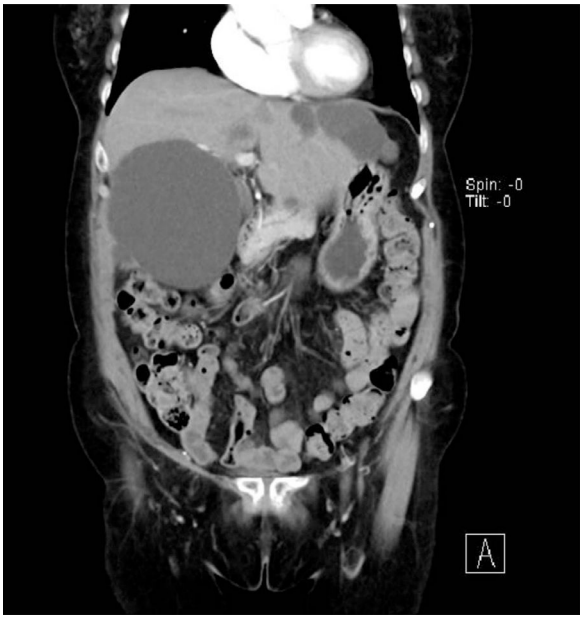
Results: 75 patients with hepatic cysts were included, 35 men (46.6%), age 48‡ 10.3 years, 40 Women (53%) age 45+ 8 years; the average number of cysts per patient treated was 1.3, with a mean cyst size of 13+ 3 cm, By location were similar in any of the two lobes, the most frequent presenting symptom was pain in 70 patients (93.3%), the rest were due to risk of rupture to the abdominal cavity and/or thorax; 90 cysts were drained and sclerosed guided by ultrasound, using absolute alcohol in a volume of 20% in relation to the size of the cyst, in 100% a decrease of the cyst was observed until remission, no complications were reported, The average follow-up was 15 months and there was recurrence in only 3 cases (4%).

Conclusions: The drainage and excision of simple hepatic cysts in expert hands represents an effective and safe alternative in the treatment of symptomatic cysts or with a risk of rupture; recurrence in this treatment is infrequent. No complications were observed.

Ethical Statement: This study was conducted in accordance with the ethical principles of our hospital center. All data were handled with strict confidentiality and for research purposes only.

Declaration of interests: None.

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.



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TSH and its Correlation in the Development of Fibrosis in Patients with Hypothyroidism in a Tertiary Care Hospital

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Introduction and Objectives: Fatty liver disease and hypothyroidism are two prevalent conditions in Mexico that pose significant public health challenges. The increase in their incidence over the past decades is due to changes in lifestyle, diet, and access to healthcare. Although hypothyroidism does not directly cause hepatic fibrosis, it is related to the body's metabolic function. Hypothyroidism can slow down metabolism and lead to lipid accumulation in the liver, a condition known as hepatic steatosis or fatty liver. This condition can progress to steatohepatitis and eventually to hepatic fibrosis, characterized by scar tissue formation. If left untreated, fibrosis can advance to liver cirrhosis with severe complications. Hypothyroidism and fatty liver disease share common risk factors such as obesity, type 2 diabetes, and metabolic disorders. Proper treatment of hypothyroidism and early identification of fatty liver are crucial to prevent progression to fibrosis. It is essential for individuals with hypothyroidism to monitor their liver health and adopt a healthy lifestyle to avoid liver complications. Currently, there are enough studies that validate the association between hypothyroidism and the development of fatty liver with varying degrees of hepatic fibrosis. To identify clinical-demographic characteristics in patients with hypothyroidism evaluated in the endocrinology service and to identify the presence of fibrosis through non-invasive evaluation.

Materials and Patients: Patients with hypothyroidism aged 18 to 80 years of both sexes evaluated by the Endocrinology service with a complete medical record, who do not have risk of alcohol consumption, hepatotropic virus infections, or use of drugs causing hepatotoxicity.

Results: A review of 85 patients with complete medical records was carried out, and a correlation analysis with numerical variables in the SPSS system found that TSH levels do not correlate with the development of hepatic fibrosis, with a Pearson's $r = -0.074$ ($p = 0.519$), which is not significant.

