The impact of an absent ductus arteriosus on clinical outcomes in fetuses diagnosed with Tetralogy of Fallot

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Objectives: Fetuses with Tetralogy of Fallot (TOF) almost universally have a patent ductus arteriosus (PDA). Two recent fetal cases of TOF had an absent PDA, requiring emergent intervention at birth. The objective of this study was to determine whether fetuses diagnosed with Tetralogy of Fallot (TOF) without a PDA have worse clinical outcomes compared with fetuses diagnosed with TOF + PDA.

Methods: All fetal cases of TOF between January 2000 and January 2012 were retrospectively identified from The Hospital of Sick Children (Toronto, Canada) database. Concomitant diagnoses of atrioventricular septal defect, pulmonary atresia, or absent pulmonary valve were excluded. Cases (TOF + no PDA confirmed at first postnatal echo) and controls (TOF + PDA, matched for gestational age (GA)) were reviewed. Optimal outcome was defined as valve sparing repair with no residual lesions. Student’s t-tests and Fisher’s exact chi-square were used to compare groups.

Results: A total of 115 fetuses were diagnosed with TOF: 11 (9%) had no PDA and 22 were matched controls (mean GA at diagnosis 32.1 ±6.5 weeks, 30.8 ±6.6 weeks, respectively). Cases had a higher proportion of right aortic arches (64% vs. 14%, P<0.001). At birth, mean right outflow gradients were 31 mmHg (25-52) vs. 27 mmHg (21-42), P=0.30. Fetal and postnatal echocardiographic data did not reveal significant differences in branch pulmonary artery sizes, pulmonary valve sizes, or ventricular function. No differences were identified for cyanosis at birth (2/10 vs. 7/10, P=0.67), or early catheter intervention (5/10 vs. 4/22, P=0.12). Overall survival was 9/11 in cases vs. 18/22 controls, P=1.00. Optimal outcome rates were similar between cases and controls (4/11 (36%) vs. 5/21 (24%), P=0.68).

Conclusions: The PDA does not appear to be relevant in fetuses with TOF to ultimate clinical outcome. The TOF physiology may allow for redistribution of pulmonary blood flow, enabling normal growth of the pulmonary vascular bed.