18th World Congress in Fetal Medicine

Early diagnosis of fetal brain migration abnormality

Ozdemir H, Turgut E, Karcaalincaba D, Erdem M, Bayram M
Gazi University Faculty of Medicine, Ankara, Turkey

Objective

Neuronal migration disorders are caused by the abnormal migration of neurons in the developing brain. Neurons must migrate to the areas where they will settle into their proper neural circuits. Migration occurs as early as the second month of gestation. When this migration is absent or incorrect, it can result in structurally abnormal or absent areas of the cerebral hemispheres, cerebellum, brainstem, or hippocampus, including schizencephaly, porencephaly, lissencephaly, agyria, macrogyria, pachygyria, microgyria, micropolygyria, neuronal heterotopias (including band heterotopia), agenesis of the corpus callosum, and agenesis of the cranial nerves. Periventricular heterotopia (PNH) is the result of abnormal development of the neuroependyma. PNH consists of collections of disorganized neurons and glial cells that are located along the walls of the lateral ventricles. We report a case that showed transient periventricular abnormalities at mid-gestation and then developed gyral abnormality.

Methods

This is a case report.

Results

A 23 year old nulliparous patient was referred to our clinic for routine mid-trimester detailed ultrasound. It was a dichorionic twin pregnancy conceived by in vitro fertilisation. One twin had a normal ultrasound examination. The other twin had normal biometry and normal anatomy except for an anomaly of the posterior horn of the left lateral ventricle. It appeared as a diffuse hyperechogenic posterior horn, with irregular ventricular walls. There was a small anechoic fluid-filled space within the ventricle. Biparietal diameter (BPD) and head circumference (HC) were normal. A neuronal migration abnormality was suspected and at 28 weeks' gestation, BPD and HC were measured three weeks smaller than expected. At 30 weeks' gestation, BPD and HC were compatible with 26 weeks of pregnancy and there was pachygyria, the echogenicity but irregularity of the lateral ventricles had disappeared. Fetal MRI was performed, showing pachygyria, a thin corpus callosum, and cortical dysplasia. Preterm labour occurred at 35^{+3} weeks' gestation and Caesarean delivery was performed due to malpresentation of the leading twin. A microcephalic female infant weighing 1780g was delivered with Apgar scores 6/9. The twin that was considered as healthy was born weighing 2400g with Apgar scores 8/9. HC of the abnormal newborn was 26 cm ($<5^{th}$ percentile). Postnatal cranial MRI and cranial ultrasound were performed, confirming the prenatal diagnosis.

Conclusion

Neuroblasts arise in the germinal matrix (in the subependymal area), and most migrate towards the cortex to predetermined locations. Irregular ventricular border and nodules protruding into the ventricular lumen are classical image findings of PNH. In our case, there was a diffuse echogenic and irregular filling of the posterior horn of the left lateral ventricle however this sign was transient.