

Body mass index and alcohol consumption are directly related with liver steatosis. Results from a prospective study of patients referred for hyperferritinemia



To the Editor,

Liver steatosis (LS) is often suspected in patients with hyperferritinemia (HF) and HF is a well-known risk factor for steatosis in patients with chronic liver disease [1]. Patients with a history of chronic alcohol consumption or features of MS often present with hyperferritinemia [2].

The objectives of this work were to study the body mass index (BMI) and alcohol consumption in a cohort of patients referred for HF in 6 hospitals from the Basque country, Spain, and to study the prevalence of hepatic steatosis determined by MRI in these patients [2].

We designed a prospective study of 312 consecutive patients with HF (>200 µg/L women; 300 µg/L men) to develop and validate a diagnostic algorithm for high iron overload based on laboratory and genetic variables [3]. Another objective of the study, and the aim of this work, was to study the prevalence of hepatic steatosis determined by MRI in these HF patients and the relationship with the body mass index (BMI) and alcohol consumption. MRI images were recorded with a 1.5-Tesla systems. BMI and alcohol consumption were determined in all the patients. Patients were classified as normal weight (BMI < 25.0 kg/m²), overweight (BMI ≥ 25.0 and < 29.9 kg/m²), and obese (BMI ≥ 30.0 kg/m²). Alcohol consumption was classified: women < 20 g/day and men < 40 g/day in the non-alcohol group (non-alcohol or mild consumption); women > 20 g/day and men 40 g/day alcohol group (moderate-heavy drinkers) [2,4].

Three hundred and twelve (272 men) were included in the study. In 286 patients, a MR study for the presence of liver steatosis was performed. One hundred ninety six had no steatosis and 90 hepatic steatosis (31.47%). We have studied the relationship between LS and the different BMI and Alcohol groups from the HF patients of the study (Table 1).

Table 1
Body mass index (BMI), alcohol consumption and liver steatosis (LS).

Study group (N=286)	Total	LS	% LS
BMI < 25 kg/m ²	11	52	21.15
BMI 25–30 kg/m ²	23	105	21.90
BMI > 30 kg/m ²	56	129	43.41
Non-alcohol group (g/day)	43	196	21.94
Alcohol group (g/day)	32	90	35.56

Liver steatosis was more frequent in the BMI > 30 group-obesity (56/129 patients with LS; 43.41% of the group), and the results were statistically significant (p = 0.000). When we compared the groups by alcohol consumption (no alcohol-mild consumption group vs moderate-heavy drinkers) the differences were statistically significant for the second group for LS (p = 0.015) (Table 1).

In conclusion, BMI and alcohol consumption are both directly associated with the presence of liver steatosis in patients referred for hyperferritinemia in our country.

Conflict of interest

The authors have no conflicts of interest to declare.

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References

- [1] Licata A, Nebbia ME, Cabibbo G, Lo Iacono G, Barbaria F, Brucato V, et al. Hyperferritinemia is a risk factor for steatosis in chronic liver disease. *World J Gastroenterol* 2009;15:2132–8.
- [2] Castiella A, Urreta I, Zapata E, Zubiaurre L, Alústiza JM, Otazua P, et al. Liver iron concentration in dysmetabolic hyperferritinemia: Results from a prospective cohort of 276 patients. *Ann Hepatol* 2020;19:31–5.
- [3] Zapata E, Castiella A, Urreta I, Alustiza JM, Salvador E, Otazua P, et al. Diagnostic algorithm for high iron overload: results from a prospective study of 312 patients with hyperferritinemia. *J Hepatol* 2016;64(Suppl. 2):S296.
- [4] Trombini P, Paolini V, Pelucchi S, Mariani R, Nemeth E, Ganz, et al. Hepcidin response to acute iron intake and chronic iron loading in dysmetabolic iron overload syndrome. *Liver* 2011;31:994–1000.

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