AEPC-YIA Session

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[AEPCYIA] AEPC-YIA Session（II-AEPCYIA）

Chair:Jan Janoušek（Children's Heart Centre, Motol and Homolka University Hospital, Prague, Czech Republic）
Chair:Mikiko Ishido（Department of Pediatric and Adult Cardiology, Tokyo Women's Medical University）

[II-AEPCYIA-2] A Biopsychosocial Model for Understanding Early Neurodevelopmental Vulnerability in Children With Congenital Heart Defects

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Background and Aim:

Children with congenital heart defects(CHD) are at increased risk for neurodevelopmental impairment, beginning early before surgical or intensive care exposure. To characterize risk mechanisms, we integrated findings addressing neurodevelopmental, biological, and psychosocial contributors to early developmental outcomes.

Methods:

A complex prospective cross-sectional study, designed as a biopsychosocial model, included 158 participants, composed of:
(1)A preoperative study that included children under 6-years of age with unrepaired non-cyanotic and cyanotic CHD. Psychomotor development was assessed using Denver Developmental Screening Test-II (DDST-II), generating domain-specific and global-developmental scores. (2)A parallel biomarker study that analysed serum neurotrophic and glial proteins like Brain_Derived_Neurotrophic_Factor(BDNF), Neuron_Specific_Enolase, protein-S100, Glial_Fibrillary_Acidic_Protein(GFAP), Tau_protein, Myelin_Basic_Protein, Adrenomedullin, Monocyte_chemoattractant_protein to evaluate their discriminative value for developmental delay. (3)A psychosocial study that assessed maternal psychological adaptation and child-related stress using the ICCAP (Impact of Congenital Anomalies on Parents) questionnaire, covering six functional domains, examined associations with children's developmental outcomes.

Results:

In the developmental study, 97% of children with cyanotic CHD and 54% of those with non-cyanotic CHD demonstrated developmental delay. Global DDST-II scores were significantly lower than established norms (p=0.03), with gross-motor and personal–social domains being the most affected. Prenatal diagnosis (p=0.012) and exclusive breastfeeding (p=0.008) were associated with better developmental performance. In the biomarker analysis, BDNF and GFAP emerged as the strongest candidate neuromarkers, demonstrating moderate discriminative accuracy for developmental delay (AUC=0.72 vs AUC=0.75). A threshold >5895pg/mL for BDNF and >0.68ng/ml for GFAP were associated with increased developmental risk.

In the psychosocial study, higher maternal stress—specifically in the "social experience" (r=–0.24, p=0.03) and "contact with medical staff" (r=–0.28, p=0.01) domains—correlated with lower child psychomotor scores.

Conclusions:

Findings across these three studies converge to demonstrate that children with unrepaired CHD experience substantial early neurodevelopmental vulnerability stemming from biological, developmental, and psychosocial pathways, independent of peri-operative factors. BDNF and GFAP may represent promising early neuromarkers for identifying children at risk. Psychosocial burden was demonstrated to add a contribution to early developmental vulnerability. Early neurodevelopmental screening, prenatal counselling, promotion of breastfeeding, and integrated biopsychosocial care pathways could improve early outcomes and allow individualized follow-up for children with CHD and their families.