Persistent pulmonary hypertension in newborns with TGA – can it be predicted prenatally?

Grzyb A. (1,2), Kolesnik A. (1,2,3), Brzezinska-Rajszys G. (2), Brodzikowska A. (2), Zubrzycka M. (2), Rewers B. (2), Bokiniec R. (5), Dangel J. (1,4)
(1) Reference Center for Fetal Cardiology, Warsaw Hospice for Children Foundation, (2) Cardiology Department, The Children’s Memorial Health Institute, (3) Descriptive Anatomy Department, Medical University of Warsaw, (4) 2nd Department of Obstetrics and Gynecology, Medical University of Warsaw, (5) Neonatology and Neonatal Intensive Care Department, Medical University of Warsaw

Objectives: Transposition of great arteries (TGA) is one of the most common cyanotic congenital heart defects. Despite prenatal diagnosis, in cases with restriction at the level of foramen ovale and/or ductus arteriosus, it can be fatal in the newborn. When additionally complicated with persistent pulmonary hypertension of the newborn (PPHN), it poses even a greater danger, as standard therapy (PGE1 and balloon atrial septostomy (BAS) may fail to provide adequate intracardiac mixing and blood oxygenation.

The aim of this study was to search for predictive factors of pulmonary hypertension in the newborns with TGA, based on prenatal echocardiographic examinations.

Methods: We evaluated echocardiographic exams of fetuses with TGA diagnosed between 2011–2016 in the referral center for fetal cardiology. Follow-up data were collected from target pediatric cardiology departments.

Results: Out of 60 patients diagnosed with TGA, 16 showed signs of PPHN, requiring treatment with iNO (14), Epoprostenol (1) or both (1). 15/16 underwent BAS within the 1st day of life. In 14/16 fetuses signs of foramen ovale restriction and limited interatrial blood mixing were observed prenatally, usually before 35th week of pregnancy. Additionally, in 6 of them, restrictive or bidirectional flow in DA was observed, which implied increased pulmonary blood flow. In 2 remaining fetuses restriction of flow at FO level occurred after 38th week of pregnancy.

From 16 newborns with restrictive FO and PPHN 13 survived and underwent arterial switch operation successfully. From the remaining group, 3 newborns died before any attempt of intervention – in all of them there were signs of restrictive FO and in 1 – restrictive DA prenatally.

Conclusions: FO and DA flow in fetuses with TGA should be assessed repeatedly, especially just prior to delivery, as it can change during pregnancy. Restrictive flow across FO (especially long-lasting), together with increased pulmonary flow in fetuses seem to be predictive of PPHN in the newborn with TGA. Such knowledge is of great importance for neonatologists and interventional cardiologists in order to provide the optimal and intensive treatment for the newborn. In all cases of TGA with PPHN prenatal diagnosis enabled to provide the optimal treatment and saved newborns’ life.