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).