetrating into the microcirculation. The association between aortic stiffening and brain white matter disease and cognitive decline has been established, even in relatively healthy populations. Therefore, it is conceivable that proximal aortic stiffness may play an important role in causing brain alterations leading to cognitive dysfunction at long-term follow-up after ASO. Further follow-up studies are required to elucidate the possible role of cardiovascular function in ASO patients as a contributing factor to brain alterations that may be related to neurocognitive impairment.

In this issue of Circulation: Cardiovascular Imaging, Shepard et al. reviewed several cardiovascular MR imaging data in a large series of mostly asymptomatic adolescent and young adult patients after the ASO (n=220; median age: 15.4 years; 66.8% men). The goal of the study was to describe the range of biventricular volumes, presence and frequency of myocardial scar, range of postsurgical great vessel dimensions, and frequency and severity of neoaortic valve regurgitation after ASO. The MR imaging protocol included state-of-the-art pulse sequences for measuring ventricular volumes, great vessel dimensions, aortic and pulmonary flow dynamics, and myocardial scar. Cine MR imaging was performed in short-axis planes for estimating ventricular volumes. Vessel dimensions were measured at predefined locations. Ventricular volumes and dimensions were indexed for body surface area.

In the absence of a matched control group, the authors used the z score to substantiate their MR findings. The z score is a good way to express the distance between an individual’s measurement and the average of this measurement of comparable individuals in a reference population. The z score system expresses cardiovascular MR measurements as several SDs below or above the reference mean. The z score is calculated by following the equation: (observed value–average value of the reference population)/SD value of reference population. Because the z score scale is linear, summary statistics such as means, SDs, and SEs can be computed from z score values.

To obtain meaningful z scores, it is essential to choose a reference population that is similar to the study population. However, Shepard et al used an older population for the determination of z scores for left and right ventricular volumes. It is therefore debatable whether these scores are a true reflection of the difference between the study population and a healthy reference population. Other confounders to consider are the MR pulse sequence that was used to measure a specific parameter (eg, endocardial border definition and resulting measurement of volumes vary depending on MR pulse sequence, field strength, and gating versus nongating), the criteria for accurate measurements (eg, luminal versus outer wall to outer wall vascular measurements, through-plane motion effect, inclusion/exclusion of trabeculation, and papillary muscles for volume and mass calculation), the expertise of the observer, the scan–rescan reproducibility, and the specific software algorithm used for postprocessing and analysis of the various MR imaging sequences and biological variation (eg, older versus younger age group and sex differences may be significant).

The authors applied a consistent imaging protocol with standardized measuring procedures and present MR-based reference values of ventricular volumes and great vessel dimensions in their cohort of ASO patients. The statistical analysis included sex as a covariate, but sex seemed not to have statistical effect in their model. Dilatation of the neoaortic root was common at 76% and was associated with mostly mild neoaortic valve regurgitation. Of note, the authors found significantly higher growth rate of the neoaorta in a subset of the ASO population as compared with control subjects. Left ventricular dilatation (defined by increased end-diastolic volume) was seen in 26% of the ASO patients and right ventricular dilatation in 20% of the population. Mostly mild left ventricular and right ventricular dysfunctions were observed in 21.5% and 5.1%, respectively. Myocardial scar consistent with myocardial infarction was only seen in 1.8% of the patients, whereas
reoperation for coronary obstruction had been performed in 3.6% of the population. The small percentage of scar in this study confirms the low yield of detecting ischemia in ASO patients reported in the literature. Furthermore, in the study by Shepard et al, the neopulmonary root showed an oval shape with decreased anteroposterior diameter and increased lateral diameter, whereas branch pulmonary arteries were smaller with near-normal differential branch flow distribution. It may be clinically relevant to assess the relationship between abnormal shape and function of the neopulmonary root and exercise capacity at follow-up as previously reported.

These observations confirm what is largely known from many small ASO patient series evaluated by MR imaging and other imaging modalities. The study by Shepard et al provides reference values from a large database of asymptomatic and young ASO patients using state-of-the-art MR imaging technology. These data are important for follow-up of this growing population and may help to define imaging criteria for decision making, reintervention, and cardiovascular risk stratification.

Disclosures

None.

References


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