

P-25 IMPEDANCE CARDIOGRAPHY AND SPLEEN STIFFNESS MEASUREMENT TO ASSESS THERAPEUTIC RESPONSE IN CIRRHOTIC PATIENTS TREATED WITH NON-CARDIOSELECTIVE BETA-BLOCKERS

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Conflict of interest: No

Introduction and Objectives: Non-cardioselective beta-blockers (NCBBs) are used as prophylaxis for variceal bleeding but have limitations in therapeutic follow-up. Impedance cardiography (IC) evaluates systemic hemodynamics, and splenic elastography (SE) quantifies spleen stiffness. A decrease in spleen stiffness measurement (SSM) is associated with a reduction in the hepatic venous pressure gradient, which is the ultimate goal of the treatment. This study aimed to describe systemic hemodynamic changes and SSM in cirrhotic patients under prophylaxis with NCBBs using non-invasive methods.

Patients / Materials and Methods: This observational and prospective study involved cirrhotic patients indicated for NCBB treatment at the Military Hospital from July 2022 to June 2024. Hemodynamic assessment was performed using IC with Z_logic® (Exxer®, Argentina) and SSM with FibroScan® (Echosens®, France). Patients were evaluated before treatment and at the target dose.

Results and Discussion: Twenty-six patients participated in the study, of which 14 were men. The mean age was 57.8 ± 18.4 years. Alcoholic cirrhosis was the main etiology (n=10). 69% were classified as Child-Pugh A. The MELD-Na score was 11.8 ± 5.3. Before treatment, patients did not present parameters of hyperdynamic circulation, and the SSM was 58.9 ± 15.1 kPa. In 19 patients, there was a decrease in SSM, with an average value dropping to 47.6 ± 17.3 kPa (p=0.018). Systemic vascular resistance (SVR) was higher in patients with a decrease in SSM (1538.8 ± 1068.9 dyn.s.cm⁻⁵ vs. 985.9 ± 164.3 dyn.s.cm⁻⁵, p=0.042) (Table). A negative correlation was observed between the change in SVR and the decrease in SSM (p=0.029, Pearson's r = -0.438).

Conclusions: Systemic hemodynamic changes and SSM in NCBB-treated patients were described. SSM showed the most significant changes. A correlation was found between the increase in SVR and the decrease in SSM once the target doses were achieved. According to these findings, SVR values could be a marker of an adequate response to NCBBs.

	Decrease in SSM (n = 19)	No decrease in SSM (n = 7)	P value
Heart Rate (beats/min)*	61 (54 – 68)	67 (56 – 87)	0.072
Cardiac Index (L/min/m ²)*	2.8 (1.7 – 4.1)	3.6 (2.7 – 4.4)	0.646
Cardiac Output (L/min)	5.8 +/- 3.5	6.6 +/- 1.9	0.595
Systolic Blood Pressure (mmHg)	110.4 +/- 17.4	113.3 +/- 17.2	0.718
Diastolic Blood Pressure (mmHg)	65.7 +/- 12.2	66.7 +/- 10.8	0.869
Systolic Discharge (ml/min)	91.9 +/- 52.2	99.6 +/- 48.1	0.753
Ventricular-Arterial Coupling Capan: EA/EES	1.0 +/- 0.3	1.0 +/- 0.3	0.956
Ventricular-Arterial Coupling Weissler: EA/EES	1.0 +/- 0.4	1.0 +/- 0.3	0.998
Arterial Compliance (ml/mmHg)*	1.6 (1.2 – 3.0)	2.0 (1.3 – 2.7)	0.886
Thoracic Fluid Content	44.6 +/- 7.1	45.3 +/- 10.4	0.841
Thoracic Fluid Content Normalized by Regression	103.7 +/- 17.0	105.2 +/- 19.6	0.859
Effective Aortic Elastance	1.5 +/- 0.9	1.2 +/- 0.5	0.483
Left Ventricular End-Systolic Elastance Capan*	1.1 (1.0 – 1.5)	1.1 (1.0 – 1.3)	0.370
Left Ventricular End-Systolic Elastance Weissler*	1.2 (1.0 – 1.6)	1.1 (1.1 – 1.2)	0.354
Ejection Fraction Capan (%)	56.6 +/- 5.3	55.0 +/- 6.9	0.571
Ejection Fraction Weissler (%)	58.9 +/- 10.3	55.9 +/- 13.5	0.571
IC/SBP (ml/mmHg.m ²)*	24.4 (15.4 – 36.8)	30.5 (26.7 – 36.2)	0.897
Arterial Compliance Index (ml/mmHg.m ²)*	1.0 (0.6 – 1.5)	1.2 (0.7 – 1.4)	0.851
Systolic Discharge Index (ml/beat/m ²)*	47.8 (26.9 – 55.7)	50.9 (30.5 – 79.5)	0.813
Systemic Vascular Resistance Index (dyn.sec.cm ⁻⁵ .m ²)*	2253.5 (1436.9 – 3590.7)	1823.9 (1571.6 – 2014.2)	0.040¶
Mean Arterial Pressure (mmHg)	80.6 +/- 13.7	82.2 +/- 12.8	0.801
Pulse Pressure (mmHg)*	45 (40 – 50)	45 (40 – 53)	0.584
SVR: Suga Index: End-Systolic Pressure/Volume Ratio (mmHg/mL)*	1.4 (1.2 – 3.6)	1.6 (1.0 – 2.0)	0.329
Systemic Vascular Resistance (dyn.sec.cm ⁻⁵)	1538.8 +/- 1068.9	985.9 +/- 164.3	0.042¶

Hemodynamic status in patients who showed a decrease or no decrease in spleen stiffness value under treatment with NCBBs. * Median and interquartile range are provided for these data. Remaining values are expressed as mean ± standard deviation. (¶: p < 0.05).

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P-26 EPIGENETIC MARKS IN PATIENTS WITH SUSTAINED VIRAL RESPONSE TO HCV AND RESIDUAL LIVER FIBROSIS ARE RESTORED BY PROLONGED-RELEASE PIRFENIDONE

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Introduction and Objectives: Patients with residual liver fibrosis following the eradication of hepatitis C virus (HCV) represent a significant clinical challenge due to the continuous risk of disease progression and the development of hepatocellular carcinoma. This study aimed to evaluate the efficacy of a 12-month treatment with