

Anatomic variations of the Circle of Willis in neonates: a high resolution MRA study at 3 Tesla

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Background

The Circle of Willis (CoW) is a ring-like arterial structure that can serve as a potential collateral pathway in case of impaired or decreased flow within one or more of the major cerebral vessels (fig.1). While Doppler Ultrasound studies provide functional information mostly about flow distribution in the CoW¹, MR Angiography (MRA) morphological studies in adults have contributed to the identification of anatomic variations in the CoW with high sensitivity and specificity when compared to the gold standard X-Ray Angiography². Those anatomical studies have shown that in a healthy adult population less than 50% had a complete CoW³. In addition there is a significantly higher percentage of entirely complete CoW in patients with Internal Carotid Artery (ICA) obstruction when compared with control subjects⁴, suggesting the formation of collaterals. The ability of the CoW to redistribute blood flow depends on the presence and morphology of its component vessels, ie hypoplasia or absence of the Posterior Communicating Artery (PCoA) in patients with ICA occlusion is considered a risk factor for ischemic stroke⁵.

Previous MRI studies of the neonatal brain have shown that the neural tissue of the preterm brain develops differently compared to the term brain^{6,7,8}.

To date and to our knowledge there are no MRA reports on the anatomic variations of the CoW or its ability to supply collaterals in preterm and term born infants. MRA, being non invasive, provides a safe technique that can be used for longitudinal studies. MRA at 3 Tesla has intrinsically increased Signal-to-Noise Ratio (SNR) and therefore allows superior small vessel visualisation which is crucial when imaging the small neonatal vessels.

Methods

The aim of this study is to use MRA at 3 Tesla to identify anatomic variations of the Circle of Willis in the developing brain. We recruited 31 infants (18 boys and 13 girls) with normal conventional T1 and T2 weighted imaging scanned with an optimised⁹ high resolution 3D Time-Of-Flight (TOF) MRA protocol (isotropic true resolution of 0.6x0.6x0.6mm³). Nineteen were preterm infants imaged at term equivalent age [GA: 29.4wks (25.7-34.6wks), PMA: 40.1wks (37.1-44wks)] and twelve were patient control infants [GA: 39.8wks (38-41.4wks), PMA: 42.2wks (38.9-45wks)]. Additionally infants with a vascular disease such as neonatal stroke (n=4), Sturge-Weber Syndrome (SWS) (n=3) and Retinopathy of Prematurity (RoP) (n=3) were assessed separately. Research Ethics Committee approval and informed parental consent was obtained prior to each scan.

All images were checked for motion artefacts. The Maximum Intensity Projections in all planes along with the source MRA images have been used to identify and confirm anatomic variations in the CoW. Images were assessed for the identification of the anatomic variations of the CoW, namely the missing A1 segment of the Anterior Cerebral Artery (ACA), the transitional/fetal type origin of the Posterior Cerebral Artery (PCA) and absence or hypoplasia of the Posterior Communicating Arteries (PCoA). The differences in the occurrence of a complete CoW between preterm and term-born infants and between infants with and without a primary vascular disease were assessed.

Results

The CoW was complete (fig.2) in more term (5/12) than preterm at term infants (2/19). The commonest anatomic variation was the absence/ hypoplasia of either or both the PCoAs affecting equally the term and preterm infants but more boys than girls (14:5). The transitional/ fetal type origin of the PCA (fig. 3) was seen more often in preterm infants at term (8/19) than in the term infants (6/12). More anatomic variations appeared unilaterally than bilaterally (12:19), with the fetal type origin affecting more frequently the left than the right side (5:2). In two out of three infants with SWS hypoplasia of the A1 segment was observed. The Anterior Communicating Artery (ACoA) was difficult to visualise even in the source images due to its small vessel size. The percentage of entirely complete CoW in infants with vascular disease was increased (6/10) when compared to those without vascular disease (4/26), a finding similar to the adult studies⁴.

Discussion

The absence of visualisation of an arterial branch in TOF MRA images might be due to aplasia, slow flow or very small vessel calibre. Nevertheless the use of an optimised⁹ high resolution three-dimensional TOF MRA protocol at 3 Tesla allows reliable identification of the anatomic variations of the small neonatal vessels that have slower blood flow. Further optimisation is needed for the accurate depiction of the ACoA.

The increased percentage of incomplete CoW in the posterior cerebral circulation when compared to the anterior vessels is in keeping with the adult literature³. The decreased percentage of complete CoW along with the increased occurrence of fetal type origin of the PCA in the preterm infants suggests a difference in vessel growth. Further longitudinal MRA studies in those infants will help us understand the development of these variations in postnatal life and provide an explanation of the differences observed between the term and preterm population in this study.

The knowledge of the anatomic variations in the neonatal CoW is indispensable not only for providing information about normal cerebrovascular development and insights into factors that influence vessel growth in the developing brain but it can also contribute to the identification of infants at risk of ischemic brain injury due to impaired collateral flow formation. It may also be useful for the detection of potential collateral pathways in infants undergoing extracorporeal membrane oxygenation (ECMO), shunting or other interventions that may disrupt the delicate hemodynamic balance of the brain. Furthermore it can help explain the improved outcomes of infants following neonatal stroke compared to the outcomes of adult stroke, as previous studies have shown¹⁰.

References

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Figure1, 2: Complete Circle of Willis configuration with all its component vessels present. Figure 3: The CoW with two different anatomic variations, absence of the A1 segment of the Anterior Cerebral Artery (thin arrow) and fetal type origin of the Posterior Cerebral Artery from the ipsilateral Internal Carotid Artery (thick arrow).

