# 01C Гастроентерохепатологија / Gatsroenterohepatology

# 01C1

## Болести на црниот дроб

## Diseases of the Hepar

## Научен одбор / Scientific Committee

Проф.д-р Розалинда Попова / Prof. Rozalinda Popova MD, PhD

Проф.д-р Ненад Јоксимовиќ / Prof. Nenad Joksimovic MD, PhD

Доц.д-р Магдалена Генадиева-Димитрова / Asst. Prof. Magdalena Genadieva-Dimitrova MD, PhD

# Session code: 01C1

*Постер презентација / Poster Presentation*

## The Onset, Outcome And Prognosis Of Hepatorenal Syndrome – Single Centre Experience A Decade Ago And Today

**Vladimir Andreevski[1], Dafina Nikolova[1], Magdalena Genadieva-Dimitrova[1], Nenad Joksimovic[1],**

[1] University Clinic of Gastroenterohepatology, Medical faculty Skopje, University "Sts. Cyril and Methodius" Skopje, Republic of North Macedonia

Keywords: *hepatorenal syndrome, hepatic failure, outcome, prognosis*

**Objectives:** To investigate and compare the onset, outcome and prognosis of patients with hepatorenal syndrome (HRS) hospitalized at our unit in two different periods, a decade ago and today.

**Material and Methods:** This is a cross-sectional retrospective study in a cohort of 1202 cirrhotic patients in total, during two 3 year periods [543 in period A - (January, 2008-December, 2010) and 659 in period B – (January, 2018-October, 2020)]. Hepatorenal syndrome was detected in 20 (3.7%) patients a decade ago and in 23 (3,4%) patients in period B. Few variables such as: age, gender, history of cirrhosis and type of liver disease, etiology, Child-Pugh classification, other complications except for HRS like spontaneous bacterial peritonitis (SBP) and hepatopulmonary syndrome (HPS), treatment and survival were analyzed.

**Results:** The average preceding time up to the occurrence of HRS in period A was around 3 years (36.8±47.8 months) and less in period B (25.8±46.7 months), although there were 4 patients who developed HRS only a month after the onset of symptoms in period A compared to 8 in period B with development of HRS only 3 months after the symptom detection. The mean age of patients was insignificantly different (55.5±13.3 in period A versus 60.1±34.1 years in B). There was a significant predominance in the gender distribution, almost three quarters of patients being males in both periods. With regard to the etiology, over a period of decade a slight change was noted. A decade ago 4 (20%) patients were with chronic liver disease of unknown etiology compared to 13% cryptogenic cases from 2018-2020. Alcoholic abuse was present at almost 60% in both periods (12 out of 20 - period A and 14/23 patients – period B). Unlike before, in period B there were 2 patients with Primary Biliary Cholangitis and Primary Sclerosing Cholangitis. All of the cirrhotic patients were scored as grade C according to the Child-Pugh classification in both periods. A decade ago and today, hepatic encephalopathy was the most predominant concomitant complication present in 17 (85%) patients compared to 95%, respectively, in patients with HRS. The news in second period was the appearance of 5 cases of SBP and 1 with HPS. Only 2 in period A and 3 cases in period B showed signs of malignancy with confirmed hepatocellular carcinoma. In period of one decade, the estimated average hospital stay rise up from 6.15 (1-14) days to 9.43 (1-29) days. Like 10 years ago the applied treatment is still unsuccessful. There is clear improvement in terms of use of supportive measures with albumin and fresh frozen plasma transfusion with only 14 (70%) cases before compared to 100% nowadays. Haemodialysis was performed in only 4 patients 10 years before versus 78.3% (18/23) these days. Regardless, the mortality rate is even worst, reaching 91% compared to 80% (16 patients) a decade ago with an average time of death at 6.8±4.4 days after the hospital admission. Although the compared periods were split over a decade, there is no raising trend in percent of detected patients with HRS out of cirrhotic one.

**Conclusion:** Like 10 years ago, our single center experience still shows lower occurrence rate compared to other reports, which points to necessity of improving the detection rate. Despite the improved use of available conservative medical treatments today, there was no recovery of the hepatic failure in almost any of HRS patients over the years. Despite the introduction of TIPS in our country, its rare use and the absence of liver transplantation, seems to be the important contributing factor related to the high mortality rate in our cohorts. Finally, gastroenterohepatologists should be always aware of HRS and never ever forget that dialysis doesn't improve the long term prognoses of these patients.

