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Short reports

Pleural effusion associated with urinary tract obstruction: support for a hypothesis

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Pleural effusions associated with urinary tract obstruction (urinothorax) appear to be unusual, only eight cases having been reported since the first description by Corriere and his colleagues in 1968.[1] The patient described here developed a pleural effusion associated with spontaneous perinephric extravasation of urine secondary to ureteral obstruction. However, the differing chemical composition of these fluids indicates that direct extension of urine into the pleural space, as previously suggested, had not occurred, and we propose that pleural effusions associated with urinary tract obstruction develop by another mechanism.

Case report

A 65-year-old black man was admitted to the Veterans Administration Wadsworth Medical Center complaining of weight loss and abdominal pain, associated with malaise, anorexia, constipation, and frequent postprandial vomiting. One week before admission a chest radiograph and barium studies were reported as normal. The past history included a prostatic carcinoma treated with radiation therapy in 1976 and a benign cyst of the left kidney demonstrated by intravenous pyelography in 1978. There was no history of cardiovascular disease. On admission, abnormal physical signs included decreased breath sounds and dullness to percussion posteriorly over the left chest. The left lower abdomen and left costovertebral angles were tender. No signs of cardiac failure or ascites were present.

Laboratory studies revealed a blood urea nitrogen (BUN) of 23 mg% (8·2 mmol/l), a serum creatinine of 1·8 mg% (159·1 μmol/l), and total serum protein of 7·6 g% (76 g/l). Serum amylase, albumin, and electrolytes were normal. A chest radiograph showed a left pleural effusion (fig 1). Diagnostic thoracentesis yielded 40 ml of straw-coloured fluid with the following characteristics: protein 0·8 g% (80 g/l), amylase 49 Somogyi units/dl, urea nitrogen 19 mg% (6·8 mmol/l), and glucose 105 mg% (5·8 mmol/l). There were 300 white blood cells/mm^3 (3×10^8/l), and 30 red blood cells/mm^3 (3×10^7/l). Routine cultures were negative and no malignant cells were found. Renal ultrasonography revealed a trans-sonic halo around the left kidney (fig 2). Percutaneous aspiration of this space yielded 50 ml of a clear serous fluid with a urea nitrogen of 139 mg% (49·6 mmol/l), creatinine 30 mg% (2652 μmol/l), protein 0·0 mg%, sodium 102 mmol/l, and potassium 11·7 mmol/l. Routine cultures were negative. Needle biopsy of the prostate revealed moderately well-differentiated adenocarcinoma. A retrograde pyelogram showed complete obstruction of the distal portion of the left ureter. At operation the left kidney was removed together with the inflamed perinephric fat, Gerota's fascia and the extravasated perinephric fluid. The kidney measured $10·8 \times 5·5 \times 5·5$ cm. Pathological findings included fibrotic thickening of the capsule, adventitial fibrosis, two cysts in the medulla, and patchy inflammation in the cortex. After operation, the patient's renal function improved and the left pleural effusion disappeared spontaneously. The effusion has not recurred.

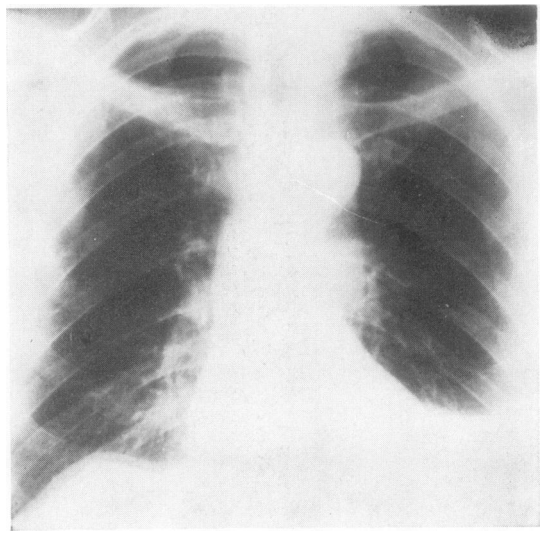

Fig 1 *Chest radiograph showing left pleural effusion.*

Discussion

Pleural effusion associated with urinary tract obstruction has been reported in eight patients, seven adults[1-4] and one neonate.[5] Urinary tract obstruction in these cases was caused by a variety of lesions including calculi,[3 4] surgical trauma,[2] prostatic hypertrophy,[4] a renal cyst[4], a posterior urethral valve,[5] and carcinoma of the prostate.[4] In those patients with unilateral urinary tract obstruction, the associated pleural effusions were ipsilateral.[1-4] When an abdominal operation was performed, a large amount of fluid was consistently noted in the perinephric and paranephric space.[2-4] Two authors reported the presence of urine in the pleural space[2 5] and the term urinothorax was adopted. In only one of these two patients, however, was

