CASE REPORT

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Subcapsular Hematoma in Fatty Liver of Pregnancy

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ABSTRACT: A case of fatty liver of pregnancy with subcapsular hematoma of the liver caused by intravenous heparin treatment is reported. The heparin was given for the thrombophlebitis of leg veins. The patient expired suddenly as a result of rupture of the subcapsular hematoma of the liver causing massive intraperitoneal hemorrhage. The pathophysiology and complications of the fatty liver of pregnancy are discussed.

KEYWORDS: pathology and biology, hematoma, liver, pregnancy

The fatty liver of pregnancy is an uncommon clinical entity that results in frequent maternal or fetal death or both. This condition was first described by Sheehan in 1940 [*1*]. There have been 60 documented cases reported with characteristic clinical, laboratory, and anatomic features although it has been called different names, such as acute yellow atrophy of pregnancy, idiopathic jaundice of pregnancy, fatty degeneration (or metamorphosis) of liver in pregnancy, and idiopathic fatty liver of pregnancy. Its etiology and pathogenesis remain unknown. A case of fatty liver of pregnancy with unusual complications was observed at the Westchester County Medical Examiner's Office, New York.

Case Report

A 28-year-old West Indian female, gravida 5, para 2, at 37 weeks of gestation, was admitted to the hospital because of pain and tenderness of right leg and bilateral superficial varicosities of the legs. She was a practicing Jehovah's Witness and denied the use of alcohol or smoking. Vital signs on admission showed blood pressure 118/78, pulse 90/min, temperature 100.4°F (38°C) and fetal heart rate 138/min. Laboratory findings on admission showed hemoglobin 14.9%, hematocrit 42.8%, white blood cell count 18.800/mL with 51% polymorphonuclear leukocytes and 4% monocytes. Partial thromboplastin time 29.6 s/27.2 s and prothrombin time 12.6 s/10.7 s. Urinalysis showed many bacteria, 1+ protein, and trace amounts of blood. Culture and sensitivity on second hospital day showed 100 000 colonies of *Escherichia coli* which was sensitive to ampicillin. Serum chemistry showed total bilirubin 0.6 mg/dL, alkaline phosphatase 170 U, lactic dehydrogenase 165 U, and serum

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glutamic-oxaloacetic transaminase (SGOT) 15 U. Diagnoses of thrombophlebitis of the legs and pyelonephritis were made and she was treated with 24 000 U of heparin intravenously over 24 h and 2 g of ampicillin intravenously every 6 h. On the fourth hospital day, dosage of heparin was raised to 36 000 U per 24 h intravenously and partial thromboplastin time was maintained at 42.8 s/28.9 s. During ten hospital days the symptoms and signs of urinary tract infection and phlebitis were much improved. On the tenth hospital day, she suddenly started complaining of sharp right thoracoabdominal pain radiating to the right upper back and arm with shortness of breath.

An electrocardiogram (EKG) showed regular sinus rhythm. She became tachypneic (36/min). Vital signs showed blood pressure 110/70, pulse 90/min. Complete blood count showed hemoglobin 14.2 g/dL; hematocrit 42.9 g/dL; and white blood cell 10 000/mL with polymorphonuclear leukocyte 62%, bands 2%, and platelet 336 000/mL. Diagnosis of pulmonary embolism was suspected and the dosage of heparin was raised to 50 000 U per 24 h. On the eleventh hospital day she became pale, clammy, lethargic, diaphoretic, and still complained of right-sided thoracoabdominal pain, dyspnea, and vomiting. Vital signs showed blood pressure 110/80, temperature 97°F (36.1°C), pulse 105/min, and respiration 22/min. Two and a half hours later she delivered a stillborn male infant which was fully developed with crown heel length of 51 cm and body weight 3450 g. Her general condition became grave following delivery as a result of worsening right-sided pain, increasing dyspnea, diaphoresis, and lethargy. Laboratory data showed total bilirubin 0.5 mg/dL, alkaline phosphatase 240 U, lactic dehydrogenase 275 U, and SGOT 55 U. She expired 2 h after the delivery.

