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Introduction and Objectives: Previously published regional real-world results of overall survival (OS) in Barcelona Clinic Liver Cancer (BCLC) B and C patients demanded a prospective cohort study nested in a systematic and continuous medical educational networking group. This study aimed to describe and evaluate the treatment decisions in patients with hepatocellular carcinoma (HCC) within BCLC B and C stages.

Materials and Methods: A multicenter prospective cohort study, conducted in different Latin American centers from Argentina, Brazil and Colombia, started on 15th May 2018 (delayed recruitment during COVID locked-down period). Patients within BCLC B or C stages were included. Survival, tumor progression and patterns of treatment suspension were evaluated.

Results: At this second interim analysis (projected final analysis March 2023), 390 HCC BCLC-B or C patients were included (n=15 excluded); mean age 65 years, 75.6% males and 89.5% cirrhotic. Median OS since HCC diagnosis was 27.2 months. Among BCLC-B patients, the most frequent therapy was transarterial chemoembolization (TACE, 42.3%); 51.8% using drug-eluting beads and 47.4% conventional TACE; with a median OS since 1st TACE of 41.9 months. Similar radiological responses after 1st TACE were observed between both modalities. Overall, 48.2% of the cohort received systemic therapy for HCC (n=188), 23.7% still on BCLC-B stage. The most frequent systemic treatments were Sorafenib (74.5%), atezolizumab bevacizumab (17.5%), and lenvatinib (12.2%), with a median OS since systemic therapy of 15.7 months. Lenvatinib or atezolizumab bevacizumab was used as the second line following sorafenib in 5 and 3 patients, respectively. The most common causes of systemic treatment discontinuation were tumor progression and liver function deterioration (15% to 36.4%). Patterns of tumor progression were not specifically associated with prognosis or treatment discontinuation.

Conclusions: Liver function deterioration occurs in a third of patients following systemic therapies. The complexity of treatment decisions underly the need for a multidisciplinary team and the role of hepatologists.

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P- 11 PROTUMORIGENIC GALECTINS 1 AND 3 ARE UPREGULATED IN THE LIVER OF MICE EXPOSED TO CONTINUOUS GROWTH HORMONE LEVELS

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Introduction and Objectives: Human and animal evidence revealed a link between growth hormone (GH) and cancer risk. GH excess is implicated in rodent hepatocarcinogenesis. Transgenic mice overexpressing GH (GH-Tg) develop hepatocellular tumors at old ages, with preneoplastic liver pathology similar to that observed in humans at a high risk of developing hepatic cancer. Galectin 1 (GAL1) is involved in liver tumorigenesis in humans. We reported that GAL1 is upregulated in GH-Tg mice liver, even before histopathological alterations are detected, and particularly enhanced in liver tumors. This study aimed to evaluate if GH modulates the hepatic expression of GAL3, another protumorigenic galectin. As many proteins exhibit sexually dimorphic liver expression, mainly determined by distinct GH secretion patterns between males (intermittent) and females (more continuous), we assessed if GAL1 and GAL3 liver expression was affected by GH secretion patterns.

Materials and Methods: Hepatic GAL1 or GAL3 were analyzed by immunoblotting in GH-Tg mice exposed to continuously elevated GH levels and in Swiss-Webster mice treated with GH during five weeks by implantation of osmotic pumps (continuous treatment) or by two daily injections (intermittent treatment). Statistics: Student's t-test or two-way ANOVA; $P<0.05$, significant; at least nine animals/experimental group.

Results: In GH-Tg mice (both sexes), GAL3 was not increased in the liver at early ages, when minimal histopathological alterations are found, but it was upregulated in young adults with preneoplastic livers and in older mice that develop liver tumors. However, GAL3 was not increased in tumors compared with the adjacent non-tumoral region. In Swiss-Webster mice, GAL1 and GAL3 expression were higher in females than in males. GH continuous treatment produced a significant increase in GAL1 and GAL3 expression in both sexes and loss of sexual dimorphism, while GH injections showed no effect.

Conclusions: GH continuous exposure upregulates protumorigenic GAL1 and GAL3 in mice liver. More studies are required to evaluate its impact on humans.

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P- 12 PATHOGENIC VARIANT OF PNPLA3 DOES NOT ASSOCIATE WITH HEPATOCELLULAR CARCINOMA IN SOUTH AMERICANS. A REPORT FROM THE ESCALON NETWORK

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