# Session code: 01C1

***Постер презентација / Poster Presentation***

## Diet-Related Improvement Of Non-Alcoholic Steatohepatitis

**A.Volkanovska Nikolovska[1], D.Nikolova[1], E.Nikolovska Trpchevska[1], M.Trajkovska[1]**

[1]University Clinic for Gastroenterohepatology, Skopje, North Macedonia

Keywords: *NAFLD, NASH, liver cirrhosis, dieting, physical activity*

Non-alcoholic fatty liver disease (NAFLD) refers to a wide pathological spectrum ranging from simple steatosis to steatohepatitis (NASH) with or without variable degrees of fibrosis. It represents an increasing health problem since it leads to the development of cirrhosis and liver cancer. In Western countries NAFLD/NASH-associated cirrhosis is becoming one of the most frequent indications for liver transplantation. Thus it is important to recognize and identify patients at risk of progression of NAFLD and implement therapeutic interventions. The goal is to prevent or reverse the liver inflammation and finally prevent detrimental consequences of advanced NASH.

NAFLD is more commonly encountered in obese and patients with diabetes. The key pathogenic trigger is insulin resistance, which through simple steatosis leads to steatohepatitis. The later is the strongest predictor of fibrosis progression in NAFLD.

The management of NAFLD/NASH is challenging as there is lack of an effective therapy and no approved pharmacological agent for the treatment of NASH. The results from clinical studies point to dietary intervention as the cornerstone of the therapy.

We present a case where improvement of NASH was achieved solely by lifestyle modification. A 37 year old male patient with elevated transaminases was referred to our clinic for evaluation. After initial assessment with detalied history, comprehensive laboratory analyses and abdominal ultrasound, a liver biopsy was performed and he was diagnosed with NASH. His initial body mass index (BMI) was 28.9 kg/m$^2$, the homeostasis model assessment-insulin resistance (HOMA-IR) was 5.6. Presence of hypercholesterolemia and hyperferritinemia was also noted. He was advised to commence a low calorie diet accompanied with physical activity. Additionaly he was prescribed hepatoprotective (silymarin) therapy, vitamin D and C supplementation and lipid lowering agents. In the following year the transaminase activity and insulin resistance were maintened despite pharmacological treatment but the patient reported that he hasn't been paying attention to his calorie intake and has been practicing only mild physical activity. Upon re-evaluation, he was advised to initiate metformin therapy, but he refused, so a dedicated nutritional counseling was performed emphisizing the risks of ongoing liver inflammation and ensuing liver damage. The patient started with calorie restrictive diet, low impact aerobic exercise (pool swimming) and continiued only with vitamin D supplementation. After 3 months he achieved a reduction of 10% of his initial body weight. Control blood analyses showed normalisation of the transaminase activity, as well as a decrease in HOMA-IR value. The BMI was 25.7 kg/m$^2$, his lipid profile improved and ferritin levels also decresed. He was advised to continue with his lifestyle modification and was scheduled for 3 months interval monitoring as to sustain his compliance.

The results achieved with dieting and physical activity presented in this case strongly support the role of lifestyle modification as primary therapy for the management of NAFLD/NASH. But there is a reasonable possibility of relapses, so dietary intervention accompanied with strategies to avoid relapse and weight regain should be implemented.

# Session code: 01C1

*Постер презентација / Poster Presentation*

## Thrombotic Complications Following Liver Transplantation Due To Budd-Chiari Syndrome

**A.Volkanovska Nikolovska[1], D.Nikolova[1], E.Nikolovska Trpchevska[1], A.Karadzova Dzambaz[1]**

[1]University Clinic for Gastroenterohepatology, Skopje, North Macedonia

Keywords: *liver transplantation, vascular complications*

Трансплантација на црн дроб претставува рутинска процедура на лекување на црнодробна слабост, но може да биде комплицирана со развој на стенози на васкуларните анастомози. Овие компликации можат да бидат причина за продолжен хоспитален престој, губење на трансплантот, ретрансплантација и смрт на пациентот. Васкуларни компликации почесто се развиваат при трансплантација од жив донор поради комплексната васкуларна реконструкција. Од критична важност за преживување на трансплантот и примателот е раното препознавање и дијагноза на овие компликации. Почести васкуларни компликации се тромбоза и/или стеноза на arteria hepatica (AH) и vena portae (VP), а поретко стеноза на венскиот одводен систем на црнодробниот трансплант.

