expression pattern to each of the 3 PCP genes, *Vangl2*, *Fz3* and *Celsr3* that are implicated in mediating axonal guidance. We will also compare the expression of Scrib1 at E11.5 to TAG-1 that is expressed on the precrossing and crossing segments in the spinal cord and L1 that delineates postcrossing axons or growth cones. This study constitutes an essential step towards identification of the molecular and cellular role of *Scrib1* during axonal guidance in the spinal cord. Identification of these mechanisms will provide significant insights into the pathogenesis of axonal guidance neurodevelopmental diseases.

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Posterior reversible encephalopathy syndrome with atypicalregions in eclamptic patients: A challenge for radiologists

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Purpose: to determine the distribution and incidence of atypical regions of involvement of PRES in eclapmtic patients by using MR imaging.

Material and Methods: A prospective study included Twenty Two registered eclamptic patients (age ranged from 20–38 years) who had clinical signs and symptoms of PRES were recruited in the study, all patients were referred from Gynecology and Obstetric department, for brain MRI to evaluate PRES after clinical suspiscion. All images were reviewed for the presence of high signal intensity on FLAIR and T2WI, for the severity of the vasogenic brain edema, on the basis of the extent of hyperintensity on FLAIR imaging. DWI were also interpreted for the presence or absence of areas of restricted diffusion corresponding to the hyperinternsity areas on T2WI and FLAIR images.

Results: Most commonly involved location was the parieto-occipital brain region, which was seen in 19 (86.3%) of the (22) patients. This was followed by the frontal lobe in 13 patients (59%), the temporal lobe in 3 (13.6%), Basal Ganglia in 3 patients and cerebellum in 3 patients. Restricted diffusion was present in 9 patients (40.9%), half of patients had moderate edema ($n = 11$) while only 2 patients had severe edema and 9 had mild edema.

Conclusion: PRES can affect anterior circulation structures and atypical regions fairly frequent than commonly known. However, a posterior predominance is certainly seen in each lobe, Atypical regions of involvement represents challenge for radiologist and necessitate strict clinical correlation and follow up.

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Hippocampal cell death following neonatal anoxia: Functional consequences on spatial memory at adulthood

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Neonatal anoxia in rodents has been used as an experimental model to understand neural cell death and cognitive dysfunctions following human systemic asphyxia.

In this study, we highlight cellular and subcellular patterns of hippocampal neuronal cell death following a non-invasive anoxia induction byglobal oxygen deprivation in neonatal rats (P1),by combining different techniques such as TUNEL, Fluoro-Jade® B (FJB), cleaved caspase-3 immunohistochemistry (IHC) and transmission electron microscopy (TEM). Our time-line follow-through is comprised of P2, P14, P21 and P60 ($n = 5$ per group). In addition, the impact of neonatal anoxia on spatial memory was evaluated at P60.

P2 anoxic subjects exhibited higher TUNEL labelling in the CA1 and CA2-3, and higher FJB-positive cells in the CA3 as compared to controls; TEM confirmed canonical features for necrotic cell death and also revealed evidences of autophagy,necroptosis and excitotoxicity. At later ages, few TUNEL+ and FJB+ labelling were observed. Stereological analysis revealed no significant differences between groups in cleaved caspase-3 IHC at all ages, but indicated increased labelling in the dentate gyrus (DG) at P2.

Adult rats (P60) exposed to neonatal anoxia exhibited deficits in acquisition and performance of the reference memory version of the Morris Water Maze task; as compared to control subjects, anoxic animals exhibited increased latencies and path lengths to reach the platform, and decreased search for the specific platform location. In contrast, no differences were observed in swimming speeds and frequency in critical quadrant, thus indicating that their poorer performance is not related to sensory and motor deficits.

These results suggest that neonatal anoxia in rodents lead to hippocampal cell losses linked to impairments on spatial memory tasks at adulthood. The mechanisms involved in hippocampal neuronal lossfollowing neonatal oxygen deprivationdeserve additional investigation in order to develop strategies to minimize its long-term disturbing effects.

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