ID: 929
TITLE: ANTENATAL MAGNESIUM SULPHATE TREATMENT FOR FETAL NEUROPROTECTION: SHORT TERM AND LONG TERM OUTCOMES IN A TERTIARY NICU
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CONTENT:

Antenatal magnesium sulphate treatment was identified as one of the most effective interventions in reducing the incidence of intraventricular hemorrhage (IVH), periventricular leukomalacia and cerebral palsy among preterm infants (1). The aim of our study was to compare rates of IVH and cognitive outcomes, in preterm infants who received antenatal magnesium sulphate with those who did not.

This retrospective study included a cohort of preterm babies (n=90) born between 23 and 32 weeks of gestation (23-25+6, n=7; 26-28+6, n=26; 29-32, n=57) from January to December 2015 in a single UK tertiary neonatal centre. The treatment group n=38 (18 male) had a median (range) gestation of 29+6 (23+5 - 32) weeks, birth weight of 1074 (550-1716) gms and the non-treated group (n=52; 23 male) had a gestation of 29+4 (23+3- 32) weeks, birth weight 1168 (490 – 1874) gms. Eighty-five infants (94%) received antenatal steroids. The short term outcome was assessed by cranial ultrasounds on day 1, 3 and 7 of life. The highest grade of IVH was recorded. Cognitive outcomes at the corrected age of two years were assessed by the Bayley Scales of Infant Development.

In the treatment group, IVH grade 1 was reported in 6 babies (15%) while the remaining 35 babies (85%) had normal head scans. Among the non-treated group, 28 (54%) were reported to be normal, 14 (27%) with IVH Grade 1, 8(15 %) with IVH Grade 2; 1(2%) with IVH Grade 3 and 1(2%) with IVH Grade 4. A two way ANOVA showed a statistically significant interaction between the effects of magnesium sulphate exposure and gestation on IVH Grades on Cranial USS, (F (2, 84) = 7.816, p=0.001) (Figure1). Incidence of any IVH was significantly higher in the non-treated group compared to those treated across the gestational age range (p=0.021).

The mean (SD) Bayley composite cognitive scores in the treated group 10.75(2.217) were not significantly different compared to the scores in the non-treated infants 8.5(3.659), (p=0.279). None of the babies had a diagnosis of cerebral palsy at 2 year follow up.

In this cohort of preterm infants, antenatal magnesium sulphate treatment was associated with a significant reduction in the incidence of intraventricular haemorrhage but no significant differences in 2 year cognitive outcomes.

Reference:
Antenatal magnesium sulphate treatment for fetal neuroprotection: Short term and long term outcomes

COI: None declared
ID: 671
TITLE: IMPAIRMENT OF DENDRITOGENESIS OF CORTICAL PYRAMIDAL NEURONS IN POSTNATAL MICE BY OXYGEN TOXICITY AND PROTECTION BY ERYTHROPOIETIN
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CONTENT:
Preterm birth is associated with a higher risk for neurological disorders, e.g. autism and others, which correlate with altered dendritogenesis. The increase of O2-levels in preterm infants caused by premature change from hypoxic fetal life in utero into room air environment can affect the developmental program of the brain. So far there is no effective pharmacological therapy available for preterm infants to prevent neurological disabilities. However, preclinical studies have demonstrated neuroprotective properties of erythropoietin (EPO). We investigated whether dendritic growth of cortical pyramidal neurons is inhibited by oxygen and whether rEPO is useful for protection.

Five-day-old healthy C57BL/6-mice were divided into three groups: A) exposure to 80% O2 for 48 h, B) parallel to hyperoxia-exposure, administration of 3 injections 5000 I.U./kg bw rEPO s.c. respectively (at the beginning, after 24 h and 48 h), C) untreated control. To analyze dendritic morphogenesis of cortical pyramidal neurons of cingulate, motor and sensory cortex, brains were impregnated with Golgi-Cox staining at postnatal day 23 (P23) and histologically prepared to perform Sholl-analysis with ImageJ-software. For Sholl-analysis 30 concentric circles with a distance of 10 µm each were put centrally around the soma and basal and apical dendrites were counted.

Hyperoxia caused a significant reduction of dendritic branching of basal dendrites of cortical pyramidal neurons in cingulate cortex within a radius of 30 and 50 µm (p<0.05). A similar reduction was revealed in superficial layers of motor cortex within a radius of 30-80 µm (p<0.01) at basal dendrites, of 40 µm (p<0.05) at apical dendrites and in deep layers a radius of 30-110 (p<0.05) at basal dendrites and of 40-80 and 110-140 µm (p<0.05) at apical dendrites. The sensory cortex has shown significant reduction of dendritic branching in superficial layers within a radius of 10-90 µm (p<0.05) at basal dendrites, a radius of 60 µm (p<0.01) at apical dendrites and in deep layers of 10-80 µm (p<0.05) at basal dendrites and of 70 µm (p<0.05) at apical dendrites. Pyramidal neurons treated with rEPO have shown a selective normalization of dendritic branching in all investigated cortical areas.

Neonatal hyperoxia leads to a significant inhibition of dendritic growth of cortical pyramidal cells in cingulate, motor and sensory cortex. Administration of rEPO prevents the damage on dendritogenesis and may thus be useful for prevention of associated neurological problems in preterm infants.

COI: None declared