The autopsy revealed a moderately well-developed and well-nourished female, measuring 172.7 cm and weighing 72.6 kg. Externally, the skin showed no evidence of jaundice. The abdominal cavity contained 3700 mL of partially clotted blood. The liver weighed 1450 g. The capsule of the anterior aspect of the right lobe of the liver was separated from parenchyma forming a subcapsular hematoma measuring 20 cm in greatest dimension (Fig. 1). A rupture of the hematoma was present in inferior aspect of liver, and a small amount of residual blood was seen in the subcapsular space. Intact liver showed diffuse light yellowish-tan

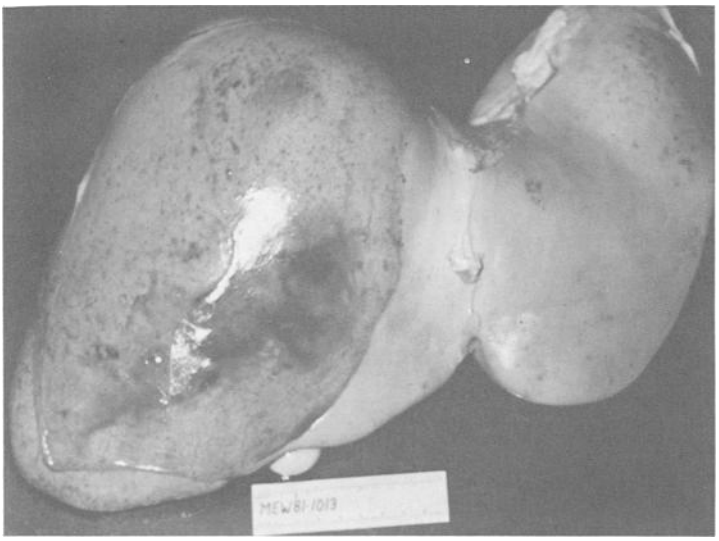

FIG. 1—*The capsule of the anterior aspect of the right lobe of the liver was separated from parenchyma forming a subcapsular hematoma measuring 20 cm in greatest dimension.*

discoloration with scattered fine punctuate petechiae-like lesions. Sections revealed diffuse light yellowish-tan parenchyma. The extrahepatic biliary system was patent. The heart weighed 300 g. Epicardium, myocardium, and endocardium were normal. Coronary arteries were patent and showed no atheromatous changes. Combined weight of the lungs was 450 g. Both lungs were well expanded and sections were normal. No evidence of pulmonary thromboemboli was seen. The pancreas was in its usual location and no evidence of pancreatic fat necrosis or parenchymal hemorrhage was seen.

Together, both kidneys weighed 440 g. Sections showed pale edematous parenchyma. The calices, pelves, and ureters were patent. No evidence of purulent discharge or hemorrhage was seen. The uterus and vagina showed usual postpartum dilatation and residual hemorrhage. No evidence of uterine perforation was seen. The brain weighed 1350 g. Sulci and gyri were well preserved and sections showed no abnormalities.

Microscopically, the hepatic architecture was intact. The hepatocytes were distended and contained fine round vacuoles in cytoplasm which did not displace the nuclei (Fig. 2). This change was prominent in centrolobular areas. Oil red O stain for fat was positive. No massive necrosis of hepatocyte was seen. There were focal minute areas of necrosis with neutrophilic infiltration in superficial areas under the subcapsular hematoma. The sections taken from the periphery of subcapsular hematoma revealed fibrin deposit mixed with erythrocytes and occasional neutrophils (Fig. 3). No evidence of bile stasis was seen. Portal areas were normal. No other organs showed similar fatty change including proximal tubular cells of the kidneys and brain. The pancreas was normal histologically.

Discussion

The fatty liver of pregnancy was first classified as a distinct clinical entity when Sheehan reported six cases with jaundice in the third trimester [*1*]. Microscopically, the liver showed intracytoplasmic fat vacuoles with no hepatocellular necrosis and little or no inflammatory reaction.

