



Theme: Pediatric Gastroenterology & Hepatology **Role of Serum autotaxin in causation of pruritis in cholestatic liver disease** (*J Pediatr Gastroenterol Nutr.* 2016;62:530-5).

Pruritis is a common symptom of cholestatic liver disorders but not all cholestatic conditions (with similar bilirubin levels) are associated with intense itching, indicating role of some extraneous factor influencing its causation and severity. This study evaluated autotaxin (ATX), a lysophospholipase, as a potential cause for pruritis in pediatric cholestatic diseases. The study group of 45 children comprised of 14 with pruritic liver disease, 9 with non pruritic cholestasis and 22 healthy controls. Serum ATX activity was significantly increased in pruritic children with cholestatic syndromes in comparison to nonpruritic cholestatic diseases and healthy controls. Authors concluded that the serum ATX activity is associated with itch intensity in children with cholestatic diseases, and that the ATX inhibitors may be useful antipruritic agents in pediatric cholestatic disorders.


 **Probiotics for reducing the risk of necrotizing enterocolitis and sepsis in preterms** (*Lancet.* 2016;387:649-60)

In this multicentric, randomized controlled phase-3 study, 1315 preterm infants (gestational age 23-30 wk) were randomly assigned to receive probiotic (*B. breve* BBG-001) mixed in dilute elemental infant formula or dilute infant formula alone, within 48 hours of birth. Sixty-one infants (9%) in the probiotic group had necrotizing enterocolitis compared with 66 (10%) in the control group (adjusted RR 0.93; 95% CI 0.68, 1.27). Seventy-three (11%) infants in the probiotic group had sepsis compared with 77 (12%) in the control group (adjusted RR 0.97; 95% CI 0.73, 1.29); 54 (8%) deaths occurred before discharge in the probiotic group compared with 56 (9%) in the placebo group (RR 0.93; 95% CI 0.67, 1.30). This well-controlled study with large sample size suggests that there is evidence of no benefit of routine use of this probiotic (*B. breve* BBG-00) for prevention of necrotizing enterocolitis and sepsis in very preterm infants.

 **Can we do away with duodenal biopsy for diagnosing celiac disease?** (*Arch Dis Child.* 2016; 101:172-6)

Celiac disease is now defined as a genetically predisposed autoimmune systemic condition characterized by the presence of a variable combination of gluten-dependent enteropathy, other clinical manifestations, and specific antibodies such as serum anti-transglutaminase IgA (anti tTG-IgA) and anti-endomysium IgA (AEA). Until very recently, biopsy demonstration of villous atrophy was considered essential for diagnosing celiac disease. In

2012, the European Society of Paediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN) proposed a new algorithm that allows diagnosis of celiac disease without a duodenal biopsy in selected, highly symptomatic cases with high titers of serum anti tTG-IgA antibodies, positivity for AEA and for human leucocyte antigen (HLA) DQ2 or DQ8. In this prospective study, children diagnosed with celiac disease without a duodenal biopsy (group 1; n=51), following these guidelines, and children diagnosed with a duodenal biopsy, matched for sex, age and year of diagnosis (group 2; n=92), were enrolled over a 3-year period. All patients were put on a gluten-free diet (GFD), and were followed up for clinical conditions and laboratory testing at 6 months every year since diagnosis (median follow up: 1.9 years). At the end of follow-up, the two groups were statistically comparable in terms of clinical and nutritional status, anti-transglutaminase IgA antibody titers, quality of life, adherence to a GFD, and number of supplementary medical consultations. It appears that diagnosis of celiac disease without a duodenal biopsy has no negative consequences, at least during a medium-term follow-up.

 **Ursodeoxycholic acid for treatment of indirect hyperbilirubinemia in infants** (*J Pediatr Gastroenterol Nutr.* 2016 62:97-100)

Hyperbilirubinemia is a common problem in neonatal period and phototherapy is a proven effective modality of its therapy. The authors investigated the role of oral ursodeoxycholic acid as an adjunct to phototherapy in neonatal hyperbilirubinemia. Eighty neonates were randomized to receive either oral ursodeoxycholic acid (10 mg/kg/d in 2 divided doses; n=40) in addition to phototherapy or only phototherapy (n=40). Total serum bilirubin levels were estimated every 12 hours, till it was below 10 mg/dL. The two groups were then compared with regard to bilirubin levels at different time points and duration of phototherapy using the generalized estimating equation (GEE) test. The mean (SD) of total bilirubin in the intervention group was 12 (1.6), 10.9 (1.1), and 9.8 (0.2) mg/dL 12, 24, and 48 hours after the beginning of phototherapy, respectively. On the contrary, these measures were 14.4 (1.3), 12.5 (1.4), and 10.1 (1.1) mg/dL in the control group, respectively ($P < 0.05$). The mean (SD) time required for phototherapy to decrease the bilirubin level to < 10 mg/dL was 15.5 (6) and 44.6 (13.3) hours in the case and the control group, respectively ($P = 0.001$). Authors concluded that ursodeoxycholic acid has an additive effect with phototherapy in neonates with indirect hyperbilirubinemia.

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