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ABSTRACT N. CS01

EMOSTASI E TROMBOSI: ASPETTI DI BASE

ELUCIDATING THE EVIL RELATIONSHIP OF METABOLIC DYSFUNCTION–ASSOCIATED FATTY LIVER DISEASE WITH CARDIOVASCULAR COMORBIDITIES: THE COMPLEX INTERPLAY AMONG HYPERCOAGULABILITY, INFLAMMATION AND LIVER FIBROSIS.

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Background and aims: Metabolic dysfunction–associated fatty liver disease (MAFLD) represents an increasingly significant public health issue. The clinical course of MAFLD can be severe, with increased risk not only of liver-related morbidity and mortality, but also of cardiovascular (CV) diseases, the leading cause of death in these patients. This study aimed to assess the overall CV risk profile using clinical, laboratory, and ultrasound parameters in a cohort of MAFLD patients stratified by liver stiffness. The project is funded by the European Union–Next Generation EU–NRRP M6C2–Investment 2.1.

Methods: Consecutive patients with confirmed MAFLD (i.e., hepatic steatosis and at least one of the following: overweight/obesity, type 2 diabetes, or normal weight with ≥2 metabolic abnormalities) were enrolled. The presence and severity of hepatic steatosis were assessed using transient elastography (FibroScan). Patients were excluded if they had recent (<1 year) arterial or venous thrombotic events, were receiving full-dose anticoagulation, had acute infection (<3 months), active cancer, or documented alcohol consumption. All patients underwent ultrasound evaluation to detect early atherosclerosis (carotid intima-media thickness [IMT], ankle-brachial index [ABI], and aortic diameter). Laboratory assessments included thromboelastometry, whole blood aggregation, and systemic inflammation via high-sensitivity C-reactive protein (hs-CRP). The primary outcome was to compare CV risk profile, early atherosclerosis, hypercoagulability, and inflammation across fibrosis stages.

Results: 268 patients with MAFLD (mean age 58 ± 13 years; 64% male) were enrolled. The mean body mass index was 29 ± 4 kg/m^2, and the mean waist circumference was 101 ± 14 cm. Obesity was present in 40% of patients, hypertension in 54%, dyslipidemia in 55%, and type 2 diabetes in 28%. Notably, 15% of patients had a history of arterial thrombosis, and 7.8% had experienced a previous venous thrombotic event. Fibrosis staging revealed that 93 patients (35%) had F0 fibrosis, 125 (46%) had F1, 29 (11%) had F2, 5 (2%) had F3, and 16 (6%) had F4. Patients with more advanced fibrosis (F2–F4) showed a significantly higher prevalence of carotid plaque ($p = 0.0028$), a trend toward lower ankle-brachial index (ABI) values (1.08 [1.00–1.15] vs. 1.11 [1.00–1.19]), and a non-significant increase in aortic diameter (19 [16.5–20] mm vs. 17.5 [16–19] mm). No significant differences in thromboelastometry or whole blood aggregometry parameters were observed across fibrosis stages. Moreover, hs-CRP levels showed a strong correlation with clot stability, as measured by maximum clot firmness (MCF) in FIBTEM, EXTEM, and INTEM assays ($r = 0.59$, $p = 0.001$). A history of thrombosis was also associated with increased FIBTEM MCF ($r = 0.27$, $p = 0.01$). Conversely, hs-CRP levels did not correlate with early atherosclerosis parameters.

Conclusions: More advanced liver fibrosis was associated with an increased prevalence of carotid atherosclerosis and a trend toward peripheral arterial impairment. While no differences in coagulation parameters were observed across fibrosis stages, inflammation was significantly associated with both fibrosis severity and clot stability. A history of venous thrombosis correlated with liver fibrosis and procoagulant profiles. These findings highlight the complex interplay between liver disease, inflammation, and thrombosis in MAFLD.

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