

Cardiac remodeling and function in aortic coarctation: from the fetus to the newborn

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Background:

Persistent left ventricular (LV) dysfunction has been described in neonates and adults with coarctation of the aorta (CoA). However, comprehensive assessment of biventricular microstructure, morphology and function in CoA, from fetal to neonatal stage, has not been previously performed. This information might improve prenatal counseling and postnatal management .

Methods:

A comprehensive morphometric and functional evaluation was performed in 22 fetuses with confirmed CoA and 44 gestational-age matched controls using 2D, M-Mode, pulsed-wave and tissue Doppler imaging (TDI) techniques in the third trimester of pregnancy and in the first 48h after birth. Microscopic fiber orientation was assessed in a fetal CoA specimen and compared to a normal heart, using phase-contrast synchrotron tomography. Sphericity index (SphI) was calculated as longitudinal/mid-transverse ventricular diameter.

Results:

Compared to controls, fetuses with CoA showed smaller and more elongated LV (LV SphI: CoA 2.45 ± 0.84 vs. controls 1.88 ± 0.26 , $p < 0.001$) and larger right ventricles (RV) with less extensive shape changes (RV SphI: CoA 1.46 ± 0.29 vs. controls 1.69 ± 0.23 , $p = 0.005$), and no signs of biventricular dysfunction. No signs of ventricular hypertrophy were observed, and synchrotron tomography revealed normal fiber organization. After birth, the LV became more globular (LV SphI: CoA 1.47 ± 0.22 vs. controls 1.76 ± 0.17 , $p < 0.001$) and showed signs of diastolic impairment (left E/E' CoA 17.75 ± 7.6 vs. controls 9.88 ± 3.45) with preserved systolic function. The RV had a normalized shape (RV SphI: CoA 1.56 ± 0.27 vs. controls 1.67 ± 0.24 , $p = 0.117$) and increased longitudinal function (increased S', E' and A', stroke volume and cardiac output) in the presence of a patent arterial duct.

Conclusions:

Fetuses with CoA present biventricular structural changes with preserved fiber organization and function. The prenatal elongated LV's shape suggested a ventricular "modeling" under an unloaded hemodynamic situation, followed by a maladaptive postnatal response, with signs of neonatal modeling and diastolic dysfunction. These results provide new insight into the causes of neonatal LV dysfunction and might help develop new strategies to prevent or ameliorate adverse early ventricular remodeling in CoA.