with infundibular and valvular stenosis of the pulmonary artery with ultrasound pressure gradient over pulmonary stenosis of about 50 mmHg. The infant, then at the age of 4 months, underwent invasive cardiac procedure during the next hospitalization, showing pulmonary branches: the initial part of the right branch was 5 mm, followed by a fully hypoplastic right branch (3 mm) with middle segment stenosis up to 2 mm; the left branch is even more hypoplastic, entirely 2.2 mm with 2 mm segments. According to the finding, surgery was performed to establish an aortic-pulmonary anastomosis in the form of an mBT (modified Blalock Taussig) compound. In addition to cardiac processing, elevated levels of liver transaminases, primarily GGT enzymes, were observed in the findings. Due to the justified suspicion of Alagille's syndrome (tetralogy of Fallot with elevated liver transaminases), an X-ray of the spine was performed, where the fusion of the bodies of the Th6 and Th8 vertebrae was found, the so-called 'butterfly' vertebrae. Among other things, an ophthalmological examination was performed where the posterior embryotoxon of both eyes was observed. Complete processing met the higher diagnostic criteria for Alagille's syndrome, and therefore genetic analysis was requested, which confirmed the heterozygous variant in the JAG1 gene and diagnosed Alagille's syndrome. During the last hospitalization, cardiac catheterization was performed, which still showed hypoplastic pulmonary branches with a narrow mBT joint.

Discussion/Conclusion Although pulmonary stenosis is the most common heart defect within Alagille syndrome, tetralogy of Fallot with hypoplasia of the pulmonary branches may also be one of the clinical manifestations. Since the pulmonary branches are still hypoplastic in our patient after aorthopulmonary anastomosis, in the idea of better pulmonary blood supply, dilatation of the pulmonary valve with a balloon was performed for better antegrade flow until a decision on further treatment modality.

Paediatric Clinical Pharmacology and Toxicology

193 POPPY SEED DEFENCE

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Acute loss of consciousness in an adolescent prompts detailed evaluation, including toxicology analysis. In case of a positive opiate test, it is of outmost importance to confirm or exclude drug abuse, which can have important legislative consequences.

We present a 17-years-old boy who was admitted due to acute loss of consciousness. After getting up, he felt dizzy and then fainted, followed by myoclonic jerks. He recovered spontaneously. His previous history, physical and neurological examination were unremarkable. Initial laboratory evaluation, ECG and EEG were normal but urine toxicology screening tested positive for opiates (4565 nmol/l = 1305 ng/ml = 1.3 ug/ml). He denied previous use of any medications or illicit drugs. However, he reported eating large quantities of poppy seed cake in the morning and during the previous day. The following day the opiate screen was negative. According to

the data in the literature, oral intake of cakes containing commercially available poppy seeds can produce urine morphine concentrations ranging from

1 to 10 ug/ml and morphine can be detected in the urine for up to 48 hours following ingestion. Opiate concentrations produced by poppy seeds ingestion depend on the origin of poppy seeds, lot, and baking technique. To confirm the possibility of false positive test, a volunteer at the Department eat the same poppy seed cake followed by urine toxicology analysis which tested opiate positive. The patient was discharged from the hospital with diagnosis of vasovagal syncope.

A detailed patient-oriented history is essential in order to establish the correct diagnosis. The possibility of false-positive opiate drug tests after poppy food ingestion should always be considered. There are no data regarding 'safe' quantity of food containing poppy seeds that would not interfere with drug test. It is important to know the limitations of toxicology analysis in order to avoid unnecessary stigmatization of a patient and serious legal drawback.

194 LATE-PRESENTING ACETAMINOPHEN SELF-POISONING

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We report a case of a 15-year-old adolescent girl who presented to the Intensive Care Unit (ICU) of Children's Hospital Zagreb 38 hours after having intentionally ingested 10 tablets of 1000mg acetaminophen and 7 tablets of 500mg acetaminophen (13.5 grams). Initially, she presented to the Emergency Department of General Hospital Virovitica, 36 hours after ingestion, main complaint was nausea, vomiting and abdominal pain. She was conscious with mild scleral icterus, eutermic, BP 97/63 mmHg, HR 73/min. Initial serum chemistry measured PT 0.27, AST 2138 U/L, ALT 2069 U/L, creatinine 75 umol/L. Vitamin K was administered and she was transferred to the ICU. Due to ingestion of toxic acetaminophen dose, N-acetylcysteine (NAC) was initiated, starting with loading dose of 6.6 grams over 1 hour, then 2.2 grams over 4 hours, continued with maintenance dose of 4.4 grams over 16 hours, following the 21-hour NAC protocol. Blood tests were performed daily, including complete blood count, renal and liver functions, ammonia, prothrombin time and blood gas analysis. Results revealed elevated levels of transaminase, peak values AST 11567 U/L, ALT 10681 U/L, LDH 9651 U/L, high ammonia values, peak value 158 umol/L, and coagulopathy, peak INR value 4.04. Abdominal ultrasound reported diffusely enlarged hypoechoic liver, measuring 14cm in the midclavicular line, with hypoechoic area, 3cm in size, which may correspond with necrosis zone.

Parenteral rehydration, hepatic diet and psychological support were implemented during the hospitalization. Serum acetaminophen levels were measured daily. 36 hours after ingestion, level was 557 umol/L.

