

Figure 2. Educational Outcomes – HCC Clinical Excellence Program



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NON-INVASIVE HEMODYNAMIC ASSESSMENT IN CIRRHOSIS: CHILD-PUGH STRATIFICATION AND A PLATELET THRESHOLD TO DEFINE THE HYPERDYNAMIC STATE

Martín Elizondo¹, Eugenia Ipar², Romina Rey¹, Leandro Cymberknop², Marcelo Valverde¹, Solange Gerona¹, Ricardo Armentano²

¹ Unidad Bi-Institucional de Enfermedades Hepáticas Complejas (Hospital Militar. Hospital de Clínicas). Programa de Trasplante Hepático. Montevideo. Uruguay.

² Universidad Tecnológica Nacional. Facultad Regional Buenos Aires. Grupo de Investigación y Desarrollo en Bioingeniería (GIBIO). Buenos Aires. Argentina.

Introduction and Objectives: Cirrhosis initially presents a hyperdynamic profile—elevated cardiac output (CO), reduced systemic vascular resistance (SVR) and arterial pressure—which may later evolve into low-output cardiocirculatory failure. Impedance cardiography (IC) enables non-invasive quantification of these changes. To integrate them, we developed the unitless Cardiac Haemodynamic Status (CHS) index, defined as $\sqrt{(\text{CO}^2 + \text{SVR}^2 + \text{arterial compliance} [\text{AC}]^2)}$, with all parameters standardised prior to calculation. This study aimed to compare CO, SVR, AC and CHS across healthy controls and cirrhotic patients stratified by Child-Pugh class (A/B/C), and to determine whether a platelet threshold lower than the conventional $140 \times 10^3 / \mu\text{L}$ more accurately identifies the hyperdynamic circulatory phenotype.

Materials and Methods: Cross-sectional study (2023–2025) of 12 controls and 40 β -blocker-free cirrhotics (A 20, B 12, C 8). Each subject underwent IC. Normality was checked (Shapiro–Wilk); groups were compared with Kruskal–Wallis and Holm-adjusted Mann–Whitney tests. All observed platelet counts ($40\text{--}190 \times 10^3 / \mu\text{L}$) were screened; the cut-off with the lowest p and highest Youden index for CHS was validated by bootstrap ROC.

Results: CO, SVR, AC and CHS differed among the four groups ($p \leq 0.006$) (Table). Versus controls, C-P A showed higher SVR/CHS but lower CO/AC; C-P B displayed an isolated CO rise; C-P C had higher SVR/CHS (all $p < 0.04$). A platelet threshold of $93 \times 10^3 / \mu\text{L}$ optimally discriminated hyperdynamism ($p = 0.006$; Cohen d 0.86; AUC 0.82). Patients below this level had higher CO, AC and CHS and lower SVR.

Conclusions: IC identifies three distinct haemodynamic phenotypes across Child-Pugh classes. The CHS index captures these profiles, while a platelet count below $93 \times 10^3 / \mu\text{L}$ appears to be a useful surrogate of hyperdynamic circulation.

Conflict of interest: None

Variable	Controls	Child A	Child B	Child C	p
Cardiac output (CO) (L·min ⁻¹)	6.78 ± 1.41	4.76 ± 1.48	8.27 ± 4.10	6.20 ± 3.25	0.006
Systemic vascular resistance (SVR) (dyn·s·cm ⁻⁵)	892 ± 207	1 677 ± 801	1 024 ± 608	1 147 ± 421	0.002
Arterial compliance (AC) (mL·mmHg ⁻¹)	2.69 ± 0.75	1.47 ± 0.52	2.41 ± 0.98	1.97 ± 0.93	0.001
CHS index	0.93 ± 0.21	1.74 ± 0.81	1.12 ± 0.61	1.21 ± 0.42	0.002

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IRON METABOLISM DISTURBANCES ARE ASSOCIATED WITH LIVER FIBROSIS SEVERITY IN MASLD: A CROSS-SECTIONAL STUDY USING NON-INVASIVE TOOLS

Romina Rey Laguarda¹, Martín Elizondo Barceló¹, Lain Lin Liu², Emilia Moreira Milanesi³, Solange Gerona Sangiovanni¹

¹ Hospital Militar, Uruguay.

² Hospital Maciel, Uruguay.

³ Hospital de Clínicas, Uruguay.

Introduction and Objectives: Metabolic dysfunction-associated steatotic liver disease (MASLD) is the most prevalent chronic liver disease worldwide. Disturbances in iron metabolism—particularly hyperferritinemia—may play a role in fibrosis progression through mechanisms involving oxidative stress and chronic inflammation.

To describe iron metabolism parameters in patients with MASLD and analyze their association with liver disease severity using non-invasive tools.

Materials and Methods: We conducted a cross-sectional study including 199 adult patients with MASLD followed at a specialized hepatology clinic (2022–2024). Clinical, anthropometric, and biochemical variables were collected, including serum ferritin, transferrin, serum iron, and transferrin saturation index (TSI). Liver fibrosis was evaluated by FIB-4 score and transient elastography (FibroScan®); steatosis was assessed by controlled attenuation parameter (CAP). Non-parametric statistical tests were applied (Spearman correlation, Kruskal–Wallis, chi-square).

Results: The mean age was 57 ± 12 years; 58.3% were women and 39.7% had type 2 diabetes. The mean BMI was $33.8 \pm 6.3 \text{ kg/m}^2$. Hyperferritinemia was observed in 43.4% of patients. Elevated ferritin, serum iron, and TSI were significantly associated with higher FIB-4 scores ($p < 0.05$). Ferritin levels were also significantly associated with liver stiffness measured by FibroScan® ($p < 0.05$). No significant association was found between iron metabolism parameters and the degree of steatosis assessed by CAP.

Conclusions: Iron metabolism disturbances, particularly hyperferritinemia, are frequent in MASLD and associated with greater risk of liver fibrosis, but not with steatosis. These findings support the potential utility of iron biomarkers as adjunctive non-invasive indicators of disease progression.

Conflict of interest: None