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LACTOBACILLUS REUTERI ATCC 55730 VERSUS SIMETHICONE IN THE TREATMENT OF INFANTILE COLIC: A PERSPECTIVE RANDOMISED STUDY

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Several clinical studies have documented the efficacy of probiotic bacteria, including Lactobacillus reuteri (L. reuteri) in the prevention and treatment of gastrointestinal disorders. In colicky infants an imbalance in the microflora with low counts of lactobacilli has been demonstrated which may be part of the etiopathogenesis of this disorder.

Objective: The aim of this perspective randomised study was to investigate the efficacy and side effects profile of L. reuteri ATCC 55730 in the treatment of infantile colic. Methods: 49 breastfed colicky infants, diagnosed according to Wessel's criteria, aged 21 to 90 days, were randomly assigned to 2 treatments, one with the probiotic L. reuteri (P; 100.000.000 CFU/day) and the other with simethicone (S; Mylicon 6 mg/kg), once a day for 28 days. A questionnaire was given to the parents to monitor daily the crying time and side effects.

Results: 46 infants completed the trial: 23 in the P group and 23 in the S group. The infants were similar regarding gestational age, birth weight, and gender ($p > 0.05$). At baseline the daily average crying time in the P group was 210 min/day (SD 31) vs 208 min/day (SD 24) in the S group ($p = 0.800$). After 7 days, crying time was significantly reduced in the P group: 140 min/day (SD 51) vs 172 min/day (SD 43) ($p = 0.025$). By day 28, the crying time was significantly reduced in L. reuteri infants to only 20 min/day (SD 14) compared to simethicone, 156 min/day (SD 24) ($p < 0.005$). No side effects were observed in either group.

Conclusions: Our findings show that in breast-fed infant colic symptoms were significantly improved within one week of treatment with the probiotic Lactobacillus reuteri demonstrating the health advantages of L. reuteri to standard therapy in these infants.

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NEONATAL ALLOIMMUNE THROMBOCYTOPENIA: LABORATORY FINDINGS AND CLINICAL MANAGEMENT IN 38 CASES

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Neonatal alloimmune thrombocytopenia (NAITP) is a rare and transient disease caused by maternal IgG antibodies that react against the paternally inherited antigens expressed on fetal platelets (PLT). Screening and identification of antibodies in maternal blood sample is the main support in the diagnosis of NAITP. We report 38 cases of NAITP referred over the past 5 years. Immunohematologic study on mothers involved: 1) IgG antibody screening by a solid phase red cell adherence methodology; 2) identification of offending antibody carried out by ELISA; 3) HPA 1, 2, 3, 4, 5, 6 and HLA genotype of both parents and neonates through by PCR-SSP technology. The study protocol on infants included: 1) PLT count, 2) clinical examination, 3) detection of antibodies bound to PLT, 4) ultrasound screens. NAITP was clinically suspected in 50 infants, born by first pregnancy. Only 38 maternal sera were reactive with the following specificity: 8 anti HPA-1a, 2 anti HPA-1b, 2 anti HPA-1a + HLA, 2 anti HPA-3a, 6 anti HPA-5b, 17 anti HLA, 1 auto HPA-5b. Specificity of HPA antibodies was confirmed by HPA genotype of parents. The infants with HPA-1a and HPA-1b immunization suffered from severe and symptomatic NAITP (bleeding and petechiae; PLT 7–12 x 103/ml). They were treated with transfusion of PLT compatible with the maternal alloantibody (10–20 ml/kg) and administration of high doses of intravenous immunoglobulin. On the contrary, NAITP due to HPA-3a, HPA-5b and HLA immunization was characterized by mild and self limiting thrombocytopenia (PLT 50 to 60 x 103/ml); no therapy was administered. No infant had intracranial hemorrhage. Neurodevelopmental outcome was good. Our results confirm that in the caucasian population HPA-1 (1a, 1b) polymorphism is most frequently involved in NAITP (10 out of 38 cases). Early diagnosis and adequate postnatal treatment may be successful in NAITP in the first pregnancy.

