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Background. Pancreatic cancer is a highly lethal malignancy with few effective therapies. Pancreatic cancer is often associated with thromboembolic disease, as the malignant condition induces a prothrombotic and a hypercoagulable state.
Case report. This is a case of a 63 years old patient, diagnosed with pancreatic cancer with hepatic and splenic metastasis detected on CT, with a level of Carbohydrate antigen (CA) 19-9 of 2556 UI/ml and several associated comorbidities: a moderate form of iron deficiency anemia (Hb -7.4 g/dL, Ht - 25.2%, serum iron - 3.28 μmol/L), large gastric varices at the level of the fornix secondary to segmental portal hypertension, but with no signs of bleeding, incomplete intestinal metaplasia and Helicobacter Pylori infection at the level of the antrum and type 2 diabetes insulin dependent. The patient presented in our Medical Clinic complaining pain and functional impotence of the right inferior limb. We performed a Doppler ultrasound that revealed femoral-popliteal-tibial thrombosis of the right inferior limb and thrombosis of the internal saphenous vein. Due to the association of the thrombotic disease with the gastric varices, the initiation of antithrombotic therapy was questioned because of the high risk of variceal rupture and massive bleeding. The patient was recommended endoscopic injection sclerotherapy, but the procedure could not be performed due to the lack of compliance. A treatment the with low molecular weight heparin (Fragmin 2500 IU) and Vessel Due F was initiated. The patient condition was ameliorated during the admission and she was discharged with oncological and gastroenterological follow-up.
Conclusions. The peculiarity of this case consists in the association of the thrombotic condition with the gastric varices, both as complications of pancreatic cancer. The treatment in this case has to be carefully chosen, as the patient is at high risk of developing both gastric bleeding and thrombotic embolism.
Key words: pancreatic cancer, thromboembolism, gastric varices

DEPARTMENT OF PEDIATRICS

13. HIGH SERUM UNCONJUGATED BILIRUBIN LEVELS IN A PATIENT WITH MUTATIONS IN THE UGT1A1 GENE – CLINICAL CASE PRESENTATION

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Background. The UGT1A1 gene plays a significant role in the glucuronidation of bilirubin, and the mutations of this gene lead to limitations in the synthesis of the glucuronyltransferase enzyme, which contributes to the increase in free serum bilirubin. This clinical condition is called Gilbert's syndrome. The patient with Gilbert syndrome has no clinical manifestations until the second decade of life. Scientific studies demonstrate that free serum bilirubin in patients with Gilbert syndrome is almost entirely unconjugated. We present the case study of a 17-year-old patient with Gilbert's syndrome, confirmed by molecular genetics tests.
Case report. Patient was born from the first pregnancy with satisfactory evolution. Weight at birth was 3500g, height 52 cm, Apgar score 8/8. She was breastfed until the age of 1.5 years. Growth and development was within normal values, but after 4-5 years of age, she began to manifest periodically poor appetite, vomiting, abdominal pain, constipation. The dynamical assessment of clinical and paraclinical examinations revealed reactive pancreatitis episodes, “S”-

type gall bladder deformity, biliary stasis, hepatosplenomegaly (on ultrasonography non-homogeneous aspect of parenchyma with mild to moderate increased echogenicity). At the age of 10, she was diagnosed with left nephroptosis, secondary chronic pyelonephritis and chronic cystitis. She was diagnosed with adenoiditis and chronic sinusitis, episodes of otitis. On July 20, 2015, patient presented with jaundice. The hemoleucogram revealed mild anemia (hemoglobin was 112 g/l, erythrocyte 3.8 mln/mcl). The biochemical examination revealed increased bilirubin levels, mainly on the basis of free bilirubin: total bilirubin was 36.0 mmol/l, conjugated bilirubin was 9.0 mmol/l, free bilirubin 27.0 mmol/l. Serum glucose was at normal level (4.9 mmol/l), thymol test - 1.0 (normal value). The transaminase levels were normal (ALT 13.6 IU, AST 20.1 IU). From the history, serum bilirubin levels were normal until adolescence. At the age of 17, the level of bilirubin increased considerably, leading to suspected viral hepatitis, which could be present considering that hepatitis was present in the family. For differential diagnosis purpose, markers of viral hepatitis were tested: anti-HBs antibodies <2 m UI/mL (negative). Immunological tests indicated normal values: qualitative C reactive protein <6 mg/L; qualitative ASLO <200 IU; rheumatoid factor <8. Given the exclusion of the infectious factor, Gilbert's syndrome diagnosis was suspected, and was confirmed later by molecular genetic testing: a homozygous insertion of a TA dinucleotide in the region of the UGT1A1 gene promoter (genotype UGT1A1 7/7) was identified in the patient.
Conclusions. The clinical and evolution features of the case present clinical interest for the differential diagnosis of jaundice and accumulation of unconjugated (free) bilirubin in the serum.
Key words: Gilbert's syndrome, hyperbilirubinemia, jaundice, gene mutation

DEPARTMENT OF OBSTETRICS AND GYNECOLOGY

14. INTRAVENTRICULAR HEMORRHAGE WITH SUBSEQUENT VENTRICULOMEGALY IN PRETERM INFANTS

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Background. Preterm infants are at risk for developing many perinatal complications such as intraventricular haemorrhage (IVH) and subsequent ventriculomegaly. Babies born before 32 weeks of pregnancy present an even higher risk for IVH, an important cause of severe cognitive and motor neurologic impairment in very low birth weight infants. The risk of IVH is inversely related to gestational age and birth weight.
Case Report. A male neonate was admitted to the neonatal intensive care unit of Mures County Emergency Hospital. He was delivered by a cesarean section at 30-31 weeks of pregnancy. Birth weight was 1300 grams, the cranial perimeter was 29 cm and Apgar score was 7/8 at 1 and 5 minutes respectively. The primary physical examination revealed mild respiratory distress that evolved in the next two days to bilateral pneumothorax, which was successfully drained. Other abnormalities were found such as hypertrophic cardiomyopathy, atrial septal defect (2 mm) and mild mitral insufficiency. Psychomotor agitation was observed and treated with Midazolam, which caused paralysis of the respiratory center. The newborn was intubated. On the 10th day of life, transfontanellar ultrasound showed intraventricular hemorrhage in resorption and secondary ventriculomegaly. Hypotonia, feeding issues and postprandial vomiting was noted, also high levels of procalcitonin and lactate dehydrogenases were found. On the 16th day of life, bacteriological examination identified an infection with Candida Albicans and Enterococcus Faecium. Treatment with Teicoplanine was initiated. On the 19th day of life, articular pain was observed and the pediatric surgery examination revealed left humeral and forearm fractures,