Conclusions: In this case series, we report that there is no direct correlation between TSH levels and the development of hepatic fibrosis. However, it is important to highlight that metabolic comorbidities favor the development of fatty liver and, consequently, the possibility of developing hepatic fibrosis. In this series of cases, four cases of advanced fibrosis were found, so it is important to emphasize requesting complete studies in patients with hypothyroidism and to complement with imaging studies.

Ethics Statement: This is a risk-free study in which no intentional interventions nor modifications of the physiological, psychological, and social variables of the individuals participating in the study were performed.

Declaration of Interests: None.

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

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Acute Hepatitis A-Induced Autoimmune Hepatitis: A Case Report

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Introduction and objectives: Autoimmune hepatitis is a chronic immune-mediated inflammatory disease characterized by hypergammaglobulinemia, the presence of autoantibodies and histologically lymphoplasmacytic portal inflammation (interphase hepatitis).[1,2] Its pathogenesis is unknown and it occurs in 3% of cases associated with hepatitis A virus infection.[3,4]

Materials and Patients: 29-year-old woman with no relevant medical history. She presented with acute hepatitis due to virus A in

February 2024 (symptomatic clinical pattern with jaundice), corroborated by serology (IgM + antibodies) remission with supportive treatment in April. In May, she presented a new jaundice event, general malaise, and asthenia, so she underwent evaluation. On examination, jaundice and hepatomegaly were observed. Laboratory studies: BT 23.5mg/dL, BD 19.9mg/dL, ALT 534U/L, AST 827U/L, FA 128U/L; suggestive of acute hepatocellular injury (R factor=14). Hepatobiliary ultrasound rules out the obstruction and reports a diffuse increase in echogenicity; Serology: IgM HAV non-reactive, IgG HAV+ (ruling out a prolonged course of hepatitis A). Consumption of drugs, alcohol, and infections by other viruses (B, C, E, Epstein Barr, and HIV) are ruled out. The measurement of serum globulins reports hypergammaglobulinemia (IgG x 1.6 ULN); in addition, increased levels of antinuclear antibodies (ANAs, dilution 1:80) are detected by Immunofluorescence (IF); histopathological report of liver biopsy: chronic interface hepatitis, portal lymphoplasmacytic infiltrate and lobular damage, (fibrosis: F2); meeting all suggested criteria for the diagnosis of autoimmune hepatitis defined by the International Autoimmune Hepatitis Group Criteria.[5] Due to all of the above, it was decided to start treatment with parenteral steroid at a dose of 1 mg/kg/day (prednisone equivalent), with a good response to medical management, observing a gradual reduction in the levels of bilirubin, liver enzymes, and immunoglobulin G levels after the first 72 hours of medical treatment.

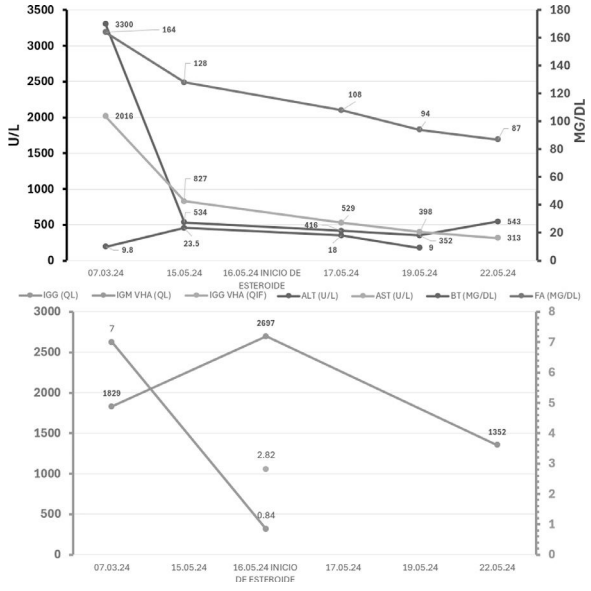
Results: Several triggers have been identified for the appearance of outbreaks of autoimmune hepatitis among them viral infections, which are reported in the literature. In rare cases, the hepatitis A virus can precede an outbreak of autoimmune hepatitis type 1. The treatment to induce immune remission is based on the administration of steroids, which usually guarantees a good response, as in the reported clinical case.

