contributing factor .The use of prednisone could be an effective add on therapy in intractable epilepsy.

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NEWBORN CONVULSIONS 2004-2009

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The aim of the study was to introduce epidemiologic data of newborn convulsions and its outcome of our county's population.

Population and method: There were investigated 44 274 lifeborn newborns from 1th of january 2004 to 31th of december 2009 of Borsod County in Hungary. 47 of them had newborn convulsions (C). Terapeutic protocol was: 1st step: Diazepam; 2nd step: Loading phenobarbitone (20 mg/kg/ day);3rd step: Phenytoin(3x10 mg/kg/ 1st day)in some cases chloral-hydrate(25-75 mg/kg/doses maximum 2x/day) then the doses were decreased for maintenance doses.

Follow up examinations were done regularly 1-2 monthly. Statistical methods were mathematical mean, standard deviation and calculation of percentage.

Results: Characteristics of ill patients: gestational age 37,56±1,18 week, birth weight 3008,7±420,23 gram, Apgar 1' 8,19±1,13, Apgar 5' 9,1±0,88,mature/ premature rate 36/11, male/female rate 24/23. The etiology was in a wide range: 13 asphyxia, 6 intrauterin infection, 5 familial,4 patient 4th-5th day convulsions etc.

C incidence was 1,06/1000 lifeborn newborns. Status epilepticus (SE) was developed at 10 patients (inc.:0,23/1000). 46,80% of all C was in the first five days. 60% of SE was observed on the 1st-2nd days. 3 babies died but nor in the early period neither as a consequence of C and SE.

EEG abnormality was observed one third of C patients. Every cases of SE had disorders of EEG. 11 epilepsies were observed, 7 of them had been transient, 2 turned into terapy resistant epilepsy.

Conclusion: There were less convulsions and status epilepticus than in the literature. Classical terapeutic protocol was useful in the most cases.

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PREVENTABLE NEUROREGRESSION-CASE REPORT AND LITERATURE REVIEW OF VITAMIN B12 DEFICIENCY

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Background: Vitamin B12 deficiency in infancy is a cause of a preventable neuroregression and complex neurological manifestations.

Case report: An 18 month male infant was admitted from an OP Community Paediatrician's clinic for gross pallor. The parents reported concerns regarding the darkening of his skin, losing motor skills and apathy going back 3-4 months. He was still exclusively breast fed although weaning was attempted on various occasions. He had gross pale, puffy hands and feet, hyper-pigmentation, tremors, apathy, variable tone and was developmentally behind. Investigations revealed pancytopaenia, low vitamin B12, normal ferritin and folate and hypersegmented neutrophils and grossly abnormal MRI His mother was vegetarian and had low B12 yet normal haematology. He had a very stormy course during treatment, developed severe autonomic dysfunction with bradycardia, hypertension, drooling and unsafe swallow on initiating treatment and had to be transferred to PICU. He eventually demonstarted increasing attention and motor abilities and discharged with follow up in clinic and CDC.

Discussion: There are various reports in literature which describe the many different neurologic manifestations in deficiency and during treatment in an exclusively breast fed infant with maternal B12 deficiency although not all of these are described in a single case.

Conclusions: Infantile B12 deficiency from maternal deficiency can cause permanent neurological damage. There should be better awareness during the antenatal period and in the prime care to avoid neonatal and infantile B12 deficiency. Can selective neonatal screening prevent the catastrophy? Treatment can result in paradoxical deterioration and therefore needs close monitoring.