286

STAFF PERCEPTIONS OF BARRIERS TO PEDIATRIC END-OF-LIFE CARE IN INTENSIVE CARE VERSUS NON-INTENSIVE CARE SETTINGS

A HUGHES[1], JC PARTRIDGE[1], B DAVIES[1], BA COOPER[1], RF KRAMER[1], A AMIDI-NOURI[1], SA SEHRING[1] [1]UNIVERSITY OF CALIFORNIA SAN FRANCISCO (USA)

Background: In the US, 55,000 children die annually, mostly in intensive care units and rarely receiving palliative care. Barriers to palliative care for children have not been well studied.

Methods: Our goal was to describe providers' perspectives on barriers to optimal end-of-life care for children. With IRB approval, we surveyed pediatric providers in a children's hospital using a confidential questionnaire asking demographics, practice characteristics, and perceptions of specified barriers to palliatve care for chidren (as Likert scales). We compared perceptions across settings, medical acuities, and children's ages using non-parametric statistics. Results: Of 229 questionnaires, 22% were copleted by physicians, 49% by nurses. 65% of providers work in intensive care. 31% of deaths occurred in patients aged <2 months. The most frequently perceived barriers were uncertain prognosis, acceptance of fatal prognosis, time and staffing constraints, language and cultural differences, and family preference for more aggressive treatment. More non-ICU providers than ICU providers reported time constraints ($p=0.001$) and staff shortages ($p=0.003$) as barriers. Non-ICU providers more frequently perceived parent discomfort with withholding/withdrawing hydration or nutrition than ICU providers ($p=0.01$). Non-ICU providers more frequently preferred more aggressive life-support than parents thought approriate ($p=0.009$).

Conclusions: Providers report more barriers to end-of-life care in non-intensive settings than in intensive settings. This may relate to higher patient loads, limited resources, or lack of organizational support for providers in less acute hospital wards where death is a less comon event. Non-intensivists may not experience the burden of interventions that intensivists witness and may advocate for more aggressive treatment than family members think appropriate. These differences in perceptions of barriers to care may help identify ways to improve end-of-life care for patients as well as ways in which to support pediatric providers as they care for dying children.

287

HYPEROXIA DECREASES, INJURY CAUSED BY PHORBOLESTER INCREASES VEGF EXPRESSION IN THE BRAIN OF NEWBORN RATS

C PEISER[1], U FELDERHOFF-MUESER[1], P KOEHNE[1], M OBLADEN[1] [1]DEPARTMENT OF NEONATOLOGY, CHARITÉ - VIRCHOW CAMPUS (GERMANY)

Objective: Vascular endothelial growth factor (VEGF) expression promotes angiogenesis and neuronal repair. The aim of this study was to investigate VEGF expression in relation to neurodegeneration, either caused by hyperoxia, a frequent problem in the treatment of preterm infants with respiratory dysfunction, or caused by brain injury, an important complication (e. g. in case of intracerebral haemorrhage) in the early neonatal period of immature newborns.

Methods: 6 d old Wistar rat pups were either subjected to 80 % oxygen or treated with phorbolester. After 0, 2, 6, 12 and 24 h of exposition animals ($n = 5$ per group) were sacrificed and the brain cortices were processed for molecular experiments on RNA (quantitative RT-PCR) and protein (Western blot) levels using VEGF specific primers and antibodies.

Results: In association with hyperoxia a decrease in VEGF mRNA expression down to about 25 % of the basal level of the control animals (no additional oxygen exposition) was noted. This effect was already evident at 2 h with a minimum of VEGF mRNA expression at 2 h and 6 h ($p < 0.01$). The protein expression also showed a decrease of VEGF signal associated with hyperoxia. The weakest signal appeared at 12 h ($p < 0.01$). In the case of phorbolester treatment we have seen an increase of VEGF expression up to about 200 % compared to the controls, with a maximum of mRNA at 6 h ($p < 0.01$) and a maximum of protein expression at 12 h and 24 h ($p < 0.05$).

Conclusion: Exposure of newborn rats to hyperoxia induces a statistically significant time-dependent decrease, treatment with phorbolester a statistically significant time-dependent increase in VEGF expression in the cortices of the animals. These effects are evident on the transcriptional and translational level.

