abies 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 NECROTISING ENCEPHALOPATHY IN
CHILDHOOD – EPIDEMIOLOGY, RADIOLOGICAL
FINDINGS AND OUTCOMES

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Background and aims Acute necrotizing encephalopathy in
childhood (ANE) is a disease characterised by acute encephelo-
pathy 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 pro-
drome. Typical radiology showed bilateral thalamic involvement
with/without areas of haemorrhage and necrosis. Causeative organisms
included Influenza A H1N1, Human Herpes Virus 6 and Metapneu-
movirus. All were treated with steroids, immunoglobulin or both.

Outcomes were evaluated at discharge and follow-up and div-
ided into good or poor (including death). One passed away from
brainstem death. All had neurological deficit at discharge: 50%
severely affected; 50% mildly affected; 00% in the former group
progressed rapidly to develop seizures, became stuporotic and were
admitted to the paediatric intensive care unit. Brain MRI demon-
strated six TDLs. Analysis of the whole group (10 patients) dis-
closed 21 chronic non enhancing lesions. Assessment of DTI
metrics of TDL as compared to chronic MS lesions disclosed sig-
nificant 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 inflamma-
tory 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-0846**  TUMEFAC TIVE DEMYELINATING LESIONS IN
JUVENILE-ONSET MULTIPLE SCLEROSIS

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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).

Objectives To describe the clinical, imaging and micro-structural
metrics of TDLs and chronic MS lesions in patients with juve-
neile-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 pro-
gressed rapidly to develop seizures, became stuporotic and were
admitted to the paediatric intensive care unit. Brain MRI demon-
strated six TDLs. Analysis of the whole group (10 patients) dis-
closed 21 chronic non enhancing lesions. Assessment of DTI
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