abilities in children born after IUGR. It seems that biological determinants may be the reason for that stronger dependence of morphological variables on other language abilities and for the formation of a compensatory language mechanisms. Children born after IUGR are risk for difficulties in reading and writing according to the structure of their linguistic knowledge: it is important for all professionals involved in follow-up process of this children.

**PO-0845 ACUTE NECROTIZING ENCEPHALOPATHY IN CHILDHOOD – EPIDEMIOLOGICAL, RADIOLOGICAL FINDINGS AND OUTCOMES**

1HY Lim, 1Y Ho, 1T Thomas, 1WS Chan. 2Department of Paediatric Medicine, KK Women and Children Hospital Singapore, Singapore, Singapore; 1Department of Paediatric Subspecialties, KK Women and Children Hospital Singapore, Singapore, Singapore

10.1136/archdischild-2014-307384.1474

**Background and aims** Acute necrotizing encephalopathy in childhood (ANE) is a disease characterised by acute encephalopathy and radiological features of bilateral thalamic necrosis. Medium and long term morbidity is not well described. We describe the mortality and morbidity outcomes in our paediatric cohort with this disease.

**Methods** This is a retrospective ten-year series. Children aged one month to 18 years diagnosed with ANE were collated from Neurology and Radiology databases.

18 fulfilled clinical criteria of acute encephalopathy. All were scored with Mizuguchi’s radiological checklist by two paediatric neurologists and one radiologist. 11 cases scored unlikely were excluded.

Data analysis focused on discharge and follow-up outcomes.

**Results** 7 patients were analysed. The median age was 3.7 years. All were previously well with normal development. All had impaired consciousness at presentation with preceding fever and prodrome. Typical radiology showed bilateral thalamic involvement with/without areas of haemorrhage and necrosis. Causative organisms included Influenza A H1N1, Human Herpes Virus 6 and Metapneumovirus. All were treated with steroids, immunoglobulin or both.

Outcomes were evaluated at discharge and follow-up and divided into good or poor (including death). One passed away from neurology at follow-up.

**Conclusions** ANE mortality at our institution is 14%. Morbidity of survivors at discharge is 100%. Long term follow-up morbidity however, improves to 50% with half achieving normal neurological function at follow up.

**PO-0846 TUMEFACITIVE DEMYELINATING LESIONS IN JUVENILE-ONSET MULTIPLE SCLEROSIS**

1S Menasce, 2S Minon, 3A Fatal, 3A Adhorn. 4Pediatric Neurology Unite Dana Children’s Hospital and Multiple Sclerosis Center, Tel Aviv Medical Center and Sheba Medical Center, Tel-Aviv, Israel; 5Multiple Sclerosis Center, Sheba Medical Center, Tel-Aviv, Israel; 6Pediatric Neurology Unite Dana Children’s Hospital, Tel- Aviv Medical Center, Tel-Aviv, Israel

10.1136/archdischild-2014-307384.1475

**Background** The pathogenesis of large demyelinating lesions is still controversial. Atypical tumefactive demyelinating lesions (TDL) associated with acute inflammation, peri-lesional oedema and gadolinium ring enhancement are infrequently described in patients with juvenile-onset multiple sclerosis (MS).

**Objective** To describe the clinical, imaging and micro-structural metrics of TDLs and chronic MS lesions in patients with juvenile-onset MS.

**Methods** Ten patients diagnosed with MS were analysed for the presence of TDLs and chronic non enhancing MS lesions. The MS lesions were defined by a region of interest encircling the lesion centre on 2–3 consecutive slices. DTI images were acquired along 31 independent orientations using a single shot echo-planar imaging sequence.

**Results** Four patients with 6 TDL, developed acute neurological symptomatology. The two girls presented with acute ataxia and aphasia, and the two boys with severe ataxia. Three patients progressed rapidly to develop seizures, became stuporotic and were admitted to the paediatric intensive care unit. Brain MRI demonstrated six TDLs. Analysis of the whole group (10 patients) disclosed 21 chronic non enhancing lesions. Assessment of DTI metrics of TDL as compared to chronic MS lesions disclosed significant differences.

**Conclusion** TDL are a possible presentation of demyelinating disorders, posing a diagnostic and therapeutic dilemma towards neoplastic lesions. The micro-structural analysis of TDL suggests a severe tissue disruption probably due to the acute inflammatory process and oedema. Our analysis provides metrical tools that together with MR spectroscopy and perfusion may aid to identify accurately TDLs, potentially sparing young patients unnecessary and possibly debilitating brain biopsy.

**PO-0847 WITHDRAWN**

**PO-0848 WITHDRAWN**

**PO-0849 EXPRESSION PATTERN OF BRAIN SPECIFIC MIR-124, MIR-134, AND MIR-9 IN AN IMMATURE RAT MODEL AND CHILDREN WITH MESIAL TEMPORAL LOBE EPILEPSY**

A Omar, S El Sharkawy. Pediatrics and Neonatology, Faculty of Medicine Suez Canal University, Ismailia, Egypt

10.1136/archdischild-2014-307384.1476

**Background and aims** Mesial temporal lobe epilepsy (MTLE) is a particularly devastating form of human epilepsy with significant incidence of medical intractability. MicroRNAs (miRs) are small, noncoding RNAs that regulate post-transcriptional expression of protein-coding mRNAs, which may have key roles in the pathogenesis of MTLE development. We aimed to detect the dynamic expression pattern of brain specific miR-124, miR-134 and miR-9 in the hippocampi of immature rats and children with MTLE.

**Methods** To study the dynamic expression pattern of brain specific miR-124, miR-134 and miR-9, we performed real-time quantitative PCR on the hippocampi of immature rats at 25 days of age. Expression was monitored in the acute, latent, and chronic stages of disease (2, 3 and 8 weeks after induction of lithium-pilocarpine status epilepticus, respectively), and in control hippocampal tissues corresponding to the same