LETTERS TO EDITOR

PELIOSIS HEPATIS PRESENTING AS HEMOPERITONEUM

Peliosis hepatis is an uncommon vascular disorder that may be caused by several etiologic agents and different pathogenetic mechanisms. It is characterized by cystic blood-filled spaces [1 mm to several centimeters], resulting from focal rupture of sinusoidal walls, with a preferential location within the liver; peliosis may be seen in spleen, lymph nodes, lungs, kidneys, adrenals, bone marrow, and gastrointestinal tract. Peliosis varies from minimal, asymptomatic lesions to larger massive lesions that may present with cholestasis, liver failure, portal hypertension, a vascular mass lesion, or spontaneous rupture. Intra-abdominal hemorrhage in peliosis hepatis is a rare complication,[1] which can be life threatening.

In the present study, a 70-year-old man presented with complaints of pain in the abdomen since 1 month and gradually progressing abdominal distension since 15 days. The pain was epigastric, nonradiating, dull, continuous and not associated with food intake, vomiting or diurnal variation. There was no history of weight loss or decreased appetite. On clinical examination, the patient was of thin build, with epigastric fullness, abdominal distension and moderate pallor. The liver was moderately enlarged, nontender, firm, with sharp borders. There was shifting dullness, and bowel sounds were heard. The investigations showed hemoglobin, 6.6 g/dL; total leukocyte count, 8800/cu. mm; hematocrit, 21%; normal bleeding and clotting time; altered prothrombin time, INR1.5; normal blood urea, creatinine, electrolytes, uric acid, and random sugar; lactic dehydrogenase, 1011/dL; decreased albumin of 2.9 g/dL; normal globulin and total protein; AST, 68 U/L; ALT, 66 U/L; and alkaline phosphatase, 397 U/L. The ascitic fluid analysis showed protein, 4.3 g/dL; glucose, 81 mg/dL; chlorides, 105 mEq/L; normal alpha fetoprotein and carcinoembryonic antigen levels; and numerous polymorphs on Gram's stain; there was no growth on culture. Ultrasonography revealed hepatosplenomegaly with heterogeneous echotexture and multiple small hypoechoic lesions in the liver and spleen. With a clinical diagnosis of non-Hodgkin's lymphoma or metastasis, laparotomy was done; there was hemorrhagic ascites and multiple spotty black lesions all over the liver. The wedge biopsy showed multiple, variably sized, cystic, blood-filled spaces without endothelial lining [Figure 1].

Cystic blood-filled spaces in the liver can be macroscopic or microscopic, and diffuse or focal. Macroscopic and randomly distributed lesions without an endothelial lining are seen

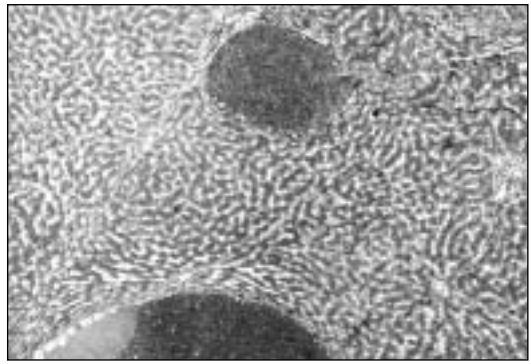

Figure 1: Blood-filled cystic spaces without endothelial lining — reticulin silver stain, ×400

in peliosis hepatis. Microscopic peliosis is often confused with extreme sinusoidal dilatation and hepatocellular dropout. In sinusoidal dilatation, the liver plate structure is intact, and lesions are periportal or midzonal, especially when associated with oral contraceptives and pregnancy. In hepatocellular dropout, there is collapse of liver cell plates without loss of reticulin fibers.

It is appropriate to classify the lesions according to the apparent etiology, as this correlates with distinctive histological and clinical features. In addition to anabolic, estrogenic, or adrenocortical steroids, macroscopic peliosis hepatis has been reported in malnutrition,[2] leukemia, tuberculosis, leprosy, vasculitis, lymphoma,[3] and AIDS. Bacillary peliosis in human immunodeficiency virus infection can manifest as massive hemoperitoneum.[4] In the present study, the patient gave a history of taking NSAIDS for vague abdominal pain, for almost a year; and the probability score by Naranjo algorithm[5] for NSAIDS use and hemoperitoneum calculates to 0 [doubtful adverse drug reaction].

Spontaneous regression can occur in peliosis hepatis with interruption of the inducing factor, or withdrawal of the specific drug in case of drug-induced peliosis. Recurrent spontaneous intrahepatic hemorrhage from peliosis has also been reported[6] and is rarely fatal; it is due to extensive parenchymal destruction. Hemorrhage complicating localized peliosis hepatis may require a partial hepatectomy. In the present study, the prolonged consumption of NSAIDS might have been responsible for progressive dilatation, rupture of subcapsular cysts, and the resultant hemoperitoneum. The patient was given only palliative treatment and advised sonography during the next visit.

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