Во нашиот труд прикажуваме трансплантиран пациент со развој на консекутивни неопструктивни и тромботични компликации на клучните васкуларни структури на црнодробниот графт.

Кај пациент на возраст од 33 години беше направена црнодробна трансплантација од жив донор поради терминална црнодробна слабост како резултат на Budd Chiari синдром, поради генетски условена тромбофилична состојба асоцирана со хетерозиготна фактор V Leiden мутација. Постоперативно пациентот беше поставен на редовна тромбопрофилакса со ацетилсалицилна киселина согласно протоколите на трансплантацискиот центар. Една година подоцна беше детектиран асцит во голема количина без значаен одговор на диуретска терапија со прогресивна спленомегалија. Радиолошкиот наод на ниво на хепатичните вени, вена кава и портната вена беше уреден. Со перкутана хепатална венографија се постави сомнение за синдром на неоклузивна хипоперфузија на АН поради што беше направена емболизација на arteria lienalis. Интервенцијата не придонесе за редукција на асцитната течност, иако функционалноста на трансплантот беше сочувана. Контролната радиолошка ре-евалуација на васкуларниот систем на трансплантот идентификуваше хронична тромбоза на хепатично-кавалната анастомоза со присуство на значајна стеноза на vena cava inferior што резултира со пораст на притисочниот градиент на ниво на венскиот одводен систем > 5mm Hg. Поради тоа, беше пласиран самоекспандирачки стент во vena cava inferior, со нормализација на притисочниот градиент. По интервенцијата, антиагрегационата терапија беше заменета со орална антикоагулантна терапија (ОАТ), што резултираше со постепена редукција на асцитната течност. Пациентот беше следен редовно со ултрасонографски Доплер на васкуларните структури и КТ ангиографија, без наод на тромботичен супстрат. Една година подоцна, и покрај редовната ОАТ, беше дијагностицирана тромбоза на анастомозата на VP, со знаци за нарушување на црнодробната функција. И покрај примена на различни достапни антикоагулантни средства, не се постигна реканализација. Поради ризик од губење на графтот, кај пациентот се планира мезо-кавален шант, како премостувачка процедура до ретрансплантација.

Заклучок: Васкуларните компликации се главен проблем после трансплантација, а нивната дијагноза и терапевтски менаџмент се голем медицински предизвик. Ендоваскуларните интервенции имаат предност во нивното решавање во однос на хирургијата. Но, како што е прикажано во нашиот случај, сукцесивната појава на васкуларни компликации на повеќе нивоа, значајно придонесува за губење на графтот и ја зголемува потребата од ретрансплантација. Единствено решение е контрола на ризик факторите и, доколку е можно, што порана дијагноза, дури и кај асимптоматските пациенти.

# 01C Гастроентерохепатологија / Gatsroenterohepatology

# 01C2

## Разно / Varia

## Научен одбор / Scientific Committee

Проф.д-р Розалинда Попова / Prof. Rozalinda Popova MD, PhD

Проф.д-р Ненад Јоксимовиќ / Prof. Nenad Joksimovic MD, PhD

Доц.д-р Магдалена Генадиева-Димитрова / Asst. Prof. Magdalena Genadieva-Dimitrova MD, PhD

# Session code: 01C2

***Постер презентација / Poster Presentation***

## Herpes simplex esophagitis a cause of odynophagia in young patient: case report

**Dafina Nikolova[1], Ance Volkanovska Nikolovska[1], Vladimir Andreevski[1], Urim Isahi[1]**

[1]University Ss. Cyril and Methodius, Faculty of Medicine, University Clinic of Gastroenteroheopathology, 1000 Skopje, Republic of North Macedonia

*Keywords: odynophagia, herpes simplex virus, esophagitis*

**Objectives:** Herpes simplex esophagitis is usually found in immunosuppresed patient, those with malignancy, patient with AIDS, patient treated with immunosupresive therapy and in terminally ill patients. Very rarely it can be found in healthy, immunocompetent individuals. We present a case of a young patient with herpes simplex esophagitis manifested with odynophagia, retrosternal pain and fever.

