

O-22 METABOLIC ASSOCIATED FATTY LIVER DISEASE: A SEVERE LIVER DISEASE

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Background: The spectrum of metabolic associated fatty liver disease (MAFLD) includes steatosis, steatohepatitis, that can progress to cirrhosis, hepatocellular carcinoma, and to advanced stage of liver disease. MAFLD associated with hepatic insufficiency has been frequent in the clinical practice.

Aim: To evaluate the frequency and characteristics of MAFLD patients with advanced liver disease.

Methodology: The case-series included MAFLD patients with advanced disease come from at a hepatology clinic. MAFLD criteria: past or present evidence of metabolic risk factors; presence or previous documentation of steatosis by imaging or histological analysis. Child-Pugh and MELD scores were used to estimate MAFLD prognosis. Portal hypertension (PH) was defined by the presence of gastroesophageal varices on endoscopy. The data were collected and analyzed using the statistic program SPSS.

Results: A total of 263 patients with MAFLD were included; 48 (18.25%) presented advanced liver disease. Most were female (79.2%); 68.8% were of African descent; median age was 69 years (IQR 59.75-76.50). Features of metabolic syndrome was frequent in these patients (60.4% presented arterial hypertension; 47.9% diabetes, and 1% dyslipidemia). Child-Pugh (C-P) A was observed in 24.4% of the cases; C-P B in 63.4%; C-P C in 12.2%. MELD scores ranged from 7-24 (mean 13.34). PH was observed in 60.41% of the cases.

Conclusions: In a large series of patients with MAFLD, the frequency of hepatic insufficiency and portal hypertension were significant. Relevant finding in this simple also was the predominance of African descent individuals, who usually have a better MAFLD prognosis. This result needs to be better evaluated.

<https://doi.org/10.1016/j.aohep.2021.100509>

O-23 METABOLIC ASSOCIATED FATTY LIVER DISEASE CLINICAL PROFILE IN LEAN PATIENTS: CAN IT BE DIFFERENT?

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Background: The prevalence of metabolic associated fatty liver disease (MAFLD) in lean patients has grown around the world and the better understanding of this liver disease, in these individuals, has become of interest.

Aim: To evaluate the profile of MAFLD in lean compared to obese patients.

Methodology: This case-series included patients with MAFLD from a hepatology clinic. MAFLD criteria included the presence of steatosis (ultrasound) and one of the following clinical conditions: overweight/obesity, type 2 diabetes mellitus (T2DM), other features of metabolic dysfunction. Fibrosis was evaluated by FIB4 and APRI scores. Two groups were considered: G1-lean adult (BMI \leq 24.99 kg/m²); elderly (\geq 60 years- BMI $<$ 28 kg/m²) patients; G2-obese adult (BMI \geq 25kg/m²); elderly (BMI $>$ 28kg/m²). SPSS software was used for data analysis.

Results: A total of 135 MAFLD patients were included: 57(42.2%) in G1; 78(57.8%) in G2. G1 characteristics: Mean age was 62.26 years (SD=11.71); 70.2% were women; 75% were of African descent (self-declared); BMI mean: 24.59kg/m²(SD=2.37). Hypertriglyceridemia (HYT) was observed in 42.9% patients, T2DM in 47.4%, arterial hypertension (AH) in 54.4%, and low HDL 41.2%. Fibrosis not observed by FIB4 or APRI in all lean cases. G2 characteristics: Mean age was 55.62 years (SD=10.4); 80.8% were women; BMI mean: 32.05kg/m² (SD=3.78); 91% were of African descent. AH was found in 57.7% of the cases; T2DM in 46.2%; HYT in 36.1% and low HDL in 38.6%. Fibrosis was not observed by FIB4 and 5.5% presented fibrosis by APRI.

Conclusions: In these lean patients, MAFLD was frequent in elderly women; hypertriglyceridemia, T2DM were relevant risk factor; and they did not present fibrosis by noninvasive scores. The prevalence of Afro descent in this MAFLD population was elevated and the ethnicity influence in these cases needs to be better understood in future studies.

<https://doi.org/10.1016/j.aohep.2021.100510>

O-24 A CASE SERIES OF CHLOROQUINE MONOTHERAPY TO INDUCE BIOCHEMICAL REMISSION IN PATIENTS WITH RELAPSED AUTOIMMUNE HEPATITIS

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Background and Aims: Azathioprine (AZA) and prednisone (PD) are the standard treatment (ST) for the onset or relapse of autoimmune hepatitis (AIH). Antimalarials were effective for maintenance of remission of AIH after immunosuppression withdrawal. Our aim is to describe a series of patients who relapsed after antimalarial withdrawal and achieved biochemical remission after reintroduction of chloroquine in monotherapy.

Methods: Eight patients received chloroquine diphosphate (DCQ) 250mg/d or hydroxichloroquine (HCQ) 400-800mg/d in monotherapy for biochemical remission after relapse. The schedule is described below: histological remission (HR) with ST \rightarrow ST withdrawal and antimalarial use for maintenance of remission for 1-3y \rightarrow antimalarial withdrawal \rightarrow relapse of AIH \rightarrow reintroduction of antimalarial instead of ST, at the request of patient due to corticosteroids side effects (SE). Four out of 8 patients underwent liver biopsy to evaluate HR after at least 18mo of biochemical remission.

Results: Mean age at diagnosis of AIH of 36.2 \pm 20.4y; 6 type-1, 2 anti-SLA/LP (5 reactive in 7 tested); 3 with cirrhosis at diagnosis. Mean doses of AZA/PD at HR were, respectively, 84.4 \pm 18.6 and 9.4 \pm 2.2mg/d. Mean interval between antimalarial withdrawal and relapse was 20.5 \pm 34.1mo. Mean ALT at relapse was 139.7 \pm 54.2 U/L. Three patients received DCQ and 5 HCQ. During HCQ intake 1 patient needed further adjustment of drug to 800 mg/d for 15 days due to worsening of ALT from 130 to 246 U/L, with subsequent reduction to the initial dose. Seven patients achieved biochemical remission, in a mean time of 14.2 \pm 19.9mo; 3/4 had histological remission. The drug was well tolerated and there were no serious SE.

Conclusions: CQ monotherapy was safe and effective for induction of remission in this subgroup of AIH. This finding raises arguments to include this drug as an option for treatment of AIH, not necessarily in monotherapy.

<https://doi.org/10.1016/j.aohep.2021.100511>