P-16 FIRST MICRO ELIMINATION INTERVENTION OF HEPATITIS B &C IN INMATES OF THE EIGHT PRISONS IN THE PROVINCE OF MENDOZA, ARGENTINA

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Introduction and Objectives: Accessing to a closed population such as prisoners opens a great possibility for controlling HCV infection through treatment with Direct-acting antivirals (DAAs). Introduction. This study aimed to determine HIV, syphilis, HBV and HCV prevalence in the eight penal institution inmates of Mendoza's province to achieve microelimination of viral hepatitis.

Materials and Methods: During 2019, HIV, HCV, syphilis and HBsAg tests were offered to all inmates. In order to characterize risk factors associated with this population, they were given a voluntary self-administered survey on sexual practices, drug use and self-perception of their health status. 4024 out of 4821 subjects were enrolled, 3,899 cisgender-men (83.02%) and 125 cisgender-women (100%); all of them signed informed consent.

Results: Prevalence for anti-HCV, HBsAg, anti-HIV and antiSyphilis were 0,82%, 0,15%, 0,15% and 2,55%, respectively. The average age of patients infected with HCV and HBV was 44 years old. In 31 out of 33 inmates, viral load and genotype were determined. The most prevalent genotype was 1a (71%), followed by 1b (19.3%), 3a (6,5%) and 2a/c (3.2%). 13 out of 31 (42%) received DAAs treatment, of which 9 (69%) had a sustained viral response (SVR), three did not reach SVR, and one is currently under treatment. 10 out of 31 (32%) inmates were lost to follow-up. Eight patients are waiting for their treatments. Only 1 out of 6 HBV-positive inmates had detectable viral load and is under follow-up.

Conclusions: There's a previous study in Argentina's federal prisons (2016) on 2.277 inmates, where HCV and HBV prevalences were higher than ours (3,3% and 0,51%, respectively). Analyzing the local survey response, this gap could be due to the percentage of injection drug use: 3,13% in provincial vs. 6% in federal inmates. Checking for HCV/HBV infections in every new inmate has been adopted as a sanitary policy until nowadays.

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P- 17 FRAILTY AND COVERT HEPATIC ENCEPHALOPATHY IN CIRRHOTIC PATIENTS AT A THIRD LEVEL HOSPITAL IN GUATEMALA

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Introduction and Objectives: In Guatemala, liver cirrhosis has a mortality rate of 41 per 100,000 inhabitants. Diminished physical reserve (frailty) is an important prognostic factor, largely determined by sarcopenia, which in turn has a role in the pathophysiology of hepatic encephalopathy. This study aimed to describe the relationship between frailty and covert hepatic encephalopathy to determine if cirrhotic patients with a higher degree of frailty have a higher probability of encephalopathy.

Materials and Methods: Cross-sectional analytical study with a non-probabilistic registry of consecutive cases with a statistical power of 80% and a confidence level of 90%. Patients with a diagnosis of cirrhosis without evident hepatic encephalopathy and without motor or neurocognitive impairment are included. Frailty status (prediction variable) was measured using the liver frailty index "Liver Frailty Index TM" and the one-minute animal naming test "ANT test" (outcome variable) was performed. These variables were analyzed using the chi square linearity test.

Results: 66 patients with cirrhosis were included, 61% female, with a mean age of 56 years; the main causes of cirrhosis found were alcohol (25.8%), Virus C (19.7%) and liver non-alcoholic fat (16.6%). Only 7.6% of the patients were robust, while 60.6% were pre-frail and 31.8% were frail. 56.1% of the patients presented with covert hepatic encephalopathy. Robust patients presented covert hepatic encephalopathy in 20%, pre-frailty in 55% and frail in 66.7% (p = 0.087), which resulted in a probability of covert hepatic encephalopathy for pre-frailty of 2.75, CI 90% [0.61-12.2] and for frailty 3.33 CI 90% [0.74-14.83].

Conclusions: In cirrhotic patients, frailty confers a greater probability of hepatic encephalopathy.

Table 1. Frailty and covert hepatic encephalopathy

Farilty status	Covert hepatic encephalophaty			
	Yes		No	
	f	%	*f*	%
Robust	1	20%	4	80%
Pre Frail	22	55%	18	45%
Frail	14	66.70%	7	33.35
p value chi square linearity test = 0.087				
PR pre Frail = 2.75, IC 90% [0.61-12.2]				
PR Frail 3.33, IC 90% [0.74-14.83]				

P- 18 WITH TREATMENT

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P- 18 TREATMENT WITH BEVACIZUMAB IN HIGH OUTPUT CARDIAC FAILURE DUE TO SEVERE HEPATIC COMPROMISE IN HEREDITARY HEMORRHAGIC TELANGIECTASIA PATIENTS: OBSERVATIONAL COHORT STUDY

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Introduction and Objectives: Hereditary Hemorrhagic Telangiectasia (HHT) is an autosomal dominant vascular dysplasia affecting 1/5000 individuals. Epistaxis, mucocutaneous telangiectasias and vascular malformations affecting internal organs (brain, lungs, liver and digestive tract) are hallmarks of HHT. Though liver involvement occurs in 80% of patients, including abnormal portal-venous, arterio-portal, and arterio-venous shunts, overt clinic is only present in 8% and may present as secondary high-output cardiac failure (HOCF), portosystemic encephalopathy, noncirrhotic portal hypertension and/or bile duct ischemia. This study aimed to report a single HHT Reference Center experience in the antiangiogenic treatment with bevacizumab (anti VEGF-Vascular Endothelial Growth Factor) for treating adult HHT patients with HOCF due to severe liver disease.