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NEONATAL PANHYPOPITUITARISM, AGENESIS OF THE CAROTID ARTERY AND MIDLINE CNS ABNORMALITIES

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Background: Panhypopituitarism is a condition characterized by inadequacy or absence of all anterior pituitary hormones due to a variety of disorders, resulting in the destruction of substantially all the anterior pituitary gland. The lack of pituitary hormones causes a reduction in the normal functioning of the endocrine glands.

Case report: Preterm baby, born at 37 weeks of gestation after emergency caesarean section for acute fetal distress. Apgar at 1 min= 4, at 5 min= 6; intubated and admitted to NICU, assisted ventilation for RDS. Clinical signs at birth: systemic hypotension, hyponatremia, craniofacial dysmorphism (right microphtalmia, right preauricolar tags, right face hypoplasia, cleft palate and bifid uvula). Additional features: patent ductus arteriosus, pneumothorax, pulmonary hypertension, persistent jaundice. Low levels of T4 and TSH were detected at neonatal screening. Substitutive therapy with oral L-tiroxina, hydrocortisone and subcutaneous GH was started and normalization of hormonal profile was followed.

Clinical and laboratory investigations: Low levels of FT4 (0,38 ng/ml), TSH (0,13 mUI/l) and basal cortisol (0,35 ng/ml) were detected. Levels of ACTH, GH and PRL were undetectable. Abdominal ultrasonography, eye fundus examination, auditory screening were normal. Brain RMI and angio-RMI showed adenohypophysial agenesis with ectopical neurohypophysis and agenesis of internal right carotid artery.

Discussion: We describe a newborn with panhypopituitarism, agenesis of the carotid artery and midline CNS abnormalities. This report supports the theory of an embryologic defect as the cause of the pituitary insufficiency. Indeed during embryogenesis the internal carotid artery and the pituitary gland develop in the 4th embryonic week. A possible relationship exists between internal carotid artery agenesis, pituitary aplasia or hypoplasia and midline CNS abnormalities.

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CHOOSING STRATEGY FOR PREVENTION OF NEONATAL GROUP B STREPTOCOCCAL (GBS) INFECTION - HAS MATERNAL ORIGIN COUNTRY TO BE CONSIDER?

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Background: During the last two decades our maternal GBS carrier and neonatal sepsis rates has varied, 3.5%–11% and 0.2–0.9/1000 LB respectively. Thus, Israeli current policy is Risk Factor Approach with no routine GBS screening.

Objective: 1. To update colonization & attack rate. 2. To determine whether our Risk factor Approach is still applicable.

Design/Methods: In Jan.-June 2002 we randomly sampled women admitted for delivery. Vaginal & rectal cultures were obtained before 1st pelvic examination. GBS was isolated using a selective broth medium and identified by latex agglutination and antigen B assay. Neonatal sepsis/meningitis rate was defined as positive blood/CSF cultures in 2002. Chi2 and multivariate analysis were performed.

Results: Of the 637 sampled women 94 were GBS positive (14.7%). Fifty (7.8%) of the mothers were from N. America origin and there colonization rate was 26% (13:50) as opposed to mothers of other origins which was 13.8% (81:587) $p = 0.02$. In multivariate analysis, including variables of maternal age, parity, blood type, duration of rupture of membranes, infant gender and birth weight, N. America Origin was found to be the single risk for GBS carrier ($p = 0.0273$). Neonatal sepsis (8:9995) rate was 0.8/1000 LB for the entire population. The percentage of the N. America Origin mothers in the sampled population was 7.8% (50/637) their component in the GBS infected group was 37.5% (3:8) $p = 0.016$. No difference was noted in GBS serotypes between origin groups.

