Digestive Diseases and Sciences, Vol. 30, No. 1 (January 1985), pp. 88–91

CASE REPORT

Unilateral Pleural Effusion Without Clinical Ascites in Laennec's Cirrhosis

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Pleural effusion is a complication of cirrhosis and ascites in approximately 5% of patients (1, 2). We report a case where a massive right-sided hydrothorax developed in an alcoholic cirrhotic who presented with gastrointestinal hemorrhage. Ascites was not evident on physical examination. At autopsy a small amount of ascites was found, as well as a transdiaphragmatic defect which permitted the ascites to pass into the right thorax.

Manuscript received January 12, 1984; revised manuscript received June 25, 1984; accepted August 1, 1984.

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CASE REPORT

A 46-year-old man was admitted with jaundice, massive right-sided pleural effusion (Figure 1), and upper gastrointestinal hemorrhage.

Mild shortness of breath and nonproductive cough had developed two weeks prior to admission and had progressively worsened. Melena occurred 48 hr before admission

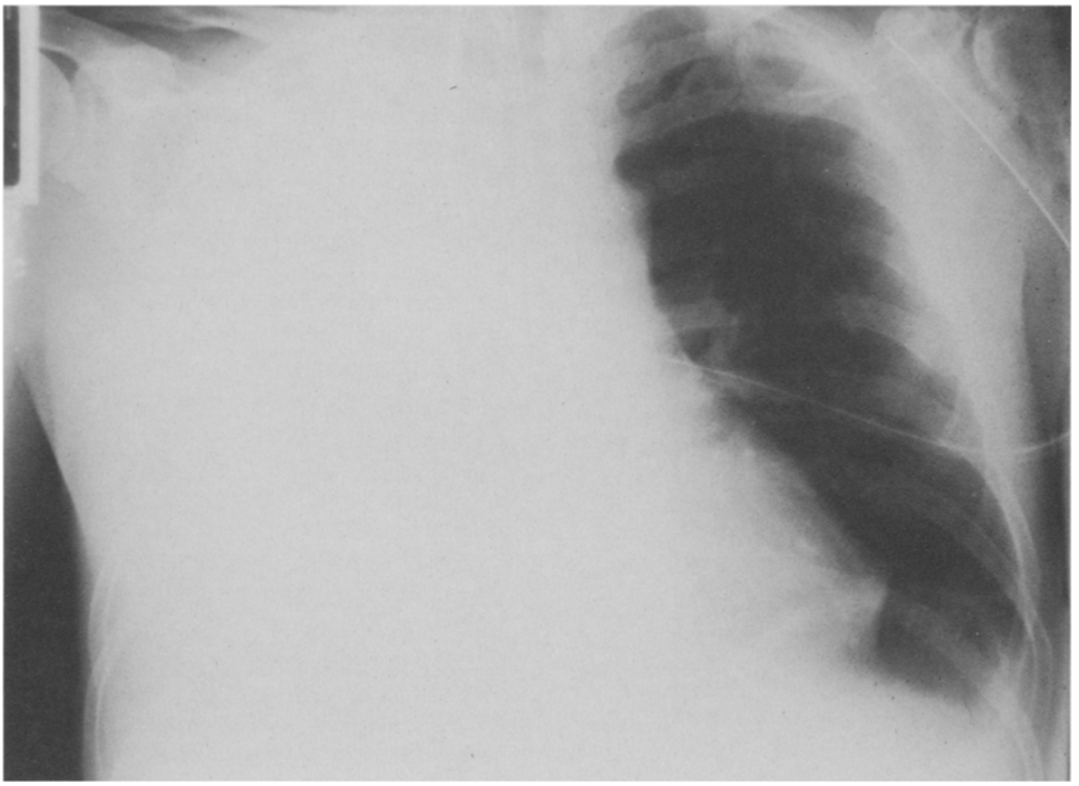

Fig 1. PA chest radiograph with a massive right-sided pleural effusion.

