Dietary intake and lifestyle habits in patients with steatotic liver disease: a cross-sectional study

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Introduction: Unhealthy dietary patterns and insufficient physical activity are well-established contributors to the development of metabolic-associated steatotic liver disease (MASLD). More recently, the consumption of ultra-processed foods (UPF) has emerged as a major nutritional concern, being linked to an increased risk of MASLD. However, data remain limited regarding the detailed characterization of dietary intake and physical activity in patients with alcohol-related liver disease (ALD) and, more broadly, in patients with steatotic liver disease (SLD).

Aim: The primary aim of this study was to identify and compare dietary habits, including UPF consumption, and physical activity levels in patients with SLD versus healthy controls. The secondary aim was to evaluate the association between UPF intake and histologically assessed liver disease severity.

Methods: A total of 247 patients with SLD and 31 healthy controls were prospectively recruited. Dietary intake and UPF consumption were assessed using a 24-hour dietary recall. Food processing level was classified according to the NOVA system, and nutrient intake quantified in % of the total energy intake (TEI) and grams using the Nubelpro software. Physical activity was evaluated using the International Physical Activity Questionnaire (IPAQ). Hepatic steatosis and fibrosis were quantified by vibration-controlled transient elastography (VCTE). Finally, a subset of patients with clinically suspected advanced disease underwent liver biopsy for histopathological evaluation.

Results: Among the 247 patients with SLD, 152 had MASLD and 95 had ALD. The biopsy subgroup included 94 patients (54 MASLD, 40 ALD). As expected, patients with MASLD compared to patients with ALD and controls, exhibited significantly higher BMI (32 vs 26 vs 22 kg/m^2, $p<0.0001$), waist circumference (112 vs 100 vs 79 cm, $p<0.0001$), and HOMA-IR values (4.1 vs 2.0 vs 1, $p<0.0001$). Type 2 diabetes was common in MASLD (41%), uncommon in ALD (5%), and absent in controls. Conversely, patients with ALD compared to MASLD patients displayed higher alanine aminotransferase (69 vs 47 U/L, $p<0.0001$), and gamma-glutamyl transferase (156 vs 44 U/L, $p<0.0001$) levels. Liver stiffness measurement and controlled attenuation parameter values were elevated in both MASLD and ALD groups compared with controls (11 vs 12 vs 4 kPa $p = 0.0004$; 328 vs 299 vs 196 dB/m, $p<0.0001$).

Nutritional analysis showed that ALD patients consumed more total calories than MASLD patients (2290 vs 1620 kcal, $p<0.0001$), despite having a lower BMI, mainly due to higher alcohol intake. When expressed as percentage of TEI%, ALD patients compared to MASLD patients and controls consumed fewer proteins, lipids, carbohydrates, and dietary fibres (proteins: 11 vs 17 vs 16 %TEI, $p<0.0001$; lipids: 23 vs 37 vs 42 %TEI, $p<0.0001$; carbohydrates: 27 vs 42 vs 37 %TEI, $p<0.0001$; fibres : 0.8 vs 1.9 vs 2.4 %TEI, $p<0.0001$). In

MASLD, macronutrient distribution was similar to controls, except for a lower fibre intake (16 vs 23 g/day, $p<0.0001$). Interestingly, in the biopsied MASLD subgroup, significant fibrosis ($F\geq2$) was associated with lower fibre consumption ($F<2$: 19 vs $F\geq2$:15 g/day, $p = 0.0259$). Regarding UPF intake, ALD patients consumed markedly higher amounts of processed foods (NOVA 3) than both MASLD patients and controls (54 vs 27 vs 30 %TEI, $p<0.0001$), largely attributable to alcohol consumption. No significant differences in NOVA categories 1–4 were observed between MASLD patients and controls. Histologically, UPF consumption was not associated with the severity of the liver disease in terms of steatosis, activity, or fibrosis in either MASLD or ALD patients.

Finally, for physical activity, the total physical activity (MET-min/week) was higher in controls compared to patients with MASLD or ALD (3013 vs 1775 vs 1533 MET-min/week, $p<0.0001$). Controls dedicated a larger proportion of their activity to leisure-time exercise (54% vs. 11% in MASLD and 17% in ALD, $p<0.0001$).

Conclusions: We demonstrated that patients with SLD exhibit lower fibre intake, and reduced leisure-time physical activity compared with healthy controls. Importantly, a low fibre intake, but not UPF consumption, was linked to fibrosis severity in patients with MASLD, suggesting that specific nutrient components may be more relevant than global UPF exposure. These findings support the value of targeting physical activity and emphasize the importance of dietary fibre as a potentially modifiable factor in SLD.