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OVERLAP SYNDROME AS A CAUSE OF SEVERE HEPATITIS IN A FEMALE PATIENT WITH SYSTEMIC LUPUS ERYTHROMATOSIS-CASE STUDY

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Case study: A 30 years old female patient known to have systemic lupus was admitted to tropical medicine department because of jaundice and hepatic encephalopathy associated with low grade fever, vomiting and right hypochondrial pain. Upon examination patient had grade I to II hepatic encephalopathy, yellow sclera, tender hepatomegaly. Laboratory tests showed: a white cell count of 13,000 cells/µl, a platelet count 90,000 cells/µl, serum albumin 1.5g/dl, CRP 20 ng/ml, Alanine aminotransferase 1400 ul, Aspartate aminotransferase 1100 ul, a total bilirubin of 15 mg/dl, direct bilirubin of 11 mg/dl and prothrombin activity of 25%. Viral hepatitis markers were negative except for IgG for hepatitis A, Autoimmune markers were as follow; ANA (antinuclear antibody) 1/220, ASMA (anti-smooth muscle antibody) negative, LKMA (liver kidney microsomal antibody) negative, SLA (soluble liver antigen) negative, AMA (antimitochondrial antibody) negative, but a positive gamma globulin IgG and a negative IgM. Imaging by computerized tomography with intravenous contrast revealed acute hepatitis with patches of necrosis. Our patient scored six according to simplified diagnostic criteria of the International Autoimmune Hepatitis Group table, two provisional supportive treatment were initiated upon admission in the form of fresh frozen plasma infusions with broad spectrum antibiotics together with anti-encephalopathy measures and lactulose enemas. Also, steroid therapy in the form of oral prednisolone in a dose of 60 mg/day was administered. Fortunately, gradual improvement of laboratory analysis occurred till prothrombin activity reached 85% where we performed a liver biopsy that showed the following histologic features with acute hepatitis pattern of injury, with portal and periportal lymphoplasmacytic infiltrate and interface hepatitis. Plasma cells were prominent, the severity of necro-inflammatory activity ranged from bridging necrosis to massive hepatic necrosis. Hepatocyte regeneration was prominent, with regenerating rosette-like structures with florid bile duct destruction.

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