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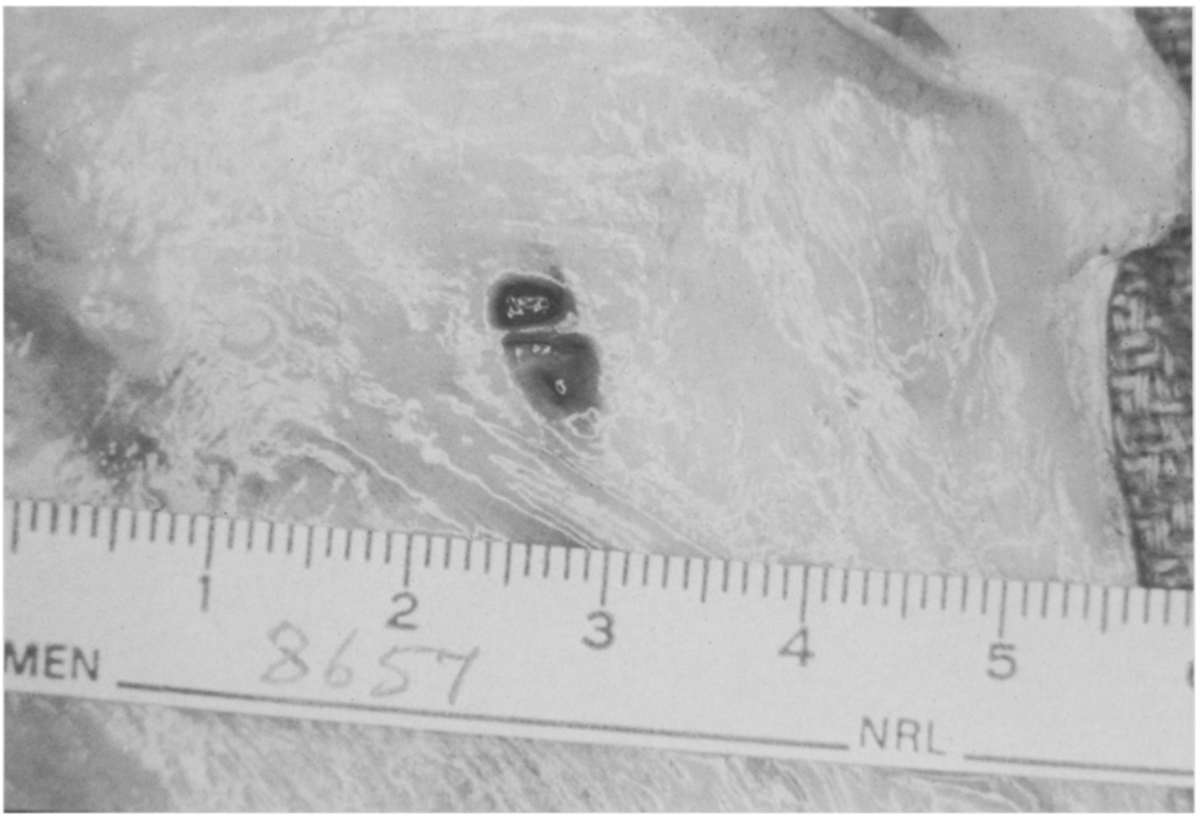

Fig 2. Photograph of the abdominal surface of the tendinous portion of the right hemidiaphragm taken at autopsy. The hole is tinted with methylene blue.

and hematemesis on the day of admission. There was no previous history of cardiac, pulmonary, or hepatic disease. He had a 40-pack-year cigarette smoking history and admitted to alcoholism since early adulthood, consuming 12 cans of beer daily.

On physical examination the patient was jaundiced and mildly dyspneic, but alert and oriented. He was afebrile, but orthostatic in the sitting position. Multiple spider telangiectasias were present over the upper chest. The right hemithorax was dull to percussion and breath sounds on the right were absent. The cardiac examination was normal. The abdomen was obese, and there was no clinical evidence of ascites. The liver had a span of 14 cm in the midclavicular line, with a nodular edge. The spleen was not palpable. Edema was absent. Rectal examination revealed melena.

Laboratory studies showed the hematocrit was 28%, the WBC 13,300 with a normal differential, prothrombin time 17.6 sec (normal 10.2–13.6), albumin 2.6 g/dl, total protein 5.6 g/dl, total bilirubin 5.3 mg/dl, alkaline phosphatase 219 IU/liter (normal less than 258), SGOT 230 (normal 8–40), and SGPT 84 (normal 4–29). An alpha-fetoprotein was 8 ng/ml (normal less than 20) and serologies for HB_sAg, anti-HB_s, anti-HB_c, and IgM HAV were negative.

The chest radiograph revealed a large right-sided pleural effusion (Figure 1). Right thoracentesis showed a benign transudate upon analysis.

Esophagogastroduodenoscopy revealed prominent esophageal varices, considerable blood in the stomach, but no active bleeding. The hospital course was complicated by recurrent gastrointestinal hemorrhage which was managed by volume support with crystalloid, fresh frozen plasma, and blood transfusions. Intravenous vasopressin and balloon tamponade were unsuccessful in control of the variceal hemorrhage, and the patient died 36-hr following admission. An autopsy was performed, and the massive right-sided pleural effusion was confirmed to be a benign transudate. There was no evidence of infection and the right lung was collapsed. Liver histology showed micronodular cirrhosis; 500 cc of ascitic fluid was found to be an uninfected benign transudate.

A defect in the diaphragm was not noted initially; 2000 cc of methylene blue were installed into the peritoneal cavity, and the dye appeared in the right pleural space. This permitted the identification of a defect in the tendinous portion of the right hemidiaphragm (Figure 2). Microscopic examination showed the defect connecting the two surfaces of the diaphragm to be lined by a thin layer of connective tissue (Figure 3).

