CT. Both gelatinases, MMP-2 y MMP-9, were found elevated in CHC (mild and severe fibrosis) vs. CT; and decreased in OH, CiOH, HGNA (mild and severe fibrosis) vs. CT, plus there are significant differences between all etiologies, p<0.001.

Discussion: In patients with CHC, MMP-2 y -9 serum concentration increases, particularly in severe fibrosis stages, although it has no effect on ECM (extracellular matrix) degradation, as they are inactive. Nevertheless, there is a significant decrease in these gelatinases in ALD and NAFLD.

Conclusions: MMP-2 y MMP-9 module depends on the etiological agent involved, which can be useful for the differential diagnosis of liver diseases.

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MMP-7 is a non-invasive biomarker of chronic liver diseases

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Introduction and Objectives: This study aimed to evaluate serum concentrations of MMP-7 in different liver etiologies and according to fibrosis stage.

Materials and methods: A cross-sectional and multicenter study was carried out, including subjects with alcoholism (WHO criteria), without (OH) and with liver injury (cirrhosis, CiOH); diagnosed by clinical, biochemical data, non-alcoholic fatty liver (NAFLD) and chronic Hepatitis C (CHC). Transitional elastography (Fibroscan) was performed in NAFLD and CHC, considering mild fibrosis (MF: F0, F1, F2) and advanced fibrosis (AF: F3, F4). As controls, subjects without alcohol consumption (CT) were recruited. For the quantification of MMP-7, Multiplex-MERCK© was used. Statistical analysis was performed using SPSS V.22 using Mann Whitney U, p<0.05.

Results: It was included 99 subjects (OH); 45 (CiOH); 48 (CHC, FL); 54 (CHC, FA); 27 (NAFLD, FL); 36 (NAFLD, AF) and 131 CT. MMP-7 was found to be elevated in CHC (FL and FA), vs. CT; and decreased in OH, CiOH, NAFLD (FL and FA) vs. CT, plus there are significant differences between all etiologies, p<0.001.

Discussion: MMP-7 is a matrilysin that degrades extracellular matrix products (proteoglycans); it increases significantly in subjects with CHC compared to CT, while in other pathologies with stages, even in advanced fibrosis, the levels are decreased compared to CT.

Conclusion: The increased MMP-7 in serum of chronic Hepatitis C and decreased in alcoholism and non-alcoholic fatty liver patients suggests that, according to the etiology, the levels can be useful to make a differential diagnosis. It is a potential non-invasive biomarker.

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Autoimmune hepatitis with superimposition of primary sclerosing cholangitis on non-specific chronic ulcerative colitis

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Introduction and Objetives: Chronic nonspecific ulcerative colitis can be associated with autoimmune hepatitis (AH) and Primary Sclerosing Cholangitis (PSC) as an overlap (prevalence 1.7-12.6%). The evolution depends on the clinical picture, biochemical pattern, and histological determination. The response to immunosuppressive treatment of inflammatory bowel disease and autoimmune hepatitis is good, but not Primary Sclerosing Cholangitis.

Case Summary: 26-year-old female resident of Mexico City. Family history of arterial hypertension and acute myocardial infarction. Smoking suspended, alcoholism denied, other history denied. Current condition: Begins 2013 with diarrhea with scant blood, bloating, abdominal pain, general malaise, weight loss; microcytic hypochromic anemia, thrombocytosis, hypertransaminasemia. Colonoscopy: Pancolitis Mayo 2. Biopsy: chronic ulcerative colitis, intense cryptitis, cryptic abscesses. Hypertransaminasemia and cholestatic syndrome persisted; viral hepatitis was ruled out, positive ANAP 1:80, and negative anti-smooth muscle. Liver biopsy: lymphoplasmacytic infiltrate without cholangiole damage or cholestasis, suggestive of HA. He received steroid, azathioprine, ursodeoxycholic acid and mesalazine. Treated latent TB. He received infliximab with improvement. August 2020: jaundice and direct hyperbilirubinemia. Cholangioresonance 2021: Primary Sclerosing Cholangitis. Liver biopsy 2021: lymphocytic infiltrate beyond the limiting plate, plasma cells, cholangiole proliferation, peripheral sclerosis, intracytoplasmic and canalicular cholestasis, focal necrosis, bridges of fibrosis (F3) compatible with AH and PBC. Biological therapy was suspended, Azathioprine was adjusted, and sent for transplant by MELD of 20. Fig. 1 y Fig. 2.

Results:

Conclusions: The patient debuted with moderate Montreal E3S2 UC and confirmed HA. She presented biochemical and clinical remission and intermittent cholestasis; She presented jaundice six years later, confirming PSC by MRI and Biopsy. The evolution of this overlap depends on age, gender and initial phenotype, regardless of the course of UC, representing a diagnostic and therapeutic challenge

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