P-71

Foetal dysfunction of the arterial duct: clinical spectrum and outcome

Eyskens B., Demeyer S., De Catte L., Heying R., Cools B., Brown S., Gewillig M.
Fetal & Pediatric Cardiology, Neonatology, Leuven, Belgium

Background: premature foetal duct closure cause right heart & pulmonary damage. Outcome of milder dysfunction (partial or transient constriction, aneurysm formation) needs to be defined.

Methods: retrospective review; foetal (942) and neonatal echo databases searched for evidence of prenatal ductal dysfunction (1998-2010). Prenatal inclusion criteria were closure – contriction - aneurysm of arterial duct. Postnatal (<14d) inclusion citeria were excessive RV hypertrophy; cyanosis by atrial R<L, and/or abnormal findings duct.

Results: 26 patients were identified. 12 prenatally (gestational age 25,7w (21-37w, incidence: 12/942 = 1.3% abnormal scans), 13 postnatally (age D2, range D0-11).
In the foetal cases the duct was closed (9), abnormally small (2), aneurismal (1). 6 patients presented at birth with significant cyanosis without duct. 5 mothers had taken NSAID during pregnancy. Patients had RVH (25), bipartite RV (16), RV aneurysm (1), significant TR (23), hydropericardium (2) and hydrops (1); PS (6) and PR (22) ranging from mild to « agenesis » of pulmonary valve (3); dilation of pulmonary trunk (6) and branch pulmonary arteries, compression of airways with “fluid-trapping” and microcystic malformation of lungs (1); fetal suprasystemic pulmonary hypertension (5); 2 pt had a thrombosed aneurysm of the duct. In 6 patients premature delivery was chosen to avoid further intra-uterine damage of the right heart & lungs. Neonatal treatment varied from observation (12), ventilation with pulmonary vasodilators (8) and ECMO (1); resection of a thrombosed aneurysmal duct as the thrombus was occluding the left pulmonary artery (2); 3 patients had balloon dilation PS. 3 patients died in the neonatal period because of respiratory insufficiency. Late treatment: balloon dilation PS (2), and homograft reconstruction with PA plasty at 4 years for late compression of coronary artery (1). 2 pts have mild psychomotor delayed development; non-compaction cardiomyopathy (1).

Conclusions: Fetal dysfunction of the arterial duct can stress at different fetal ages many different levels of the right heart and pulmonary circulation, resulting in a very wide range of secondary pathology. The clinical outcome ranges from normal to death; neonatal death was due to lung damage. Premature delivery might be indicated in selected patients.