Concentrations were detectable until the seventh day after ingestion, so NAC was discontinued after 6 days of treatment, when concentrations were undetectable and liver enzymes decreased. Psychiatric evaluation was conducted, she was characterised as a perfectionist, very ambitious, not previously known to have any psychiatric or organic health issues. The

reason behind her suicide attempt was emotional reaction to stressful event.

She was closely monitored for development of any of criteria for liver transplantation (hepatic encephalopathy, severe acidosis, severe coagulopathy INR $5 <$), but she did not develop any. Thus, even though NAC administration was delayed, it was efficient in the prevention of hepatotoxicity. She was discharged on day 15 after ingestion, with decreasing AST and ALT values (47 U/L and 437 U/L, respectively). On her check-up one month later transaminase levels were in normal range.

195 INTRAVENOUS ACETAMINOPHEN OVERDOSE – A THERAPEUTIC ERROR

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A 8-month-old female infant, weighing 7kg, with persistent cloaca, anal atresia, right renal agenesis, grade 3 vesicoureteral reflux, double-barrel colostomy and left nephrostomy was admitted to Department of Nephrology of Children's Hospital Zagreb for a urinary tract infection. On the sixth day, due to clinical deterioration and inadequate response to given antibiotic therapy she underwent a central venous catheter placement, amikacin was replaced with cefepime. That night, around midnight, she got a high fever and, as antipyretic, 100 ml of intravenous acetaminophen solution was administered. Shortly after, the nurse contacted the doctor on call and admitted to making a therapeutic error – instead of 100 mg of acetaminophen, she administered 100 ml of intravenous acetaminophen solution (10mg/ml), thus administering 1000 mg (142 mg/kg). Four hours after the administration serum acetaminophen concentration was 465 mcg/ml. She was transferred to intensive care unit and intravenous N-acetylcysteine (NAC) was started immediately, starting with loading dose of 1 gram in 25 ml 5% glucose solution over 1 hour, then 350mg in 50 ml 5% glucose solution, continued with maintenance dose of 700mg in 100 ml 5% glucose solution over the next 16 hours, following the 21-hour NAC protocol. Blood tests (liver and kidney functions, ammonia, prothrombin time, blood gas analysis) were performed daily, all values were in normal range. Infant remained well and without hepatic impairment. The treatment of NAC infusion over 21 hours was efficacious. This was a case of unintentional overdose, error in dose calculation and therapeutic error. Therapeutic errors such as 10-fold overdosing are common, especially during the night shift, so additional caution is needed. Dose of intravenous acetaminophen on medication order has to be written both in milligrams and millilitres to avoid dosing and administration errors.

196 COMBINED INTRANASAL DEXMEDETOMIDINE AND KETAMINE VS INTRANASAL DEXMEDETOMIDINE AND ORAL MIDAZOLAM FOR PROCEDURAL SEDATION IN CHILDREN: A RANDOMIZED MULTICENTRE TRIAL

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The aim of this study is to compare combination of intranasal dexmedetomidine and ketamine to intranasal dexmedetomidine and oral midazolam to evaluate induction time in children undergoing procedural sedation. In this specific setting a procedural sedation approach avoiding general anesthesia, the need for an intravenous line and the use of drugs with higher risk of paradoxical reactions (midazolam), emergency reactions

(sevoflurane) or respiratory depression (propofol) would be welcome.

Our multicentre trial was conducted in a tertiary pediatric teaching hospital and in a secondary hospital on patients in need of procedural sedation who referred to our hospitals from November 2019 to March 2020.

Sedation was performed by trained paediatricians with specific expertise and training in paediatric sedation, airway management and cardiovascular resuscitation. All consecutively admitted subjects were randomized to receive one-time dose (4 mcg/kg) of intranasal dexmedetomidine and 3µg/Kg of intranasal ketamine or one-time dose of 4mcg/Kg of intranasal dexmedetomidine and oral midazolam (0.5mg/Kg) with the intended goal to compare induction-time, effectiveness and adverse effects of such sedation.

The patient who did reach an adequate sedation level underwent an intravenous line positioning and a dose of ketamine or propofol.

Fifty patients were recruited to receive intranasal dexmedetomidine and ketamine and fifty patients were recruited to receive intranasal dexmedetomidine and oral midazolam. The induction time was significantly lower in the intervention group when compared to the control group, while sedation success rate was similar in both groups and any major adverse effect was observed in both groups.

The combination of intranasal dexmedetomidine and ketamine should be considered as a possible option in for procedural sedation in children, in particular in settings in which is essential to shorten induction-time, without any further adverse effect and with a success rate comparable to oral midazolam and intranasal dexmedetomidine.

197 THE MAJOR CAUSES OF DRUG INTOXICATIONS IN CHILDREN

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In this study we aimed to investigate the most common drugs that caused ADIs in 0-18 year old patients and to observe the prevalence of suicide attempts.

A retrospective study was performed using medical charts up to 7 years (2012-2018) from 'Muratsan' University hospital complex (UHC) ICU and toxicology. We have separated the patients in four age groups(0-1;1-7;7-14;14-18 years old). The overall number of patients who had ADIs and were admitted to resuscitation unit was 1260. Mean age of patients was 4.7 years. We have included the most common drug intoxications typical for each age group.

114 patients under 1 year old have been diagnosed with ADI: 34(29.8%)-non-opioid analgesics, 15(13.2%)-cardiac