**Case presentation:** 18 years old patient referred to our clinic because of acute and severe odynophagia, retrosternal pain and fever in the last five days. She had no previous medical conditions. On physical examination the patient had fever up to 39C and tachycardia of 105/min. She had no abdominal tenderness, no palpable masses or hepatosplenomegaly. Laboratory analysis showed normal total white blood cell (WBC) count of 4.7x109/L. The patient underwent an upper gastrointestinal endoscopy. Upper gastrointestinal endoscopy revealed friable and inflamated esophageal epithelium with multiple confluent ulcerations. Several biopsies were taken and patohystological analysis showed inflamamatory infiltrate with acute vascular proliferation and presence of lymphoid accumulations and eosinophils. This finding showed acute erosive esophagitis. In addition, HSV serology was done and HSV IgM and HSV IgG were positive. Blood serology for CMV IgM was negative. The patient was treated with oral acyclovir 200mg five times a day, for two weeks. Improvement of the symptoms was reported the second day, with complete resolution after completing the 14 days course of antiviral treatment.

**Conclusion:** Herpes simplex esophagitis is a condition associated with immunosuppresd patient. It is typically presented with odynophagia, severe retrosternal pain and fever. Rarely, it can be found in immunocompetant patient and effectively treated with antiviral medications.

# Session code: 01C2

*Постер презентација / Poster Presentation*

## Lymphangiomas of the liver and spleen: rare case presentation

**Nikolovska Trpchevska E[1], Popova Jovanovska R[1], Volkanovska Nikolovska A[1], Nikolova D[1]**

[1] University Clinic of Gastroenterohepatology, Skopje, North Macedonia

**Keywords:** Liver, Spleen, Lymphangioma, Cyst

**Objectives:** Lymphatic malformations are benign lesions of vascular origin that show lymphatic differentiation. It is considered the lymphatic equivalent of a hemangiomas of blood vessels. A hepatic lymphangioma is a rare benign neoplasm and is usually associated with lymphangiomas of other viscera. It can occur at any age and most lesions are found incidentally. Splenic lymphangiomas are relatively rare benign tumors that correspond to abnormal dilatation of lymphatic channels that can be either congenital or acquired. On imaging, they usually present as lobulated and multiloculated cystic lesions without solid component or significant enhancement. Here we report a rare case of a hepatic cystic lymphangioma in a 73-year-old man and multiple lymphangiomas (cyst) in spleen.

**Material and methods:** It was discovered on a routine ultrasound examination and the patient had no obvious symptoms. A surgical resection of adenocarcinoma of prostate was performed one year ago. There is no need of hemotherapy after surgical treatment. Abdominal ultrasonography and computed tomography (CT) showed “hepatic neoplasm” and multiple cystic focal lesions in the spleen. Bone biopsy excluded hematological abnormalities. Laboratory examination with tumor markers and X-chest ray were normal. Screening gastroscopy and colonoscopy was performed, and the patient had reflux esophagitis and pendular polyp in sigmoid colon. After polypectomy of sigmoid polyp, histopathology findings show tubulous polyp. After all examinations, spleen biopsy was performed.

**Results:** Histological examination of spleen revealed multiple cystic structures lined with epithelial cells on the inner walls, accompanied by interstitial swelling and necrosis, marked as lymphangiomas.

**Conclusion:** A hepatic lymphangioma can be solitary, cystic or associated with multiple liver lesions and is characterized by cystic dilatation of lymphatic vessels in the hepatic parenchyma. A solitary lymphangioma is unusual. Presentation ranges from asymptomatic incidental finding to a large multicentric, symptomatic mass require surgical intervention. They may occur alone on the spleen or as a part of systemic lymphangiomatosis. In our case patient has been followed up for nearly two years with no worsen and enlarged lesions.