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Acute Hepatitis in Pregnancy – Diagnostic Challenges

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A 35-year-old nulliparous woman, 22 weeks of gestation, with no relevant personal or family history, on Doxylamine Succinate+ Dicyclomine Hydrochloride + Pyridoxine Hydrochloride and Chlorpromazine, was admitted in the Obstetric Department for hyperemesis gravidarum. Physical examination was unremarkable. After stopping her previous medication, she was started on ondansetron. Blood analysis revealed INR 1.0; AST 525U/L; ALT 952U/L, GGT 55U/L, FA 72U/L, total bilirubin (TB) 2.05mg/dL, with direct of 1.37mg/dL. Abdominal ultrasound only revealed vesicular microlithiasis. Choledocholithiasis or hyperemesis gravidarum were the preliminary diagnostic hypotheses. She denied ethanolic or toxicophilic habits, consumption of herbal products or recent travels. Initial etiological research showed: antiHCV-, HBsAg-, AcHBc-, AcHBs-, antiHAV IgM-, antiEBV IgM-, antiCMV IgM-, AntiHEV-, antiHSVI/II IgG- and IgM+, ANA-, IgG, alpha 1 antithrypsin, ceruloplasmin, ferritin, thyroid function and lipid profile within the normal range. Kayser-Fleischer rings were excluded.

During hospitalization there was a decrease in TB and maintenance of the remaining values of liver tests, as well as hyperemesis. She underwent percutaneous liver biopsy which revealed subacute active hepatitis, a mild, multifocal, intralobular lymphocyte infiltration and punctually degenerative hepatocytes; without interface hepatitis, emperipolesis, viral cytopathic effect, hemosiderin pigment or fibrosis. (Figure 1 & 2). The ongoing study finally disclosed: negative liver autoimmunity and serum HSVI/II PCR; and normal bile acids and urinary copper.

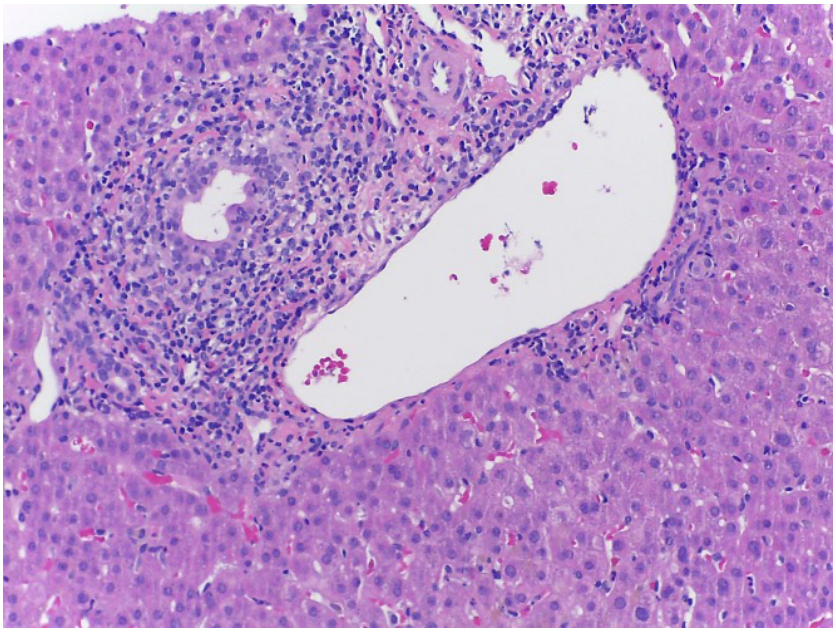

Figure 1: HE, 200x: Hepatic Parenchyma with Space Door Expansion, With Mild, Intralobular Lymphocyte Infiltration, With Rare Plasmocytes, Neutrophils and Eosinophils.

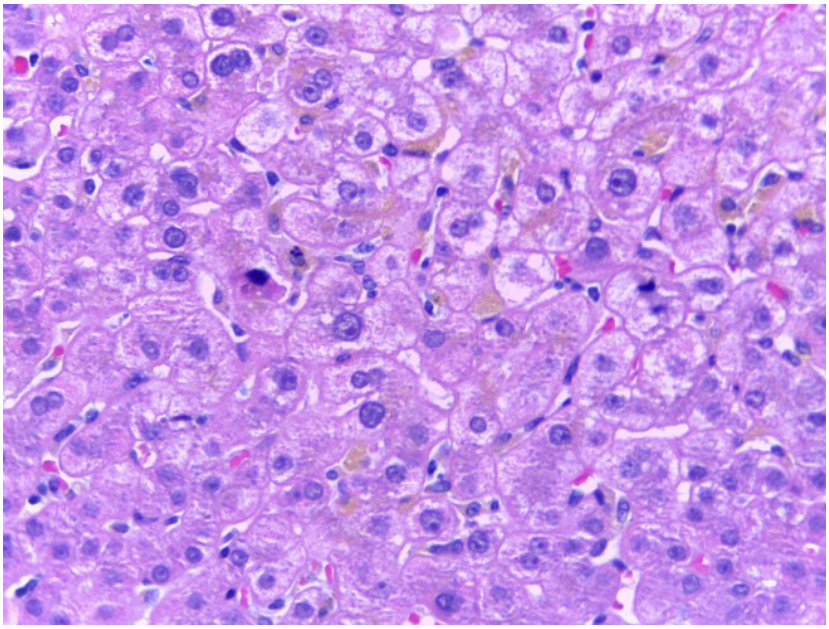

Figure 2: HE, 400x: Hepatic Parenchyma with Punctually Degenerative Hepatocytes.

The most probable hypothesis is drug induced liver injury due to chlorpromazine. There was a favorable evolution, with normalization of liver enzymes in 2 months.

The incidence of abnormal liver tests in pregnant women is ~3–5%. Clinical evaluation relies on the accurate determination of intrinsic liver disease or liver disease related specifically to pregnancy. The later can be classified into those of early pregnancy (hyperemesis gravidarum) and those of late pregnancy, such as pre-eclampsia with hepatic impairment, low platelets (HELLP) syndrome and intrahepatic cholestasis of pregnancy.

The need of liver biopsy for diagnosis of disease in pregnancy is uncommon as most etiologies can be determined by biochemical, serological, and clinical parameters. However, if required, percutaneous liver biopsy can be performed safely. In our patient liver biopsy was necessary as drug induced liver injury is a diagnosis of exclusion.

Chlorpromazine can cause mild and transient serum enzyme elevations and is also a well-known cause of clinically apparent acute and chronic cholestatic liver injury. Liver test abnormalities have been reported to occur in up to 40% of patients on long term therapy, but elevations are uncommonly above 3 times the upper limit of normal. The pattern of serum enzyme elevations is typically cholestatic or mixed. Aminotransferase elevations do not require dose modification/ discontinuation of therapy. The acute cholestatic hepatitis is typically self-limited and benign, but should prompt immediate discontinuation [1-3].

We pretend to illustrate the differential diagnosis of acute hepatitis in pregnant women, with this rare and challenging case.

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