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An alcoholic with pleural effusion

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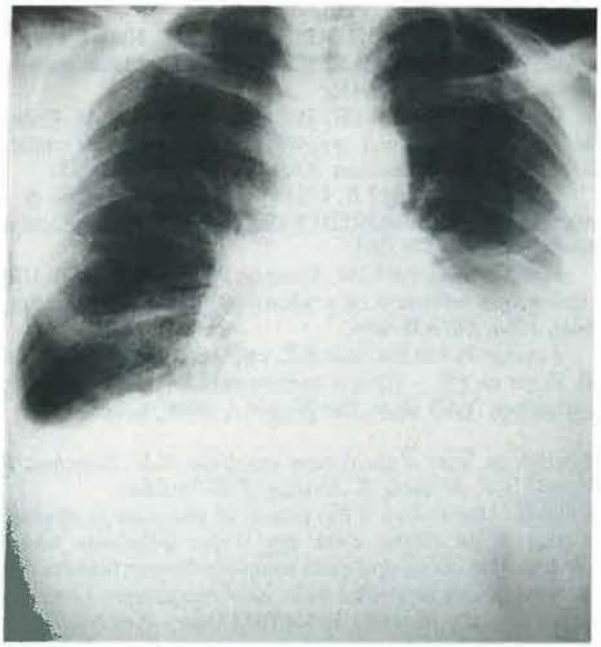
A 48 yr old man was admitted with progressive shortness of breath on exertion. There was no wheezing, orthopnoea or peripheral oedema. In the past he had experienced multiple blunt abdominal traumas (car accidents) without requiring medical treatment. He was a 90 pack-year smoker. Until 6 months before admission he drank 10–15 units of alcohol daily. Eight kg weight loss was noted during the last 6 months without further complaints.

Physical examination revealed a lean man, with normal body temperature and a respiratory rate of 24 per min, blood pressure 150/90 mmHg, heart rate 72 per min, normal central venous pressure. Dullness and diminished breath sounds were found over the basal parts of the thorax, especially on the left side. The heart was not enlarged, the heart sounds were normal without murmurs. No peripheral oedema was noted and there were no signs of ascites. The electrocardiogram was normal.

A chest roentgenogram demonstrated bilateral pleural effusion (fig. 1A) (courtesy of G.J. v.d. Woude, Dokkum). Treatment was begun with percutaneous pleural drainage (with continuous suction) on the left side. The amount of fluid on both sides initially decreased. After 4 wks, with minimal fluid production on the left side, the amount of fluid on the right side suddenly increased (fig. 1B).

SEE NEXT PAGE FOR DIAGNOSTIC TESTS AND DIAGNOSIS

A



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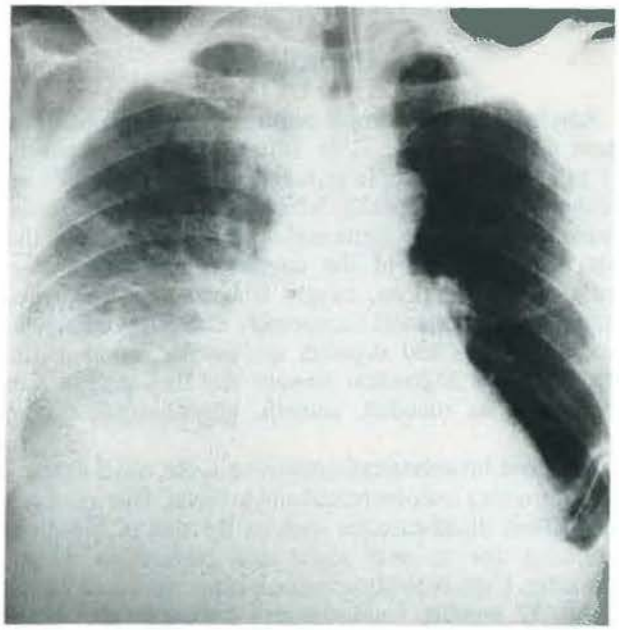

Fig. 1. – A: Posteroanterior chest roentgenogram showing bilateral pleural effusion. B: With a chest tube in the left pleural cavity the pleural effusion on the right side increased.

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Diagnostic tests

Laboratory evaluation revealed: normal blood cell counts, serum electrolytes, liver and renal function tests; serum amylase 1,598 $U \cdot l^{-1}$ (normal <300 $U \cdot l^{-1}$); albumin 27 $g \cdot l^{-1}$.

Pleuracentesis, performed on the left side, gave the following results on the aspirated fluid: LDH 393 $U \cdot l^{-1}$; total protein 23 $g \cdot l^{-1}$; amylase 63,138 $U \cdot l^{-1}$, fluid/serum amylase ratio 39.5; $0.4 \times 10^9 \cdot l^{-1}$ leucocytes; cytology negative.

