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CHRONIC DIARRHEA IN INFANTS AND CHILDREN

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Background and aim: The aim of this study is to determine the causes of chronic diarrhea and the prevalence of Parasitosis-Giardiasis, Coeliac disease (CD), Cystic fibrosis, Sugar malabsorption, Inflammatory bowel disease, Toddler's diarrhea in our unit.

Methods: The study includes 116 patients with chronic diarrhea, defined as more than 3 loose or liquid bowel movements a day for at least 4 weeks. Physical examination focuses on overall appearance and signs of dehydration, growth parameters and abdominal findings. Tests include electrolyte levels, sweat Cl and Na levels for cystic fibrosis; stool microbiology for viruses, bacteria or parasites when infection appears to be present, and stool pH for disaccharide intolerance. Levels of certain antibodies including anti-gliadin antibody (AGA) transglutaminase (TTG); Albumin, markers of inflammation (ESR; CRP thrombocytosis), endoscopy, biopsies.

Results: Parasitosis (mostly giardiasis) was considered to be the cause of diarrhea in 30 (25.8%) patients, Cystic fibrosis 15 (12.93%), Food intolerance-coeliac disease 21 (18.1%), Sugar malabsorption 17 (14.6%), Inflammatory bowel disease 4 (3.44%), Toddler's disease 18 (15.5%), Others 10 (8.6%).

Diarrhea stopped in the 85 patient after specific treatment.

Conclusions: Causes of the chronic diarrhea are multiple. In most cases there is an organic cause that justifies diarrhea.

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RELATIONSHIP BETWEEN PARALYTIC ILEUS AND ACUTE DIARRHEA

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Background: Authors have studied the relationship between paralytic ileus and acute diarrhea in children in Kosovo.

Material and methodology: The study is retrospective and covers the period of one year. All children, aged 0 - 5 yrs, hospitalized due to their acute gastroenteritis at the Department of Pediatric Gastroenterology of the University Clinical Center of Kosova in Prishtina. Children with metabolic disease were excluded. Data on the laboratory work up and plain abdominal x-ray were obtained from the patients history.

Results: 992 children with acute diarrhea were included. 17 were diagnosed with paralytic ileus. All ileuses were radiologically confirmed. All children has abdominal pain and distension. Only one had no vomiting (1/17 or 5.88%). The mean value for sodium was 136 and of potassium 3.59 with variations between 128 and 167 for sodium and 2.1 and 6.3 for potassium. In one child dehydration was hypernatremic. The mean value for blood urea was 3.30 and 71.33 for creatinine, with maximum values in the severely dehydrated children of 6.5 for urea and 270 for creatinine. pH in patients was between 7.25 and 7.44 with mean value of 7.34.

Conclusion: The onset of abdominal pain, distension and vomiting in children with acute diarrhea should prompt for exclusion of the paralytic ileus.

Key words: Gastroenteritis, acute, paralytic ileus

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PORTAL VENOUS GAS DETECTED BY ABDOMINAL ULTRASOUND IN THE DIAGNOSIS OF SUSPECTED NECROTIZING ENTEROCOLITIS

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Aim: To prospectively assess the validity of detecting portal venous gas on abdominal ultrasound (PVG-US) in the diagnosis of suspected NEC.

Methods: We conducted a prospective cohort study over a three-month period commencing in January 2010. We recruited 6 babies that presented with symptoms and signs suggestive of NEC as defined by the Walsh and Kliegman² staging criteria. All babies had a plain abdominal x-ray performed at presentation. We followed standard protocols for the management of NEC. All babies had an abdominal ultrasound to detect portal venous gas within 48 hours of presentation using a Siemens Sonoline G60 ultrasound machine with a C-80 curvilinear

probe. Average gestational age was 29+6 and mean birth weight was 1296g. Our study is ongoing.

Results: Portal venous gas was detected on abdominal ultrasound in 2 out of 6 of the babies. Both babies were nursed in close proximity in the neonatal unit and developed NEC within 72 hours of each other. A third baby with presumed NEC but no PVG-US presented within the same period in the same 13-bedded unit. This subset may represent crossover infection. One of the confirmed NEC cases died post open laparotomy.

Discussion: NEC remains an enigma in neonatal intensive care units. Abdominal ultrasound to detect portal venous gas is a simple, cost-effective, non-invasive, bedside test that is a useful aide in the diagnosis of suspected NEC. A negative scan may permit us to feed those babies with suspected NEC.

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UPPER GASTROINTESTINAL BLEEDING BETWEEN PREMATURE AND MATURE BABIES IN EARLY INFANCY

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Aims: Upper gastrointestinal (UGI) bleeding is not rare among pediatric patients. However, there are limited reports describing this clinical condition in young infants. This study was aimed to assess and analyze the characteristics of UGI bleeding in infants, and try to define the differences between prematurely and maturely born patients.

Methods: A retrospective review for admission up to the age of 6 months with the clinical diagnosis of UGI bleeding along with positive fetal hemoglobin confirmed by Apt-Downey test in newborns or mucosal lesions seen in the UGI tract by endoscopic examination between June 2000 and October 2007.

Results: Thirty patients were enrolled, 14 premature and 17 mature infants. More than half of the patients got the disease before 2 days of age, with more than two thirds before one month. UGI bleeding was more prevalent in premature boys ($p=0.04$). Most premature infants bled from their UGI tract with concomitant disease, mainly respiratory distress syndrome under airway positive pressure support. Hemorrhagic gastritis was found

in 57% of the cases who received UGI endoscopy, being the most frequent. UGI bleeding stopped in 2 days on average after treatment. No recurrent UGI bleeding occurred in those patients followed for about 3 years.

Conclusions: Reason for UGI bleeding is still obscure in neonates and young infants. Premature boys seem to be more vulnerable. Conservative treatment made UGI bleeding evolve rapidly and favorably in early infancy. Both premature and mature patients recovered from UGI bleeding satisfactorily and showed no significant differences clinically.

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FAMILIAL LIPOPROTEIN LIPASE DEFICIENCY

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Background: Mutations in the LPL gene cause familial lipoprotein lipase deficiency. Symptoms of familial LPL deficiency usually begin in childhood and include abdominal pain, acute and recurrent inflammation of the pancreas, skin lesions called eruptive cutaneous xanthoma and an enlargement of the liver and spleen. The risk for two carrier parents to both pass the defective gene and, therefore, have an affected child is 25% with each pregnancy.

Aims: Early diagnosis, routine surveillance and treatment of familial LPL deficiency may help to manage some of the symptoms and sometimes prevent related problems.

Methods: Clinical genetic testing for familial lipoprotein lipase deficiency may be available through an in person genetic consultation for children who are considered at risk. Triglycerides and total cholesterol were measured using commercially available kits (Boehringer Mannheim).