Materials and Methods: Observational cohort study. Baseline clinical/analytical characteristics were recorded and echocardiographic values for cardiac index in L/min/m^2 and cardiac output in L/min (CI and CO) before and after bevacizumab treatment were compared when available with a paired signed rank test.

Results: Thirteen patients were included from July/2013-June/to 2022, nine were women and the median age was 68 [IQR: 53-71]. All of them with HOCF; eleven had edema/ascites and six also had refractory iron deficiency anemia. Regarding liver compromise: nine had hepatomegalia, all had diffuse telangiectasias, six portal-venous, ten arterio-venous and eight arterio-portal shunts, while only two had ischemic bile duct injury. Basal median CI was 4.1 [3.8-4.8] and CO was 7.5 [6.1-8.6] (n=11). Median Bevacizumab number of the received doses was 6 [4-6]. At least one post-treatment result during the first year of treatment was available in 8 patients with a median CI of 3.5 [3.1-4.3] ($p<0.05$) and CO of 6.2 [4.5-3.0] ($p<0.05$). Two patients received liver transplantation while on treatment.

Conclusions: These results supporting bevacizumab treatment in HHT patients with severe liver disease are in line with previous reports.

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P-19 SPLENIC IRON OVERLOAD IN PATIENTS WITH HEREDITARY HEMOCHROMATOSIS

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Introduction and Objectives: Hereditary hemochromatosis (HH) is a polygenic disease characterized by elevated transferrin saturation (TfS) due to progressive and systemic iron overload. T2*-weighted magnetic resonance imaging (T2*MRI) is useful for assessing hepatic and splenic iron overload (SpIO). Until now, SpIO in patients with HH has been controversial. This study aimed to evaluate splenic iron overload in HH patients.

Materials and Methods: A Retrospective analysis was made in 113 patients studied with T2*MRI 1.5 Tesla, with phenotypic and liver histologic criteria of HH, without other causes of iron overload. All of them had a hepatic iron overload. In order to evaluate differences between patients with SpIO (Group 1 - G1) or without (Group 2 - G2), age, sex, serum ferritin (SF), serum iron (SI), serum transferrin (Tf), TfS, HFE gen mutations, hepatomegaly, libido loss (LIB), cirrhosis by liver histology, oral glucose tolerance test (OGTT), diabetes (DM); cutaneous (CI), joints (JI) and cardiac involvement (CVI) were compared between both groups. Statistical analysis: median with IQ range 25-75% and Mann-Whitney test. P-value <0.05 was considered significant.

Results: By T2*MRI, SpIO (G1) was observed in 53 cases (46.9%) and not detected (G2) in 60 (53.1%). Median age: G1, 47 years (39-60) vs. G2: 43 (30.5-53) (p=0.074); HFE mutations, G1: 20.75% vs. G2: 11.67% (p=0.14); SF>1000 ng/ml, G1: 35.85% vs. G2: 18.33% (p=0.029); hepatomegaly, G1: 67.92% vs. G2: 46.67% (p=0.018) and AI, G1: 33.96% vs. G2: 13.33% (p=0.009). Statistical differences were not observed when comparing sex, cirrhosis, SI, Tf, TfS, OGTT, DM, CI, CVI and LIB.

Conclusions: This study shows that nearly half of the patients with HH have splenic iron accumulation. This finding is more frequent in those with SF >1000 ng/dl, hepatomegaly and joint involvement. This preliminary data support to continue studying other polymorphisms that could be involved in HH and the impact of splenic iron overload on the disease.

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P- 21 DOCOSAHEXAENOIC ACID AND ITS DERIVATIVE MARESIN1 IMPROVE CHRONIC LIVER DAMAGE ASSOCIATED WITH THE PROMOTION OF APOPTOSIS PATHWAYS AND LIVER REGENERATION IN A SPRAGUE-DAWLEY MODEL.

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Introduction and Objectives: Liver fibrosis is a complex process characterized by excessive accumulation of extracellular matrix (ECM) associated with chronic injury inflammation and an alteration of liver architecture as a result of most types of chronic liver diseases such as cirrhosis, hepatocellular carcinoma and liver failure. The ω-3 Docosahexaenoic (DHA) fatty acids and their derivative Maresin-1 (MaR1) have been shown to have pro-resolutive, anti-inflammatory, and hepatoprotective liver effects on acute models of liver study, but their role in apoptosis and liver regeneration remains to be elucidated. This study aimed to analyze the role of DHA+MaR1 in the prevention and restoration of liver fibrosis damage, enhancing a regenerative phenotype in an animal model of chronic liver damage.

Materials and Methods: Sprague-Dawley rats were inducing liver fibrosis by injections of diethylnitrosamine (DEN) 50mg/ml twice a week and treated with DHA with or without MaR1 (4ng/g daily) for ten weeks. Biochemical parameters, biopsy analysis, qRT-PCR (RIPK3, Bax, BCL-2 and P53), protein expression of Ki67, pBCL-2 and the apoptotic index by the terminal-deoxynucleotidyl transferase-mediated nick end-labeling (TUNEL) was assayed. All data were statistically analyzed by GraphPad Prism v9 software.

Results: DHA+MaR1 animals, levels of AST, ALT, and albumin were normalized compared to DEN alone. Inflammation and necrotic areas were reduced by DHA+MaR1 treatment, improving liver cytoarchitecture. Cell proliferation, evaluated as mitotic activity index, was increased in the MaR1 group. Upregulation of Ki67, P53, and Bax was observed in the DHA+MaR1 groups, while the expression of Bcl-2 and