Impact of all-oral antiviral therapy on portal pressure and hemodynamics on HCV-infected cirrhotic patients.

Sabela Lens1, Edilmar Alvarado-Tapias1, María Carola Londoño1, Elba Llop1, Javier M. González1, José Ignacio Fortea1, Luis Ibáñez Samaniego1, Rafael Bañares6, Virginia Hernandez-Gea1, Jaime Bosch1, Cándido Villanueva2, Xavier Forns1, Juan Carlos García-Pagan1, Javier M. González4, Jose Ignacio Fortea5, Luis Ibáñez Samaniego6, Rafael Bañares6, Virginia Hernandez-Gea1, Jaime Bosch1, Cándido Villanueva2, Xavier Forns1, Juan Carlos García-Pagan1, Virginia Hernandez-Gea1, Jaime Bosch1, Cándido Villanueva2, Xavier Forns1, Juan Carlos García-Pagan1, Virginia Hernandez-Gea1, Jaime Bosch1, Cándido Villanueva2, Xavier Forns1, Juan Carlos García-Pagan1

1Hospital Clinic, IDIAPS, CIBEREHD, 2Hospital de la Santa Creu i Sant Pau, CIBEREHD, 3Hospital Puerta del Hierro, Universidad Autónoma, CIBEREHD, 4Hospital Ramon y Cajal, Universidad de Alcalá, CIBEREHD, 5Hospital Universidad Marqués de Valdecilla. Universidad de Cantabria. IDIVAL. CIBEREHD, 6Hospital General Universitario Gregorio Marañón, Universidad Complutense, IISGM, CIBEREHD

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Abstract Body

Background:
Data on the hemodynamic changes induced by sustained virological response (SVR) after all-oral therapy in patients with clinical significant portal hypertension (CSPH, HVPG≥10mmHg) are scarce. Previous data suggest that patients with CSPH, despite achieving SVR, remain at risk of liver decompensation (LD).

Methods:
Multicenter prospective study of patients with HCV-related cirrhosis and CSPH before all-oral antiviral therapy (BL or baseline). By study protocol, patients underwent HVPG, right-heart catheterization and liver stiffness measurement (LSM) at BL and 24 weeks after end of treatment if SVR (FU or follow-up). Patients starting beta-blocker (BB) therapy between HVPG measures were excluded.

Results:
118 cirrhotic patients with CSPH were included. Most patients (92%) were CTP-A; 80% had esophageal varices (40% large) and 31% had at least one previous LD (14% variceal bleeding, 21% ascites). Overall, HVPG decreased from 16.4±4.5 to 14.5±4.6 mmHg after SVR (mean change -1.9±3; p<0.01). A clinically relevant decrease (≥10%) was observed in 65 (54%) patients (≥20% in 34%). In multivariate analysis, the only variable associated with ≥10% decrease was BL-HVPG (OR 0.22[0.07-0.66];p<0.01). After achieving SVR, CSPH persisted in 86% of patients. Decrease in mean HVPG after antiviral therapy was similar in patients with (n=52) or without BB, however, due to higher BL-HVPG, CSPH persisted in 95% of patients with BB compared to 77% without BB (p<0.01). In 82 patients with paired LSM, BL-LSM was 31±15kPa with a mean reduction of 6 ±12kPa after SVR (p <0.05). Previously described cut-offs of 13.6 and 21kPa presented high NPV (92%) and PPV (97%) for the presence of CSPH on follow-up, respectively. Pared right-heart catheterization (n= 82 patients) showed a significant rise in MAP due to increased systemic vascular resistance (+14% and +25%, p<0.05) with stable cardiac output. Interestingly, mPAP and pulmonary vascular resistance also rose after therapy (+15% and +21%; p<0.05). Indeed, pulmonary arterial hypertension (mPAP≥25mmHg) developed or exacerbated in 9 and 4 patients, respectively but only 2 presented increased pulmonary vascular resistance.

Conclusions:
Despite achieving SVR, CSPH persists 24 weeks after therapy in most patients with HCV-related cirrhosis treated with all-oral antiviral therapy, indicating risk of decompensation. Previously described LSM cut-offs to rule-in or out CSPH are still useful after SVR. Interestingly, improvement of systemic hemodynamics after SVR was associated with pulmonary hypertension in some patients, indicating the need for continued careful monitoring on long-term follow-up.