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Is analysis of physiological late gestation ventricular-arterial disproportion futile?

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Background Ventricular-arterial disproportion is a frequent observation in the third trimester and mostly considered physiological. It is also an indirect prenatal sign for postnatal coarctation. We sought to investigate the remnant risk for coarctation and its possible risk factors in a fetal population with physiological late gestation asymmetry.

Methods This was a monocentric, retrospective study conducted from 2014 till 2017 in a Fetal Cardiology Unit. All fetuses referred for third trimester asymmetry with pulmonary artery/aorta ratio > 1.2 were included. Annular sizes, arterial Z-scores, valve ratios, isthmus size if available and minor associated cardiac defects were analyzed.

Results 151 fetuses were referred at a mean gestational age of 33.5 SA. After fetal echocardiography, 2% (3/151) were considered at high risk for coarctation because of a hypoplastic arch and/or a small left outflow tract or isthmus. They were scheduled to deliver in a tertiary referral center. Two of these 3 newborns developed coarctation. In 98% of fetuses asymmetry was considered physiological with a normally sized aortic arch without the ''shelf sign''. All infants had an early ambulatory echocardiography that revealed coarctation in 7/148 cases (4.7%). There were no significant differences in mean arterial annular sizes, aortic and pulmonary Z-scores, pulmonary artery/aorta ratio, atrioventricular annular sizes, tricuspid/mitral valve ratio, presence of ventricular septal defect or left persisting superior caval vein between the two groups.

Conclusion Remnant risk for coarctation in a fetal population with physiological late gestation asymmetry is < 5%. Prenatal counselling can be reassuring since the majority of infants will have a normal heart. However early cardiac evaluation of these newborns remains recommended to exclude the rare event of postnatal coarctation in absence of prenatal predictive signs.

Keywords Left-right asymmetry; Ventricular-arterial disproportion; Late gestation; coarctation; Prenatal diagnosis

Disclosure of interest The authors declare that they have no competing interest.

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4D cardiac magnetic resonance flow in patients with pulmonary hypertension associated with congenital heart disease

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Introduction Right heart catheterization is an invasive exam that is currently the gold standard to assess pulmonary hemodynamics for diagnosis and follow-up of pulmonary arterial hypertension (PAH) associated with congenital heart diseases (CHD). Cardiac magnetic resonance 4D flow (4D CMR flow) emerges as a promising non-invasive imaging. We assess the accuracy of 4D CMR flow to measure pulmonary cardiac output (Qp).

Methods We prospectively included 28 patients with PAH associated with CHD. Qp was measured invasively using Fick principle (direct oxygen consumption measure) during aright heart catheterization (QpF) and compared to Qp measured by 2D (Qp2D) and 4D flow CMR (Qp4D) on the same day.

Results Twenty eight patients was included (median age was 42 years old [35—52]) with PAH and CHD (pre-tricuspid shunt $n = 23$, 82.1%, median mean pulmonary artery pressure 46 mmHg [40-58]; median pulmonary vascular resistance 15.0 WU.m^2 [7.5—25.6]) 4D CMR flow analysis were feasible in all patients. Qp4D and QpF were strongly correlated (rho = 0.87, $P < 0.0001$; $r^2 = 0.68$, $P < 0.0001$). Using Bland Altman analysis, mean difference was 0.0 ± 1.0 L/min. Mean difference within two observers (interobserver variability) was 0.3 ± 0.4 L/min. Mean difference within one observer (intra-

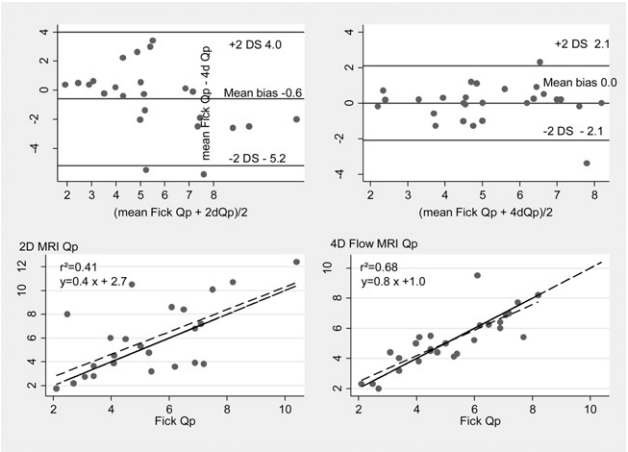

Fig. 1 Comparison of (QpF) to Qp measured by 2D (Qp2D) and 4D flow CMR (Qp4D).

observer variability) was 0.1 ± 0.3 L/min. Qp2D and QpF were moderately correlated (rho = 0.54, $P = 0.008$; $r^2 = 0.41$, $P = 0.0007$). Using Bland Altman analysis, mean difference was 0.6 ± 2.3 L/min (Fig. 1).

Conclusion Qp measured by 4D CMR flow is well correlated to QpF. Further studies are needed to explore other potential interests of using 4D CMR flow to assess PAH, including the derivation of additional hemodynamic parameters such as pulmonary artery compliance, wall shear stress and pulse wave velocities, which could provide further insights into pulmonary artery remodeling and interactions between pulmonary arterial stiffening and right ventricular dysfunction.

Keywords Pulmonary arterial hypertension; Congenital heart disease; Pulmonary cardiac output; 4D cardiac magnetic resonance

Disclosure of interest The authors declare that they have no competing interest.

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