# ACUTE VOLUME EXPANSION AND TIPS FAILS TO INDUCE CHANGES OF IMPENDING HEART FAILURE IN PATIENTS WITH REFRACTORY ASCITES

**D. Filì, C. Falletta, C. Hernandez-Baravoglia, R. Miraglia, G. Vizzini, G. Di Gesaro, C. Minà, F. Tuzzolino, A. Luca, B. Gridelli, J. Bosch**

Mediterranean Institute for Transplantation and Advanced Specialized Therapies (ISMETT), Palermo, Italy - University of Pittsburgh Medical Center (UPMC), Pittsburgh, USA

Hepatic Hemodynamic Laboratory, Hospital Clínic-IDIBAPS and Centro de Investigación Biomédica en Red de Enfermedades Hepáticas y Digestivas (CIBERehd), Barcelona, Spain

## BACKGROUND

Patients with decompensated cirrhosis, mainly those with refractory ascites and/or renal failure exhibit ECG/Ecocardiographic features of so-called "cirrhotic cardiomyopathy". It has been proposed that this may put them at risk of heart failure in stress circumstances, such as after TIPS.

## AIM

To evaluate in patients with cirrhosis requiring an elective TIPS for refractory ascites, if acute volume expansion may elicit signs of impending heart failure, and if this can predict hemodynamic changes occurring after TIPS.

## METHODS

15 patients with decompensated cirrhosis (Child Pugh B-C) and refractory ascites referred for TIPS and 8 patients with compensated cirrhosis (Child Pugh A) were included. Patients with significant valvular or ischemic heart disease, chronic renal failure (creatinine > 2.5 mg% or on hemodyalisis), total portal vein thrombosis, contraindications to beta blocker withdrawal or active alcohol assumption within one month were excluded. All patients underwent right heart catheterization (RHC) and hemodynamic measurements at baseline and after rapid volume expansion (Voluven®, 500 ml iv/ 10 min). In addition, patients with refractory ascites underwent RHC before TIPS, immediately after and at 24-hour. Right atrial pressure (RAP, mm Hg), pulmonary artery pressures (PAP, mm Hg), pulmonary capillary wedge pressure (PCWP, mm Hg), cardiac output (CO, l/min), cardiac index (CI l/min/m²), systemic vascular resistance (SVR, dine/sec/cm), heart rate (HR, beats/min) and blood pressure (BP, mm Hg) were measured.

## RESULTS

Between May 2008 and October 2011 we selected 15 patients with decompensated cirrhosis and refractory ascites referred for TIPS (Group A) and 8 patients with compensated cirrhosis (Group B). Patients baseline characteristics are listed in table 1.

| Variable | Group A | Group B |
|---|---|---|
| **Age** yr (average-range) | 54.4 (25-68) | 56.1 (46-62) |
| **Gender** (M/F) | 12/3 | 8/- |
| **Etiology** | | |
| Viral cirrhosis | 12 | 7 |
| Alcohol cirrhosis | 3 | 1 |
| **Child Pugh Score** | | |
| A | - | 8 |
| B | 7 | - |
| C | 8 | - |

Table 1. Baseline characteristics of patients with refractory ascites (Group A) and with compensated cirrhosis (Group B)

Table 2 summarize the hemodynamic data obtained in patients with decompensated cirrhosis before and after Voluven® infusion and before and after TIPS insertion. Data are shown as Mean (SD).

Volume expansion induced a significant increases in RAP (p<.01), mean PAP (p<.01), PCWP (p<.01), CO (p<.01) and CI (p<.01), whereas HR did not change significantly (79 *vs* 81 beats/min, p=0.4).

TIPS insertion caused similar changes: it induced a significant increase in RAP (p<.01), mean PAP ( p<.01), PCWP (p<.01), CO (p<.01) and CI (p<.01) with no significant changes in HR ( p=.2).