Conclusions: We identified N America origin women as an increased risk factor for GBS colonization in the Israeli society. The high N American origin neonatal GBS sepsis rate was correlated with the higher colonization rate in these mothers. Given our high carrier & attack rate in N. America origin women & babies we now recommend routine prenatal screening in that ethnic subgroup.

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THIRD VENTRICLE ENLARGEMENT IN INFANT WITH TRISOMY 21 - A NEW FINDING.

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On routine head sonography of babies with trisomy 21 (T21) we noticed that the third ventricle (3V) seemed to be larger than expected.

Objective: To determine whether the size of the 3V, as observed on head ultrasound is statistically different in babies with and without T21.

Methods: Measurements of routine head sonography of all term babies with T21 were done within seven days of birth and were compared to those of randomized term infants without T21. Studies were done with 7.5 probe through the anterior fontanel. Statistical significance was calculated by t test and defined by a $p < 0.05$.

Results: 65 babies were evaluated, 44 with T21 and 21 infants without T21. Although both groups were of similar gestational age (39+/-1 vs. 39+/-1 weeks), babies with T21 were generally smaller (3.075+/-0.361 vs. 3.375+/-0.399 kg; $p = 0.004$) with smaller head circumferences (H.C.) (33+/-1.3 vs. 34.9+/-1.7 cm; $p = 0.001$). Despite the smaller overall head circumference (see table below), both the width and the length of the 3V were enlarged in the T21 babies. For comparison, size of the lateral ventricles were similar in the two groups.

	Rt Vent	Lt Vent	3V Length	3V Width	
T21 (n=44)		0.15+/-0.06	0.17+/-0.1	0.90+/-0.26	0.27+/-0.08
Ctrl (n=21)		0.13+/-0.04	0.15+/-0.05	0.63+/-0.3	0.19+/-0.06
Sig	0.18	0.4	0.001	0.001	

Discussion: Our data present an isolated finding of enlarged 3V in infants with T21. When viewed in the context of a smaller overall H.C. in these infants and a lateral ventricle size that is similar to that of the control infants, this finding is even more intriguing in that it does not appear to represent a generalized brain atrophy. Further research should be done in order to assess possible maldevelopment of brain structures surrounding the 3V, as well as the clinical and prognostic implications of these findings.

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INCREASED EXPRESSION OF ANTI-INFLAMMATORY IL10 AND IL10 RECEPTOR IN THE NEWBORN RAT BRAIN CAUSED BY HYPEROXIA

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Background: In neonatal medicine, oxygen is frequently used in the treatment of respiratory diseases, and preterm infants are generally exposed to relative hyperoxia compared to in-utero conditions. Recently, oxygen has been identified to cause apoptosis in the brain of newborn rats and rodents. Therefore, it may contribute to the development of cerebral palsy and neurocognitive defects often seen in preterm infants. Pro-inflammatory cytokines, i.e. IL-1b and IL-18, have been proven to be involved in neuronal apoptosis caused by hyperoxia. In contrast, IL-10 is an anti-inflammatory and anti-apoptotic cytokine that may have neuro-protective properties.

Methods: We analysed expression of IL-10 receptor (IL-10R) and IL-10 in cortex and thalamus of six day old newborn Wistar rats (P6) exposed to 80% oxygen for 0, 2, 6, 12, 24 and 48 hours. mRNA levels were determined by semiquantitative reverse transcription polymerase chain reaction, and protein expression was analysed by immunoblotting.

Results: Compared to rats not exposed to hyperoxia, mRNA of IL-10 and IL-10R increased markedly during oxygen exposure with peak levels at 6 and 24 hours, respectively (IL-10R: $p < 0.01$, IL-10: $p = 0.01$). A four-fold upregulation of IL-10R protein was found at 24 hours.

Conclusions: Expression of anti-inflammatory IL-10 and IL-10R in the brain of newborn rats is upregulated during exposure to neurotoxic doses of oxygen. IL-10 may act as an internal neuroprotective agent against hyperoxia-induced cell death.