288

THE EFFECT OF SECOND COURSE OF ANTENATAL BETAMETHASONE (BM) ON NEONATAL MORBIDITY IN PRETERM INFANTS: A RANDOMIZED TRIAL

OM PELTONIEMI[1], MA KARI[2], E HALMESMÄKI[2], T RAUDASKOSKI[1], O TAMMELA[3], J UOTILA[3], L LEHTONEN[4], U EKBLAD[4], K HEINONEN[5], S HEINONEN[5], S ANDERSSON[2], P JOUPPILA[1], M HALLMAN[1] [1]UNIVERSITY HOSPITAL , [2]UNIVERSITY CENTRAL HOSPITAL, [3]UNIVERSITY HOSPITAL, [4]UNIVERSITY CENTRAL HOSPITAL, [5]UNIVERSITY HOSPITAL (FINLAND)

Background: Single course of BM decreases neonatal morbidity when administrated within 7 days before preterm delivery. The benefits of second course of BM are unknown.

Aims: To evaluate the efficacy of second course of BM in increasing the intact survival of preterm infants.

Methods: In this randomized, blinded trial, pregnant women (before 34.0 gestational weeks) were eligible if they had remained undelivered more than 7 days after antenatal BM. Single dose of BM (12 mg) or placebo was given, when the preterm delivery was anticipated. Primary outcome was intact survival without respiratory distress syndrome and/or intracerebral hemorrhage (grade 3–4).

Results: 251 mothers participated the study. All infants were delivered prior to 35 weeks of gestation. At present the outcome of 285 infants has been analyzed. The intact survival was 48% in BM and 58% in placebo groups ($P=0.1$). The intact survival was non-significantly higher in BM group (58% vs. 41%, $P=0.3$), when the study drug was administered more than 24 h before birth (group 1). The incidence of intact survival was lower in BM group (44% vs. 61%, $P=0.01$), when the intervention took place less than 24 h before birth (group 2). In group 1, the risk of patent ductus arteriosus was 16% in BM and 41% in placebo group ($P=0.04$), vs. 29% and 27% in group 2. In group 2, systolic and diastolic blood pressures were increased in BM arm ($P=0.02$ and $P=0.03$). In group 1, blood pressure levels were similar between study groups. The second course of BM did not influence the acute outcome of preterm infants. The preterm infants benefited from antenatal BM, when treatment was given more than 24 h before delivery. BM given less than 24 hours before delivery was associated with increased blood pressure after birth and with a decrease in intact survival.

289

CONTAINMENT OF TWO OUTBREAKS OF SERRATIA MARCESCENS IN A NORTHERN ITALY NICU

G. PEROTTI[1], M.E. BERNARDO[1], C. PENNATI[1], M. BARBARINI[3], L. PAGANI[2], M. STRONATI[1] [1]DIVISIONE DI PATOLOGIA NEONATALE E TERAPIA INTENSIVA, I.R.C.C.S. S. MATTEO , [2]DIPARTIMENTO DI SCIENZE MORFOLOGICHE EIDOLOGICHE E CLINICHE, SEZIONE DI MICROBIOLOGIA, UNIVERSITÀ DI PAVIA, [3]DIVISIONE DI PATOLOGIA NEONATALE AZIENDA OSPEDALIERA S.ANNA COMO (ITALY)

S. marcescens is a recognized nosocomial pathogen especially in NICU. We describe the course and related measures of containment of two outbreaks (I-II) of S. marcescens in a 30-bed NICU. Two patients developed sepsis by S. marcescens in June 2003 following this active surveillance was established to detect infected/colonized patients and the source of the outbreak. During the outbreak period the NICU remained open to new admissions. Infected and colonized patients were cohorted in an infected area. Hygienic measures were strictly enforced for unit staff and visitors. All infants received probiotics daily. Isolates were genotyped with Pulsed Field Gel Electrophoresis (PFGE) protocol. Colonized patients were not treated with antibiotics and were discharged soon. In the period June-August 2003, 54 newborns were screened for S. marcescens, 17 (31%) infants were found colonized by S. marcescens in the digestive tract. In the same period 2 colonized patients developed sepsis from different germs (SGB, Candida albicans). In June 2004, 10 months after the first outbreak, a new fatal case of S.marcescens sepsis occurred in a VLBW in the same NICU. The following control measures revealed three (9%) colonized neonates in the Unit. By PFGE typing we identified one epidemic clone (A) in 15 infants plus the index case and two other unrelated clone (B-C) in 2 patients in outbreak I. In outbreak II one epidemic clone (D) in 3 infants and an unrelated clone in an other patient were identified. The comparative PFGE analysis excluded any relationship between the first and the second episode of sepsis and confirmed that S. marcescens was not endemic. After 6 and 2 weeks respectively the outbreaks were eradicated without any recurrence. The administration of probiotics and avoiding unnecessary exposure to antibiotics may have played an important role in the containment of the outbreak

