Letter by Li et al Regarding Article, “Risk Factors for Abnormal Developmental Trajectories in Young Children With Congenital Heart Disease”

To the Editor:

Mussatto et al wrote an interesting and important article about the abnormal development of young children with congenital heart disease (CHD), especially as it relates to neurological development. The authors have identified indicators responsible for the abnormal development trajectories. More importantly, this article revealed that detection of abnormal development associated with CHD at a very early stage could result in efficient interventions for young children to help improve their academic and motor function.

Abnormal development in children with CHD is an old issue. Because treatments have progressed greatly in the past several decades, researchers have begun to monitor protection and treatment beyond the heart itself. Many studies have associated brain injury with CHD. More cardiac surgeries, longer hospital stays, poorer linear growth, and the need for tube feeding were demonstrated risk factors. However, could we regard them as the indicators for worse conditions of the patients that might be directly associated with the specific characteristics of particular CHD types?

Previously, we published a meta-analysis of neurodevelopmental delay with critical CHD. In this research, we selected studies using functional MRI to assess the origin of the delays. Taking advantage of functional MRI, we could evaluate brain function at a very early stage. The overall average diffusivity was significantly higher with lower fractional anisotropy in CHD cases. In addition, there was a reduction of N-acetylaspartate/choline in CHD cases with higher lactate/choline. To our surprise, our analysis confirmed brain damage in cases with significant changes in N-acetylaspartate/creatine and N-acetylaspartate/choline within the immediate term postoperatively, relative to preoperative values. However, the difference did not persist at short-term follow-up and, therefore, we concluded that neurodevelopmental delay was mainly a result of prenatal injuries associated with CHD, not cardiac surgeries.

Accordingly, we concluded that abnormal development trajectories originated from fetal life, and the impact from neonatal life was limited. With the rapid development of the theory of the developmental origins of health and disease, researchers started to focus on the factors that might influence the prognosis of particular diseases during pregnancy. Although it is very important to search for the indicators related to abnormal development during early life in CHD children, this was not our goal. With these emerging techniques, we could obtain more information about fetal diseases to fully understand the abnormal development trajectories in children with CHD.

References

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Disclosures
None

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