Ultrasonography of the abdomen showed cystic changes of the pancreatic head without ascites. Computed tomography demonstrated a pancreatic pseudocyst (fig. 2) and there was evidence of slight amounts of perioesophageal fluid. No signs of fibrosis and/or calcification were present.

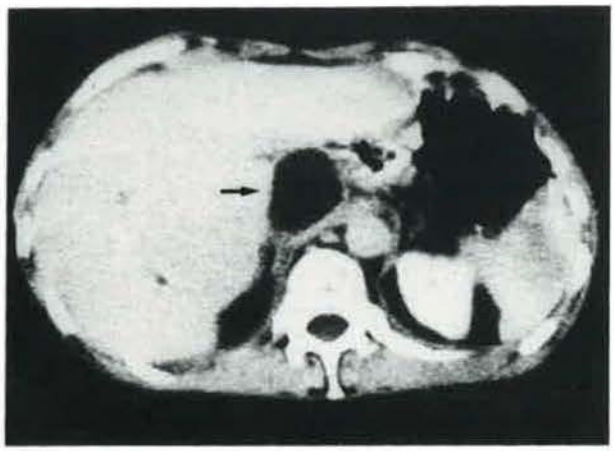

Fig. 2. – Computed tomography at the level of the pancreas. A cystic mass is seen in the head of the pancreas (arrow).

An endoscopic-retrograde-cholangio-pancreaticography was not performed because of unnecessary risks of infection.

Diagnosis: acute pancreatitis and perforation of a pancreatic pseudocyst to the pleural cavities.

When the amount of pleural effusion on the right side suddenly increased, pleuracentesis was performed on this side also and revealed high amylase levels and therefore a second thoracic drain was introduced. There were no signs of ileus, and accordingly the patient was fed by a naso-jejunal tube.

After 2 months the pleural drainage was terminated, and no recurrence of the pleural effusion occurred. Computed tomography showed disappearance of the pancreatic pseudocyst. Serum amylase and albumin levels returned to normal and the patient was discharged in good physical health. Currently, 1.5 yrs after discharge no recurrence of complaints has occurred.

Discussion

Pancreatic pseudocyst may complicate pancreatitis from any aetiology. The most common causes of pseudocysts of the pancreas are alcoholism, biliary tract disease and trauma. The cause of the pancreatitis in this patient is presumably alcoholism. However, an additive role in the pancreatic pseudocyst formation in this patient might be recurrent abdominal traumas.

The major complications of pancreatic pseudocyst are rupture, abscess formation and haemorrhage, all with a considerable mortality [1]. Perforation into the abdominal cavity was first recognized in the 1950's. More recently large pleural effusions have been described [1, 2]. Pleural effusions due to pancreatic diseases are mostly reactive with slightly elevated amylase levels. Very high levels of amylase in the pleural fluid are rare and can only be explained by rupture of a pancreatic pseudocyst with perforation into the pleural cavity such as by drainage of pancreatic fluid into the pleural cavity. Treatment with drainage by a chest tube, with concomitant conservative treatment of the pancreatitis, is usually effective in this situation [1, 3].

Our patient had no ileus, and thus prolonged nasogastric suction with total parenteral nutrition for treatment of the pancreatitis was not indicated [4].

If drainage by a chest tube fails, percutaneous catheter drainage of the abdominal pseudocyst can be considered for treatment [1, 2, 5, 6]. Conservative treatment of patients with internal pancreatic fistulae into the chest who present chronic massive pleural effusions has been more successful than for patients with internal fistulae into the abdominal cavity [2]. Therefore, in the case of pancreatic ascites, conservative treatment for more than a few weeks is probably not justified [7]. But, in the case of massive pancreatic pleural effusions, we believe that primary nonoperative therapy is justified and thoracic drainage should be continued as long as necessary and accountable, even for several weeks if required. Whenever conservative treatment is unsuccessful, surgical treatment has to be performed [1, 8, 9].

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Epanchement pleural chez un alcoolique. A.R.J. Girbes, P.E. Postmus, W. Jansen, E.J. v.d. Jagt, J.H. Kleibeuker.
RÉSUMÉ: Présentation de l'observation d'un alcoolique avec épanchement peural étendu. La pleurésie résultait d'une pancréatite aiguë avec formation d'une fistule reliant les pseudo-kystes pancréatiques à la cavité pleurale. Le traitement conservateur de ce malade, par drainage et aspiration pendant piusieurs semaines, s'avéra efficace sans complication. Cette attitude initiale qui, selon la littérature, est justifiée, doit, en cas d'échec, être complétée par la chirurgie. *Eur Respir J., 1990, 3, 934–936.*