

Scintigraphic Demonstration of Trans-Diaphragmatic Migration of Ascites

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＝국문초록＝

동위원소 주사로 확인된 복수의 이동에 의한 수흉

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염증성 복부질환에서 늑막침윤이 일어나는 기전에 대하여는 잘 알려져 있으나, 비염증성 복수의 늑막강내로의 이동에 관여하는 기전에 대하여는 여러가지 가설이 제시되어 있다. 저자들은 만성 신부전증 환자에서 Tenckhoff 카테타의 복강내 이식후 발생한 우측 수흉이 투석액의 이동에 의한 것임을 동위원소주사법을 통하여 확인하였는 바, 이에 관여하는 기전에 대한 문헌고찰과 함께 보고하는 바이다.

Introduction

There are many diseases in which pleural effusion develops without direct extension of the offending organism or cell into the thorax. They are intra-abdominal or retroperitoneal in location and involve the transfer of fluid from below the diaphragm into the pleural space. Pleural involvement from subphrenic inflammation is easy to under-

stand, but the precise mechanism of migration of noninflammatory ascites through diaphragm is not clear in the patients with liver cirrhosis, Meigs' syndrome or peritoneal dialysis.

We report a case where trans-diaphragmatic migration of peritoneal fluid has been demonstrated in a patient with peritoneal dialysis by scintigraphic method, with a brief review of literatures about the possible mechanisms.

Case Report

A 46-year-old woman was admitted for dyspnea and diffuse abdominal pain with duration of two

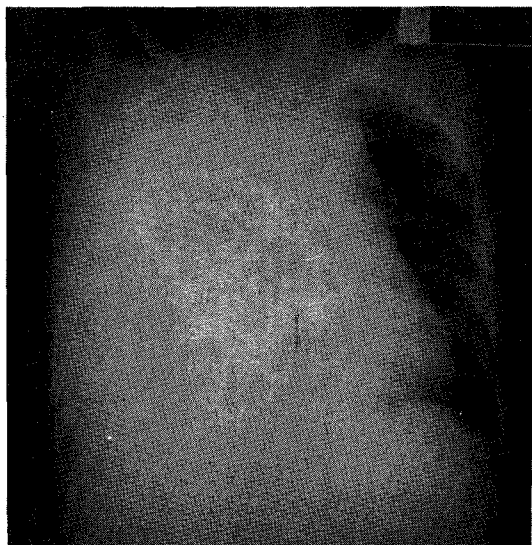


Fig. 1. Chest frontal radiograph shows a massive right pleural effusion producing total opacification of the right hemithorax.

days. She had been previously diagnosed as having a renal disease, and a diagnosis of CRF was made one month earlier.

On admission, she looked acutely ill, and was afebrile with regular pulse rate of 78/min. and a blood pressure of 120/90 mmHg. Other physical findings and laboratory tests support the diagnosis of chronic renal failure.

The initial chest radiograph shows streaky and spotty fibrocalcific densities in both upper lung fields suggestive of inactive pulmonary tuberculosis. Tenckhoff catheter was implanted into the peritoneal cavity at operating room under local anesthesia. One day after implantation, the patient complained of breathlessness of acute onset. Subsequent chest radiograph demonstrated a massive right pleural effusion (Fig. 1). Thoracentesis showed a fluid that had the characteristics of a transudate as did Peritosol® (dialysate solution). One day after the thoracentesis of 1,300 cc, the right hemithorax was entirely refilled.

To certify that right hydrothorax was due to

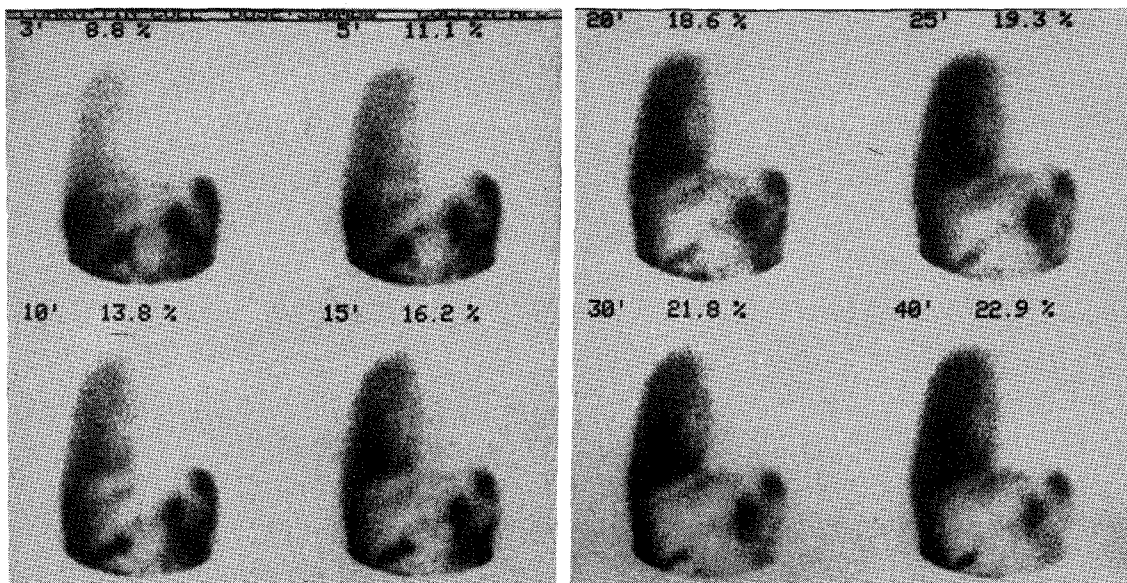


Fig. 2. Serial scintigraphic images of abdomen and thorax were obtained after intraperitoneal injection of 5m Ci of ^{99m}Tc -tin colloid. Migration of radioisotope appears at 3 minute image, and increased continuously.