At 24 hours, cardiopulmonary pressures returned towards baseline, whereas CO (9.5 l/min, p<.01) and CI (5.2 l/min/m², p<.01) continued elevated but now due to a significant increase in HR (92 beats/min, p<.05). No patient developed any sign of impending heart failure.

| | ®Voluven infusion | | | TIPS insertion | | | | |
|---|---|---|---|---|---|---|---|---|
| **Variables** | **Baseline** | **After** | **p*** | **Before** | **Soon After** | **p**** | **24 hours after** | **p***** |
| **RAP** | 4.7 (2.8) | 9.9 (3.6) | <.01 | 10.1 (3.3) | 14.2 (3.4) | <.01 | 6 (2.9) | NS |
| **PAP, mean** | 13.3 (3.5) | 21.9 (5.9) | <.01 | 17.5 (4) | 25.2 (4.2) | <.01 | 15.9 (4.4) | <.05 |
| **PCWP** | 8.3 (3.4) | 15.4 (4.7) | <.01 | 12.3 (4) | 19.4 (3.4) | <.01 | 10.2 (3.8) | NS |
| **CO** | 6.8 (1.4) | 8.2 (1.9) | <.01 | 6.2 (1.8) | 8.2 (1.6) | <.01 | 9.5 (1.8) | <.01 |
| **CI** | 3.7 (0.7) | 4.6 (1) | <.01 | 3.4 (0.8) | 4.5 (0.9) | <.01 | 5.2 (0.9) | <.01 |
| **SVR** | 961 (278) | 767 (285) | <.01 | 779 (262) | 596 (199) | <.01 | 570 (186) | <.01 |
| **HR** | 79 (12) | 81 (11) | NS | 81 (11.4) | 83 (11.6) | NS | 92 (17.8) | <.05 |
| **BP mean** | 83 (13) | 84 (12) | NS | 69.1 (4.2) | 76.2 (10.2) | <.01 | 75 (12.1) | <.05 |

Table 2: Hemodynamic data before and after Voluven® infusion and before and after TIPS insertion in patients with refractory ascites. p*: p value vs baseline; p**: p value vs before TIPS; p***: p value vs baseline

Patients with compensated cirrhosis exhibited an almost identical response to volume load as patients with decompensated cirrhosis: indeed, Voluven® infusion induced a significant increase in RAP (p<.05), mean PAP (p<.01), PCWP (p<.01), CO (p<.01) and CI (p<.01) with no significant changes in HR (p=.4). (NS Vs. changes in patients with refractory ascites). Table 3.

| | ®Voluven infusion | | |
|---|---|---|---|
| **Variables** | **Before** | **Soon after** | **p** |
| **RAP** | 7.6 (6) | 11.3 (4.4) | <.05 |
| **PAP, mean** | 14.3 (4.5) | 20.6 (3.) | <.01 |
| **PCWP** | 10.8 (5.5) | 16.6 (4.1) | <.01 |
| **CO** | 5.9 (1.5) | 6.9 (0.9) | <.01 |
| **CI** | 3.1 (0.6) | 3.6 (0.5) | <.01 |
| **SVR** | 1082 (273) | 908 (134) | NS |
| **HR** | 68 (4) | 71 (8) | NS |
| **BP mean** | 86 (12) | 89 (8) | NS |

Table 3: Hemodynamics before and after Voluven® infusion in compensated cirrhotic patients

## CONCLUSIONS

**Acute volume expansion increased cardiopulmonary pressures and CI in patients with cirrhosis. Contrary to what expected, response to acute volume load was not worse in patients requiring TIPS for refractory ascites, and predicted hemodynamic changes immediately after TIPS. No patient developed signs of impending heart failure. Increased cardiopulmonary pressures had returned to baseline 24-hours after TIPS suggesting restored volume handling and diuresis, but CI persisted elevated at the expense of increased heart rate. This hemodynamic behavior is against what is expected in a true cardiomyopathy, suggesting that significant cirrhotic cardiomyopathy is extremely rare and does not represent a real risk in patients requiring TIPS.**