

Area Under the Curve (AUC) values of 0.703, 0.683, and 0.704, respectively ( $p < 0.001$ ). Kaplan-Meier survival analysis showed better outcomes for patients with  $\text{PhA} > 3.8^\circ$  ( $p = 0.001$ ). Two multivariate models were developed to account for collinearity: in both, a lower  $\text{PhA} < 3.8^\circ$  was associated with a higher mortality risk, with Hazard Ratios (HR) of 1.98 (1.02-3.84) and 2.01 (1.02-3.94), independent of MELD score and Child-Pugh stage, respectively.

**Conclusions:** Sarcopenia, assessed by phase angle, is associated with complications before and after liver transplantation, particularly infections, higher number of hospitalizations, and increased mortality.

**Ethical statement:** This study was approved by the local ethics committee and conducted according to its standards.

**Declaration of interests:** None

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### Liver transplant in syndromic biliary atresia

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**Introduction and Objectives:** Biliary atresia (BA) is an obliterative cholangiopathy, there are at least two phenotypes, one of them is the syndromic form, which occurs in 10-15% of cases, it is associated with polysplenia, heart disease, heterotaxy and malrotation intestinal. The objective of this review is to present the experience when performing liver transplantation

**Materials and Patients:** Female patient daughter of a diabetic mother, carrier of BA type III who underwent Kasai surgery at 89 days of life, carrier of intestinal malrotation, preduodenal portal vein, intraventricular communication of 1.7 mm without hemodynamic repercussion and dyslipidemia, without biliary clearance and pondostatural arrest, worthy of performing a liver transplant from an unrelated living donor, due to complications of cirrhosis such as ascites, malnutrition and cholestasis, at the time of surgery, annular pancreas, were found as additional findings to those described agenesis of cava, agenesis of the celiac trunk, presented early partial thrombosis of the portal vein, meriting anticoagulant and antithrombotic treatment, with resolution of the condition, without requiring surgical intervention. Currently, after one year of follow-up with adequate evolution, without cholestasis or transaminasemia, adequate growth, immunosuppression with a calcineurin inhibitor, the dyslipidemia resolved. Our patient does not have polysplenia.

**Results:** The clinical case of syndromic BA is presented, although BA is rare, the syndromic presentation is even more, so we present a successful case, with complex vascular malformations combined with extrahepatic malformations, mainly cardiac, with which mortality at time of performing the transplant is high, greater than 90%. Our patient had a satisfactory surgical and clinical evolution.

**Conclusions:** BA is the main cause of liver transplantation in pediatrics; reported cases of the syndromic type are rare. The complete evaluation and planning of possible vascular malformations associated at the time of transplantation should alert the medical and surgical team.

**Ethics statement:** No patient-identifying data is used in this presentation.

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### PAI-1 participation in hepatocyte epithelial-mesenchymal transition induced by HCV NS5A or Core proteins.

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**Introduction and objectives:** Chronic HCV infection induces the development of liver fibrosis mediated by hepatocyte epithelial-mesenchymal transition (EMT). Plasminogen activator inhibitor (PAI-1) has been associated with fibrosis. Objective: To evaluate the participation of PAI-1 in the EMT of hepatocytes induced by HCV proteins.

**Materials and patients:** Huh7 cells were transfected with 1  $\mu\text{g}$  of the plasmid to express HCV NS5A or Core proteins, cells were co-cultured with hepatic stellate cells (LX2), at 48 and 72 hours of co-culture, the expression of LX2 activation biomarkers were determined by western blot and RT-qPCR. Likewise, transcriptional expression of 84 genes associated with fibrosis in Huh7 was determined by RT-qPCR array. Bioinformatic analyses were performed with Enrichr and STRING. *serpine1* (PAI-1) was selected as one of the differentially expressed genes, Huh7 cells were transfected with NS5A or Core, and after 24 hours gene silencing was performed with siPAI-1 (5nM) and pharmacological treatment with TM5275 (25  $\mu\text{M}$ ) to inhibit the PAI-1 function. After 24 and 48 hours of treatment, the expression of the viral proteins was validated by chemiluminescence and western blot. Likewise, the PAI-1 inhibition was validated and the translational expression of EMT markers (TGF $\beta$ 1, Snail, E-cadherin, and Vimentin) was evaluated in Huh7 cells by western blot and densitometric analysis.

**Results:** NS5A and Core expression in Huh7 cells co-cultured with LX2 cells, induced transcriptional overexpression of TGF $\beta$ 1, Col1 $\alpha$ 1, and Timp1, suggesting LX2 activation. We observed 28 genes differentially expressed in Huh7 (NS5A+) and 46 genes were differentially expressed in Huh7 (Core+) during co-culture with LX2 cells. Bioinformatics analyses were performed, and *serpine1* (PAI-1) was identified as a differentially expressed gene. On the other hand, at the translational level, NS5A induced the overexpression of TGF $\beta$ 1 and Snail (4-fold) and subexpression of E-cadherin (0.6-fold). Likewise at the translational level, Core induced the overexpression of Snail (2.5-fold) and subexpression of E-cadherin (0.4-fold), compared to the control, suggesting the EMT of Huh7. A gene silencing of 60% of PAI-1 was obtained in all groups. this silencing induces a reduction of 50% of vimentin expression at the translational level in all groups. On the other hand, TM5275 decreased the expression of TGF $\beta$ 1 by 60% both in the control group and in the NS5A transfected cells. Likewise, TM5275 increased the expression of E-cadherin at the translational level by 60% both in the control group and in the Core transfected cells.

**Conclusions:** HCV proteins regulate the expression of molecular markers and signaling pathways in hepatocytes associated with the development of EMT, such as PAI-1. At the same time, PAI-1 inhibition negatively regulates this EMT, which is important to understand the pathophysiology of HCV damage.

**Ethical statement:** not applicable