Patients and methods: Thirteen (13) healthy preterm neonates [birth weight (BW): 1740g (1500-2200), gestational age (GA): 32,5wk (31-34)] and 13 healthy term neonates [(BW: 2960g (2500-4310), GA: 37,5wk (37-39)] were studied. Six (6) healthy adults were used as controls. Peripheral blood samples were obtained from neonates during the 2nd and 3rd weeks of life. IL-17 levels were measured in the serum by ELISA.

Results: Serum IL-17 levels did not differ between preterm and term neonates. However, IL-17 levels were significantly lower in both preterm and term neonates, as compared to those of adults ($p > 0,001$ for both terms and preterms).

Conclusion: Our data reveal dramatically decreased IL-17 release in the serum of neonates compared to adults, mirroring the immaturity of the neonatal immune responses. Importantly, our findings suggest that deficient IL-17 release in neonates may hamper their host defense against extracellular pathogens often leading to overwhelming septicemia and death.

851

HEREDITARY ANGIOEDEMA AND LONG-TERM PROPHYLAXIS

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Hereditary angioedema is a rare autosomal dominant disease characterized by recurrent episodes of angioedema caused by a quantitative or functional defect of the plasma protein C1 esterase inhibitor (C1 INH). Treatment is divided into short and long-term prophylaxis with androgens, antifibrinolytics and C1 inhibitor replacement.

We present the clinical characteristics of three female patients, two of them members of the same family and one isolated case. Eleven year old and 5 year old sisters were admitted to our hospital with complaints of relapsing skin swelling. In the second patient abdominal pain attacks were also developed. The third patient, a 13 year old girl was suffered from recurrent bouts of swelling most often affecting extremities and genitalia since 7 year old. We analyzed blood levels of C3, C4, and C1 INH. Both of the sisters had decreased serum levels of C4 and C1 INH and were diagnosed as hereditary angioedema type I. The third patient had reduced serum levels of C4 and normal C1 INH and the

genetic analysis on the C1INH gene showed a mutation of aa 444 Arg substituted by Cis consistent with type II hereditary angioedema. All the patients were started on tranexamic acid for long term prophylaxis but in the first and second patients the treatment had to change with danazol due to the lack of enough response to the tranexamic acid. The third patient presented occasional (1-2 mild episodes per year) peripheral angioedema and her disease was controlled with tranexamic acid.

852

UNDER AGAROSE MIGRATION ASSAY - A METHOD TO STUDY GRADED CHEMOTACTIC ACTIVITY OF LEUKOCYTES IN NEWBORN INFANTS

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Background: Preterm newborn infants are at risk of developing chronic inflammation and tissue injury in their lungs and CNS due to prolonged and exaggerated accumulation of leukocytes. **Aim:** To study migration of leukocytes from newborn infants with a modified under agarose cell migration assay.

Methods: Blood was collected from healthy adults (n=9) and from healthy, term newborn infants (umbilical cord; caesarean section; spinal anaesthesia; n=9). Isolated leukocytes were added to outer wells and chemoattractants to central wells. Gels were incubated for 2 hours whereby only polymorphonuclear (PMN) leukocytes migrated. Dose responses of intermediate (n=4; IL-8) and end-target (n=5; fMLP) chemoattractants were tested on PMN.

Results: Similar dose response curves of PMN migration to gradients of IL-8 and fMLP were observed in newborn infants as in adults, but with less PMN migrating with newborns than with adults. The PMN leukocyte migration per distance in newborns was 80% of adults with IL-8, and >90% of adults with fMLP. There was a high linear correlation between the number of PMN and the distance of migration irrespective of chemoattractant studied (IL-8: $r=0.85$ and $r=0.65$ respectively for newborns and adults; $p < 0.001$ for both) (fMLP: $r=0.76$ and $r=0.87$; $p < 0.001$). The pattern of migration differed between fMLP and IL-8.

Conclusion: Similar graded chemotaxis of PMN was observed with under agarose cell migration assay in newborn infants as in adults, making this an attractive method for functional clinical studies of leukocytes in newborn infants during different stages of maturity and disease.

853

AUTOIMMUNE DISEASE IN CHILDREN WITH DEFICIT IN IMMUNOGLOBULIN A

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Introduction and objectives: The immunodeficiencies (ID), by the subsequent impairing of the immunoregulation, may be at the origin of certain autoimmune diseases (AID). The relationship between the ID and the AID is triggered by opportunistic infections, the latter favorised by the underlying ID. The deficit in immunoglobulin A is one of the most frequent ID associated with AID.

Material: We realized a prospective study on 87 patients diagnosed with AID. We analyzed the initial immune status, for all these patients. For another group of 53 children, diagnosed with selective IgA immunodeficiency, we realized a six year period survey of the level of the T suppressor lymphocytes, the T helper/ T suppressor ratio, and of the presence of auto antibodies: Anti DNA, rheumatoid factor (RF).

Results: 8 patients (8%), from the group diagnosed with AID, were also identified with selective deficit in IgA at the moment of the initial diagnosis. In the group of 53 patients with underlying IgA immunodeficiency, 2 patients developed over the 6 years of the survey, a significative titer of anti DNA antibodies. In one patient the presence of the RF was detected, 4 children presented a decrease of the T suppressor level, with a rise of the immune ratio. None of these patients presented clinical signs suggesting an AID.

Conclusions: The Ig A immunodeficiency may be a risk factor for subsequent AID. There is a higher risk for AID in patients who develop anti DNA antibodies, RF or a persistent decrease of the T suppressor lymphocytes.

854

ALLERGEN SPECIFIC IGE AND TOTAL IGE LEVELS IN PRESCHOOL CHILDREN WITH ACUTE URTICARIA

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Background and aim: The common causes of acute urticaria are viral respiratory infection, foods allergens and medications. The aim was to identify the contribution of foods, viral infections and drugs to induce acute urticaria in relation to specific and total IgE levels.

Methods: The authors present a prospective study included preschool aged children admitted in pediatric clinic for acute urticaria over a 2-year period. The diagnosis of acute urticaria is based on personal medical history and clinical manifestations. Allergen-specific IgE levels were measured for a panel of 11 foods (cow's milk, egg white, whole egg, codfish, peanut, hazelnut, soy, cereal mix, wheat, tomato, citrus mix) and drug (penicillinum). The measurement of specific IgE using serologic assays RAST/EAST (EAST class qualitative range 0-4). Total IgE levels of >100 kUI/L were considered positive.

Results: 38 preschool children with acute urticaria were included in the study. 28 children (73.6%) had respiratory infection associated with antibiotics intake. The duration of urticaria was 5.80+/-2.40 days. Foods ingestion were considered responsible for urticaria in 10 cases (26.4%). The levels of specific IgE were correlated with acute urticaria for eggs (1-4), cow's milk (2-3), peanuts (2-4), codfish (2-4), soybeant (2-4), wheat (1-3), cereal mix (2-4), citrus mix (1-4), tomato (2-3). Total IgE level was elevated in 24 cases.

Conclusions: Infections, foods, drugs induced acute urticaria in children. The correlation of total and specific IgE levels and symptoms was determined. The results indicate that children with higher total IgE and specific IgE levels have a major risk of recurrent urticaria.