migration of peritoneal fluid, 5m Ci of ^{99m}Tc -tin colloid was injected intraperitoneally and scintigraphic images were obtained serially at 3, 5, 10, 15, 20, 25, 30 and 40 minutes after injection of the radiopharmaceutical. At 3 minute the radioisotope appeared in the right hemithorax and increased continuously (Fig. 2). After further thoracentesis and removal of the catheter, a repeat chest radiograph revealed that most of the pleural effusion was no longer present. The conclusion was drawn that pleural effusion occurred through peritoneo-pleural communication, particularly by way of diaphragmatic defects.

Discussion

Since ascites may be associated with pleural effusion, it might reasonably be anticipated that peritoneal dialysis as well as liver cirrhosis might sometimes lead to hydrothorax; at least two such cases with peritoneal dialysis have been reported¹⁻²⁾. Since such patients are uremic, it may not be possible to distinguish pleural effusion secondary to ascites from that associated with uremia itself. Uremic pleuritis as a sole cause can be excluded because there are many patients who have uremia but never develop pleural effusion, and this can not explain pleural effusion occurring in non-uremic patients and unilaterality. The most probable mechanism proposed in the literatures is that hydrothorax is derived directly from the peritoneal to the pleural space either by way of diaphragmatic lymphatics and in others by way of diaphragmatic defects²⁻⁹⁾.

Lemon et al*. described lymphatic channels that carry particulate matter from the peritoneum to the thorax and found that those of the right hemidiaphragm are larger and carry more fluid than those of the left. Johnston et al⁵⁾. confirmed these observations by an experimental work; it was shown that, in the presence of ascites, carbon particles or radioiodinated serum albumin instilled into the peritoneal space passes into the pleural space in a patient

*Cited from reference 4

with right hydrothorax and that flow is always from the peritoneum to the pleura and never in the reverse direction. The autopsy showed no gross defect in the right hemidiaphragm.

On the other hand, severe and prolonged distention of the peritoneal sac by fluid or air stretches the diaphragm and its closely attached parietal peritoneum and pleura to such an extent that the fibers pull apart. The widened interstices permit the diaphragm to become microscopically and even grossly permeable to air or fluid³⁾. These have been demonstrated either by rapid passage of dye from the ascites into pleural effusion, or induction of hydropneumothorax by intra-abdominal instillation of oxygen^{8,9)}. Lieberman et al^{7,8)}. have established this hypothesis with the following evidence, based on a study of a group of cirrhotics;

- 1) ^{131}I injected into the ascites appeared in higher concentration in the pleural fluid than in the blood plasma or lymph.
- 2) Thoracentesis reduced the volume of the ascites.
- 3) Air (500~1,000 cc) injected into the peritoneal sac of the sitting patient appeared in the pleural fluid within 1 to 48 hrs.
- 4) A small opening or leaking blister in the diaphragm was identified at thoracoscopy or autopsy in several of these patients.
- 5) Separation of collagenous bundles in the tendinous portion of the diaphragm was demonstrated at autopsy in each of the patients.

After all, in some patients fluid transfer from the peritoneal to the pleural space occurs by way of diaphragmatic lymphatics and in others by way of diaphragmatic defects.

To find the source and route of pleural fluid, that is, to prove the migration of fluid from the peritoneal to the pleural space, scintigraphic study can be done by intraperitoneal injection of radiopharmaceutical agent such as ^{99m}Tc -sulfur or tin colloid or ^{99m}Tc -MAA^{2,6,9)}.

Verreault et al⁹⁾ postulated that there is a relationship between ascites and pleural effusion if radioisotope injected intraperitoneally is mixed with ascites and its migration from peritoneal to pleural space is right sided as is the pleural effusion. And the speed at which the radioisotope migrates from peritoneal to pleural space may be a differential point between the two pathophysiological mechanisms described previously.

If it happens within a few minutes as was seen for our patient, a diaphragmatic defect is probably present, particularly when the accumulation is as intense as peritoneal activity. If it takes a longer period of time as was reported by Verreault et al. migration of ascites fluid may be attributed to diaphragmatic lymphatics particularly when that accumulation is less intense than peritoneal activity.

Conclusively, scintigraphic study can show the source of pleural effusion and can suggest the mechanism of migration of ascites in the patients with liver cirrhosis, Meigs' syndrome, and also in patients with peritoneal dialysis. And thus we can manage the patient properly by coping with possible situations if hydrothorax developed.

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