290

NEONATAL THYROTOXICOSIS: PATHOGENESIS, CLINICAL FEATURES AND DIAGNOSIS

E. MAZZONI[1], A. PETRACCA[1], M. CAPELLI[1], M. MASTROCOLA[1], S. GUALDI[1], A. CASSIO[2], G. COCCHI[1] [1]INSTITUTE OF NEONATOLOGY , [2]PAEDIATRIC CLINIC (ITALY)

Introduction: Neonatal Thyrotoxicosis (1:4000) is caused by the transplacental passage of thyroid stimulating immunoglobulins (TSIs) from mothers with Basedow disease/Hashimoto thyroiditis. Hyperthyroidism is characterized: by growth retardation, goitre, tachycardia, hydrops, craniosinostosis, increased foetal motility, accelerated bone maturation in the foetus; tachycardia, arrhythmia, periorbital oedema, irritability, poor weight gain in the newborn. Symptoms may be observed at birth or after several days.

Objectives and methods The objectives of our study were to evaluate the incidence of thyroid disease during pregnancy and suggest a guide-line for the management of baby at risk. The study was performed between January 1999 and December 2004 in mothers with Basedow disease. The period of maternal diagnosis, the medical treatment, the TSIs level at the delivery were considered. For every neonate we observed: the clinical signs, the level of thyroid hormone and antibodies; ecography of thyroid and of encephalon, X-ray of carp.

Results: We detected 9 newborns (1/2459) all preterm. Babies of mothers without treatment were hospitalized for the cardiac risk. In babies of treated mothers, clinical signs appeared between 10 -15 days of life. TSIs level of mothers at delivery was well above the upper limit. However babies had a resolution from an endocrinological and clinical point of view and a good prognosis for neurological and psychological development.

Conclusions: It is important to recognize and treat Basedow disease in mothers as soon as possible. It is necessary: to consider euthyroid or hypothyroid mothers with a high level of TSIs; to measure TSIs levels in the third quarter of pregnancy and at the delivery; to screen the foetus with a sonografic approach during the pregnancy. For babies at high risk of thyrotoxicosis, the neonatal observation and the measurement of TSH, fT4 TSIs at birth, at age 2–7 days and at 10–14 days are important.

291

ORBITAL MYOSITIS PRESENTING AS CLUSTER HEADACHE

R PIANIGIANI[1], GROSSO S[1], GALLUZZI P[2], CERASE A[2], BUONOCORE G[1], MORGESE G[1], BALESTRI P[1] [1]DEPARTMENTO OF PEDIATRICS , [2]DEPARTMENT OF NEURORADIOLOGY, AZIENDA OSPEDALIERA SENESE (ITALY)

Background. Cluster headache affects men in the third decade of life. Episodes centers in and around eye, start acutely, last 1 to 2 hours, and occur several times a day. Associated features are ipsilateral conjunctival hyperemia, lacrimation, ptosis, miosis, and facial sweating. Orbital myositis belongs to the group of idiopathic orbital inflammatory syndromes. Clinical picture involves proptosis, conjunctival hyperemia, lid edema, and painful eye movements.

Case report. We report on a 13 year-old girl presenting with recurrent and alternating orbital myositis mimicking a cluster headache. At the age of 10 years she first experienced an episode of ocular pain, diplopia, and palpebral edema involving the right eye. Lacrimation, conjunctival hyperemia, nasal rhinorrhea and headache were also observed. The episode lasted 1 month. Neurological examination was normal. Further four episodes affecting the right and the left eyes, alternatively, occurred during a period of 18 months. Cluster headache was initially diagnosed. Neither FANS nor tryptans were able to control clinical symptoms. Magnetic resonance imaging of the orbits showed evident edema and thickening of the correspondent rectus lateralis which subsided after corticosteroids. Idiopathic ocular myositis was therefore diagnosed. The following investigations were performed in several occasions and resulted normal or negative. They included: (i) levels of complement factors and of immunoglobulins; (ii) Raitest and Waaler-Rose test; (iii) research of antinuclear antibody, antibody to ds-DNA, antibodies to extractable nuclear antigens (RNP, Sm, SS/A, SS/B), antisynthetasis (Jo1), antineutrophil cytoplasmic antibody (IFI, antiproteinase 3, antimyeloperoxidase, antilactoferrin). Thyroid function was normal. Viral and bacterial infections were also excluded. Orbital myositis may clinically mimic cluster headache.

Conclusions. Orbital myositis should be considered in those patients with atypical cluster headache signs such as painful eye movements and ocular pain which do not resolve after 3–4 hours. An early diagnosis is necessary to plan a proper therapeutic strategy.