Methods: This randomized controlled trial included 141 HCV who received double dose (40ug) or standard dose (20ug) and 70 healthy volunteers who received standard dose (20ug) at 0, 1 and 6 months. Anti-HBs titers were measured at 1 month after last dose. Vaccine response was defined by anti-HBs ≥10 U/L. Non-responders received the fourth dose according to the group that were previously randomized. Multivariate regression was modeled as a logistic regression.

Results: 128 completed the study. Median age 51 years, 61% female, 52% White, 40% F2-3, and 75% GT1, median 6 log10 HCV RNA. Overall seroconversion rate was 76.7% (n=60) in double dose and 73.5% (n=68) in standard dose, compared to 91.2% in controls (n=68). 23 patients received the fourth dose; 7 seroconverted (30.4%) and seroconversion rate for double and standard doses were 42.9% and 11.1%, respectively (p=0.18). Controlling for confounders, only older age (p<0.001) and GT1 (p=0.005) were associated with a decreased anti-HBs response.

Conclusion: In HCV-infected patients without cirrhosis, responses to HBV vaccination are significantly impaired and this reduced response cannot be overcome by the use double dose. Besides that, 4th dose HBV vaccination can be a strategy efficacious this vulnerable population.

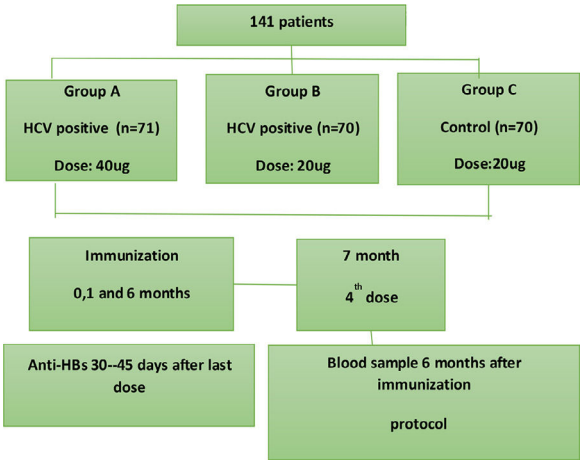

Figure 1- A randomized study comparing two doses of anti-HBV vaccination.

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O-19 INCIDENCE, PATTERN OF PRESENTATION AND RISK FACTORS FOR HEPATOCELLULAR CARCINOMA AFTER DIRECT ACTING ANTIVIRAL TREATMENT IN PATIENTS WITH HEPATITIS C VIRUS CIRRHOSIS

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Background: Brazilian public health system currently provides universal free all oral direct-acting antiviral (DAA) therapy for patients with hepatitis C virus (HCV) infection. Despite high rates of sustained virological response (SVR), patients with cirrhosis remain at risk for hepatocellular carcinoma (HCC).

Objectives: The aim of this study was to investigate incidence, risk factors and tumor pattern at presentation in a cohort of Brazilian HCV-related cirrhotic patients treated with DAAs.

Methods: This prospective cohort study included patients with HCV-related cirrhosis treated with DAAs and followed for at least 24 weeks after therapy at the Viral Hepatitis Outpatient Clinic of Hospital de Clinicas de Porto Alegre, Brazil, between August 2016 and November 2017. Ultrasound screening was performed within 24 weeks before DAA therapy and patients with presumed past or current HCC were excluded. Primary outcome was HCC incidence. Secondary outcomes were risk factors for HCC ocurrence and tumor pattern at presentation. Multivariate analysis was used to identify independent variables associated with HCC development.

Results: A total of 234 patients with HCV cirrhosis were included. Fifty-six percent were males with a mean age of 61.2±10.9 years. Overall SVR was 97.4%. Child-Turcotte-Pugh (CTP) A, B and C at baseline was found, respectively, in 89.3%, 9.4% and 1,3%. Mean Model for End Stage Liver Disease (MELD) score was 9.17 ± 2.82. Esophageal varices were found in 43.6% of the patients. Type 2 diabetes was present in 18.8%. *De novo* HCC was diagnosed in 9% (21/234) of the patients during follow-up. Tumor pattern at presentation according to BCLC staging was 0, A, B, C and D in 19,1%, 47.6%, 4.8%, 28.6% and 0%, respectively. Multivariate analysis showed significant relative risk (RR) for HCC occurrence associated with the following variables: baseline MELD score ≥10 (RR: 1.8; p=0.05); absence of SVR (RR: 6.9; p=0.04); baseline platelet count <120 × 10^9/L (RR: 5.0; p=0.04) and baseline albumin level <3.5 mg/dL (RR: 4.6.

Conclusions: A high incidence of HCC was found after DAA therapy compared to the literature, particularly among patients with more advanced cirrhosis, particularly those with baseline albumin levels < 3.5 g/dL plus platelets < 120 × 10^9/L. Absence of SVR was also significantly associated with HCC development. The majority of patients presented with very early (BCLC 0) or early (BCLC A) HCC, although a significant proportion showed advanced stage (BCLC C) at presentation.

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O-20 ASSOCIATION BETWEEN UNCOUPLING PROTEIN 3 POLYMORPHISMS AND NONALCOHOLIC FATTY LIVER DISEASE AND METABOLIC SYNDROME

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