The etiology of this disease is unknown. Kunelis reviewed 54 reported cases and found 16 cases were treated with intravenous tetracycline for pyelonephritis [*2*]. He stressed the signif-

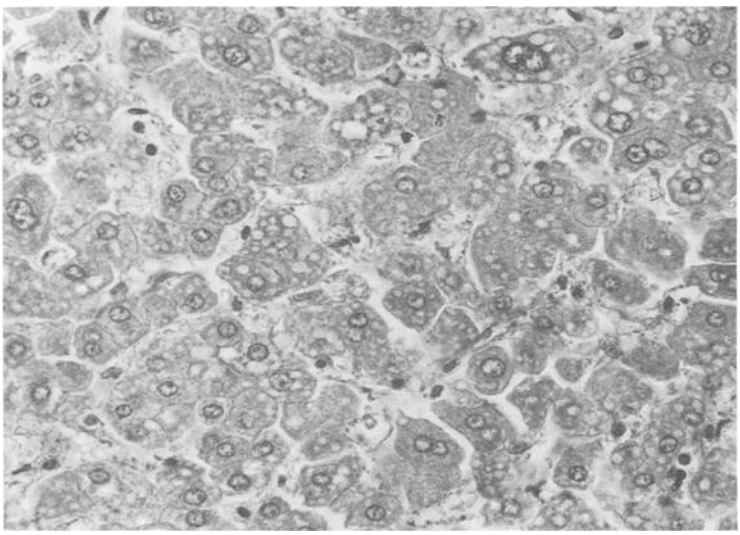

FIG. 2—*Distended hepatocytes that contained fine round vacuoles in cytoplasm which did not displace the nuclei.*

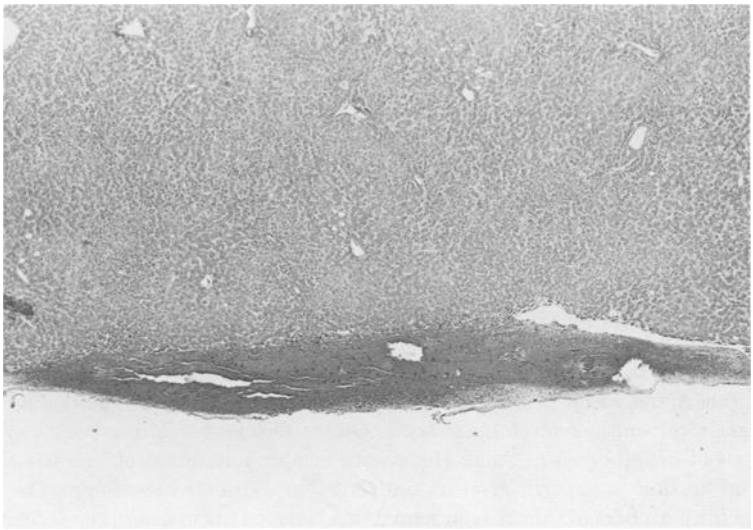

FIG. 3—*Fibrin deposit mixed with erythrocytes and occasional neutrophils in sections taken from the periphery of subcapsular hematoma.*

icance of tetracycline treatment. He also stated that the pyelonephritis may set the stage for fatty liver. Fatty liver as a result of treatment with intravenous ampicillin has not been reported. In view of the fact that the usage of tetracycline is considerably reduced in recent years, it will be interesting to see whether pyelonephritis or ampicillin have a significant relationship to fatty liver of pregnancy.

Analyses of lipid in fatty liver of pregnancy [*3,4*] showed increased free fatty acid content in contrast to increase in triglyceride in nutritional fatty liver. Toxic effects of free fatty acid producing focal necrosis and inflammation were suggested [*5*]. Hemorrhagic disorders in fatty liver of pregnancy, particularly disseminated intravascular coagulation, were observed [*4,6,7*], but subcapsular hematoma of liver with rupture has not been reported.

Obviously, administration of heparin for the treatment of thrombophlebitis was a contributing factor for the development of subcapsular hematoma of the liver. The increased dose of heparin when pulmonary embolism was suspected was a further aggrevating factor for the subcapsular hematoma and subsequent rupture.

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