

P323 AN UNUSUAL CASE OF NEONATAL PROLONGED UNCONJUGATED HYPERBILIRUBINAEMIA

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10.1136/archdischild-2019-epa.672

Aim Our aim is to present the case of an Irish neonate who presented to our Paediatric Department with prolonged hyperbilirubinemia as a consequence of breast milk quality and Gilbert's Mutation (UGT1A1*28 mutation).

Methods We describe the clinical features, course of illness and haematological findings, management and the challenges of same and in addition the outcome to date in our patient.

Results An Irish male neonate of non-consanguineous union, with gestation of 36 weeks, unremarkable perinatal history, exclusively breast fed, first came to medical attention at third day of life with DCT-negative unconjugated hyperbilirubinemia requiring phototherapy for two days. He represented with increased bilirubin levels again 3 days later and again at two weeks of age, each time necessitating phototherapy. Systemic examination was normal. Thyroid & liver function tests, full blood count, urine culture, urine for cytomegalovirus and toxicology screen, lactate dehydrogenase were normal. He was fed exclusive expressed breast milk by bottle and was observed to have high volume intake of 275 ml/kg/day, with overfeeding resulting in vomiting. By 5 weeks of age, he had not regained his birthweight and had dropped from the 25–50th to the 0.4th centile. On further work up, his screen for Hereditary Spherocytosis was negative, haptoglobin was low <0.10 g/L, liver ultrasound scan was normal. His UGT1A1 enzyme levels were sent the results showed that he was heterozygote for UGT1A1*28 mutation, indicating carrier status for Gilbert's Syndrome.

Breastfeeding was discontinued, and his weight and hyperbilirubinaemia improved dramatically.

Conclusion Breast-feeding jaundice is traditionally considered as a consequence of inadequate supply, with breast-milk jaundice secondary to increased recirculation of bilirubin due to deconjugating breast-milk enzymes. This case was unusual in that supply was established to be adequate, rather the nutritional quality of the breast-milk was theorised to be suboptimal, secondary to severe social stressors affecting mother. This, in combination with Gilbert's syndrome status, resulted in moderate prolonged unconjugated hyperbilirubinaemia and failure to thrive in this infant.

It may be helpful for Paediatricians to maintain an index of clinical suspicion for the mutation in children with refractory prolonged unconjugated hyperbilirubinemia. We rarely advocate breast-feeding discontinuation, however in this instance it resulted in dramatic clinical improvement of this infant's condition.

P324 SEVERE CELIAC DISEASE PRESENTED AS ACUTE ON CHRONIC LIVER FAILURE

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10.1136/archdischild-2019-epa.673

Introduction Celiac disease is an immune-mediated small intestinal enteropathy with an increasing global prevalence. Despite worldwide awareness, the diagnosis is frequently delayed because of a broad range of clinical manifestations of this disorder.

Case-report We report a 12-year-old boy who was referred from another hospital with the suspicion of a chronic hepatopathy, with peripheral oedema, abdominal distension (possibly ascites), hypoalbuminemia, coagulopathy (INR 2.5) and low ceruloplasmin level. The disease progressed gradually with these symptoms for 2 weeks. Furthermore, the patient complained of intermittent diarrhoea, weight loss (8 kg), growth failure, chronic abdominal pain and fatigue for 18 months. The patient presented severe wasting and stunting (BMI 13.7), pallor, dry skin, clubbing, lack of energy and bradycardia. Ultrasonography detected diffusely hyperechoic liver, polyserositis and dilated bowel loops. ALT was slightly elevated, bilirubin level and ammonemia were normal, INR 1.2 after vitamin K parenterally administered (Koller test) and urinary copper excretion rate was low. We excluded the hypothesis of liver failure. Antibody TG >200 U/ml, positive EMA and HLA-DQ2 typing, led us to the final diagnosis of celiac disease. Treatment included a gluten-free diet, albumin, calcium and magnesium supplements, with an improved clinical appearance.

Conclusions The coagulopathy caused by vitamin K malabsorption and hypoproteinemia could be misdiagnosed as liver disease, leading to delayed diagnosis and inappropriate treatment of celiac disease. Further attention should be given towards the uncommon presentations of celiac disease.

P325 COW'S MILK PROTEIN ALLERGY IN CHILDREN – CLINICAL PRESENTATION, DEMOGRAPHIC DATA AND FAMILY HISTORY IN A STUDY POPULATION

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10.1136/archdischild-2019-epa.674

Introduction Food allergy in children is a frequent topic of clinical studies, considering the increasing number of the affected individuals and the diversity of the symptoms these children demonstrate. Cow's milk protein allergy is the most common food allergy in the paediatric population, with an estimated prevalence of 2–6% in infants.

Cow's milk protein allergy can induce a variety of clinical symptoms, therefore establishing the correct diagnosis is often difficult. Beside the various clinical presentation, demographic and additional medical data of the research population and their families were also reviewed in this study.

Methods The study was conducted at the Paediatric Gastroenterology Department of the Balassa János County Hospital in Szekszárd, Hungary. The research population (n=47) included children (0–18 years) with symptoms suggesting cow's milk protein allergy. This component of our research represents the data from questionnaires filled in by the parents. The evaluation of the results was performed with SPSS statistical software.

Results 47 children were included in our study (57.4% male, mean age: 7.36 years, SD: 4.22).