Conclusions: Hepatitis A virus infection is associated with altered immunotolerance with subsequent activation of lymphocytes and the production of specific antibodies that trigger molecular mimicry, which is directly involved in the pathophysiology of autoimmune hepatitis. Exposure to external factors is considered necessary to trigger the autoimmune reaction against hepatic structures.

Ethics statement: The procedures indicated above are under the General Health Law on Research in Human Beings regulations, as as well as the Declaration of Helsinki and subsequent amendments.

Conflict of interest statement: None.

Financing: Regional Hospital Lic. Adolfo López Mateos ISSSTE.



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Cholestatic Injury Induced by Naproxen: A Rare Cause of NSAID-Induced Hepatotoxicity. Case Report.

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Introduction and Objectives: Naproxen-induced liver injury is very rare (1-3 cases per 100,000 exposed individuals), typically occurring 1-6 weeks after ingestion[1]. The damage can manifest with or without immunoallergic features and varying degrees of hepatocellular injury and cholestasis[2]. We illustrate a case of a patient who developed cholestatic injury.

Materials and Patients: A 51-year-old man, with the only relevant history being self-prescribed ingestion of 220 mg gel capsules of naproxen sodium two weeks prior for post-exercise muscle pain, presented on 04/05/24 with asthenia, vague abdominal pain in the right hypochondrium, jaundice, acholia, and dark urine. He sought medical emergency services two days later, where pronounced mucocutaneous jaundice, hepatomegaly, and hepatodynia were observed. Paraclinical tests showed hyperbilirubinemia: total bilirubin (TB) elevated due to direct bilirubin (DB), marked elevation of alkaline phosphatase (AP) and gamma-glutamyl transferase (GGT), and slight elevation of aspartate aminotransferase (AST) and alanine aminotransferase (ALT) (R factor = 2 mixed). Hepatic and biliary tract ultrasound reported diffuse increased hepatic echogenicity, ruling out biliary tract obstruction. Serological testing for hepatotropic viruses and TORCH screen were negative, as was the serological profile for autoantibodies. A percutaneous ultrasound-guided liver biopsy was performed; biopsies demonstrated intrahepatic cholestasis, minimal and focal lobular and portal interface hepatitis, macrovesicular steatosis; special stains negative for hemosiderin and glycogen deposits, fibrosis and copper-bound proteins; suggested of drug induced liver damage **(Figure 1)**.

Results: Drug-induced liver injury is a diagnosis of exclusion, only to be suspected when major causes of liver damage have been ruled out. Naproxen, a non-steroidal anti-inflammatory drug (NSAID) derived from propionic acid, has been reported to cause hepatotoxicity phenotypes of hepatocellular injury (acute hepatitis) and cholestasis through metabolic, immunoallergic and idiosyncratic mechanisms[3]. In this patient, the toxicity was non-dose-dependent, with an acute presentation characterized by a predominant elevation of cholestatic markers. Only supportive measures were provided, with close clinical and biochemical monitoring to identify early signs of liver dysfunction. The patient showed favorable evolution towards remission, characterized by symptomatic improvement and a progressive decrease in cholestatic markers within the first few days **(Figure 2)**. After nine days of hospitalization, discharge was decided due to improvement, with a follow-up appointment for continued monitoring.

Conclusions: In suspected naproxen-induced cholestatic injury, a liver biopsy is not required for diagnosis[4] but is useful for understanding etiology, severity, extent, and prognosis. Discontinuing the causative agent is the first measure, and medical treatment should be directed solely by the clinical and biochemical evolution of the patient.

Ethical statement: The procedures performed comply with the regulations of the General Health Law on Research in Humans and the Declaration of Helsinki of 1975 and subsequent amendments. According to Article 5 of the General Health Law, this research