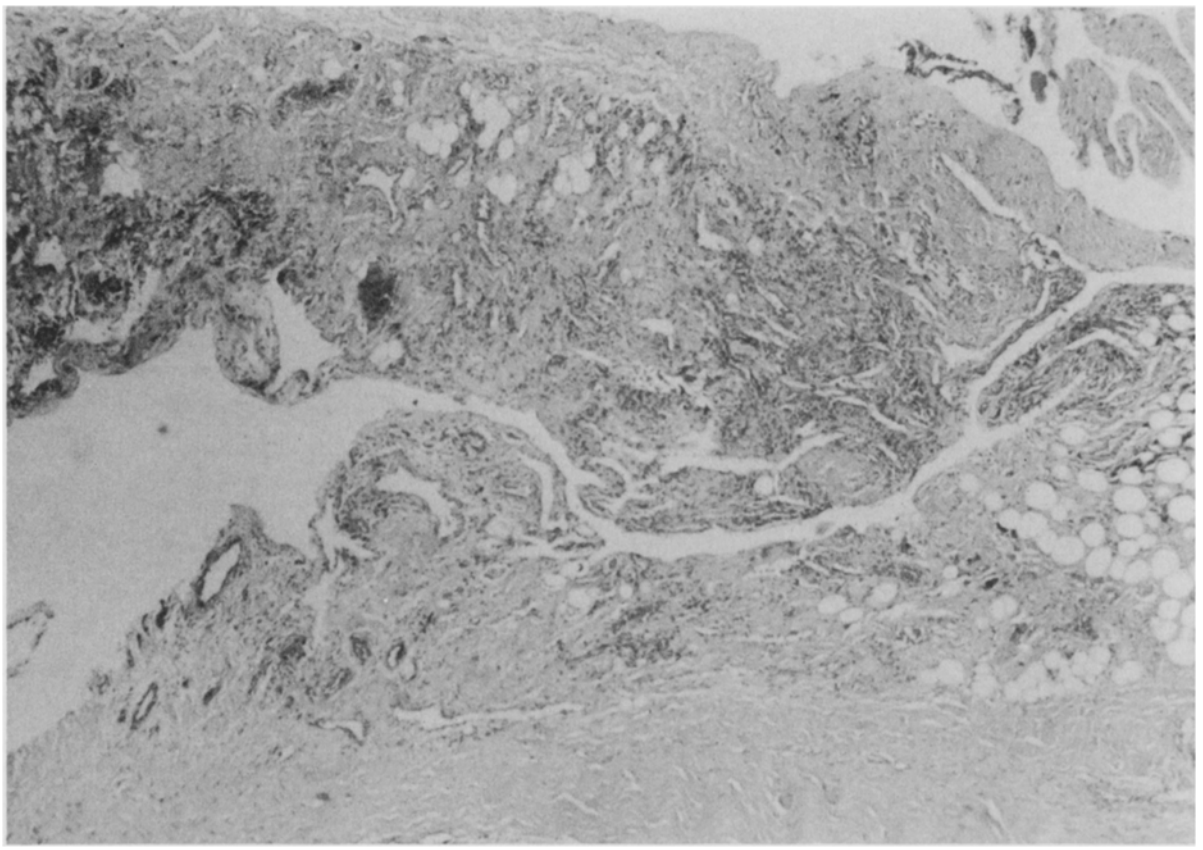

Fig 3. Photomicrograph of the right hemidiaphragm in the tendinous portion showing separation of the collagenous bundles with a free communication formed between abdominal and thoracic cavities (hematoxylin–eosin, ×40).

DISCUSSION

Although pleural effusion occurs in 5% of those with cirrhosis and ascites, this case emphasizes the fact that pleural effusion complicating cirrhosis and portal hypertension can be massive and that ascites may not be clinically evident (2–6). A recent report demonstrated that the physical examination of patients with equivocal ascites by experienced examiners is frequently equivocal and characterized by a high rate of false positives (7). Abdominal ultrasound is the test of choice concerning the presence or absence of ascites.

The presence of a communication between the abdominal cavity and the pleural space has been described previously (1, 8–13). Johnston and Loo proposed that the source of a massive hydrothorax complicating cirrhosis was the absorption of ascitic fluid through the diaphragmatic lymphatics (14). Lieberman and coworkers have described the pathogenesis of hydrothorax complicating cirrhosis and ascites as a diaphragmatic defect resulting from the pressure of ascites causing a separation of the taut collagenous fibers of the tendinous portion of the diaphragm and evagination of the thin serosal lining in the thoracic cavity (1, 9). Subsequent rupture of this bleb accounts for the intrathoracic accumulation of ascitic fluid. The negative intrathoracic pressure with a relatively positive intraabdominal pressure directs the ascites through the communication into the thorax. The same pathogenesis has been proposed as the cause of massive hydrothorax which may occasionally complicate peritoneal dialysis (15–19).

Several techniques can be used to confirm the diagnosis of hydrothorax complicating cirrhosis and ascites: (1) injection of a dye or radiolabeled albumin into the peritoneal cavity with its recovery from the thorax, (2) induction of a pneumoperitoneum with resulting hydropneumothorax, and (3) radionu-

clide imaging by injection of technetium-99 sulfur colloid into the peritoneal cavity with subsequent detection of the radioisotope over the thorax (8–11).

The treatment of the condition includes standard salt restriction and diuretic therapy, drug-induced pleural symphysis, and the installation of a peritoneovenous shunt (20–23).

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