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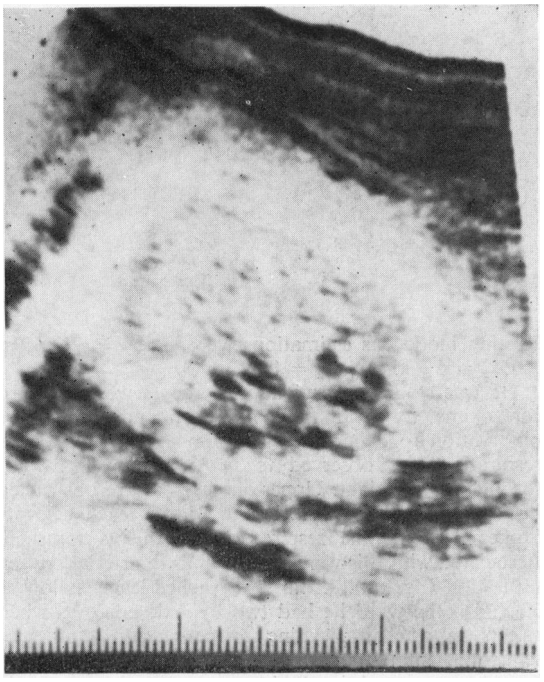

Fig 2 *Renal ultrasonograph demonstrating trans-sonic halo (extravasated perinephric urine) around the left kidney.*

the urea nitrogen concentration of the pleural fluid actually recorded. It was 55 mg% (19·6 mmol/l) compared with a serum value of 23 mg% (8·2 mmol/l).[5] In the other cases it was inferred that the pleural effusion was directly related to the urinary tract obstruction since the effusion consistently disappeared with resolution of the obstruction.[1 3 4] On the basis of this evidence, several authors have suggested that the pleural effusion was urine which had traversed the diaphragm via lymphatics and collected in the pleural space.[1-4 6] Indeed these lymphatics, which allow passage of material from the peritoneal cavity to the pleural cavity, have been well described,[7 8] but in addition, Lemon and Higgins[7] by injecting particulate material into the pleural space demonstrated in dogs the presence of two descending transdiaphragmatic lymphatic vessels. Each vessel was shown to pass down the posterior wall of the thorax and communicate with lymph nodes in the region of the kidney. These nodes in turn communicate with the cisterna chyli. In man, a similar arrangement exists. The lower posterior intercostal lymphatic vessels enter the thoracic duct by means of a trunk that descends on each side of the vertebral column through the diaphragm, to open into the cisterna chyli.[8] It appears, therefore, that a route exists for passage of fluid from the pleural space via the retroperitoneal tissues to the cisterna chyli and hence the thoracic duct.

In a recent review Black[9] notes that the pleural space is a potential one which generally contains only a few ml of transudative fluid. The fluid is formed by the parietal pleura and is absorbed by capillaries and lymphatics in the visceral pleura. Transudative pleural effusions are known to form when systemic or pulmonary venous hypertension is present, when the plasma colloid osmotic pressure is reduced by hypoproteinaemia, when the intrapleural pressure becomes excessively negative, or when there is transport of transudative peritoneal fluid across the diaphragm into the pleural space. The pleural effusion in our patient was a transudate with a urea nitrogen of 19 mg% (6·8 mmol/l). This contrasts to 139 mg% (49·6 mmol/l) in the perinephric urine making direct extension of this fluid into the thorax unlikely. As in other patients, inflammation and oedema of the retroperitoneal tissue was demonstrated at laparotomy and may have resulted in compression of the lymphatic vessel as it passes through the diaphragm to enter the retroperitoneal space. Could the transudative effusion in our patient, in the absence of congestive heart failure, hypoproteinaemia, or ascites, simply have been due to impaired drainage by the descending trans-diaphragmatic lymphatic vessel? It is usually believed that the formation and absorption of pleural fluid can be explained solely by the opposing hydrostatic and oncotic pressures acting on the visceral and parietal pleura. Nevertheless, elevation of right atrial pressure in right heart failure is known to reduce lymphatic drainage from the thoracic duct, occasionally resulting in a pleural effusion. If the descending lymphatics described here, with their similar intraluminal pressure, play a significant part in draining the pleural space, then their obstruction would similarly result in a pleural effusion. Our observation certainly argues against previous claims that pleural effusions associated with urinary tract obstruction are caused by the collection of urine in the pleural space, and provides evidence in support of another hypothesis for the development of a transudative pleural effusion.

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