HEPATİC HYDOTHORAX WITH AND WITHOUT ASCITES
Scintigraphic Demonstration of Functional UnidirectionalDiophragmatic Leaks

ASİTLİ ASİTSİZ HEPATİK HİDROTORAKS
Tek Yönlü Diyafragmatik Kaçağın Sintigrafiyle Gösterilmesi

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SUMMARY

Hydrothorax is not a very common symptom in patients with hepatic cirrhosis, especially when there is no clinical evidence of concomitant ascites. Before accepting the hepatic origin of the hydrothorax it is necessary to exclude other cardiac, pulmonary or renal pathologies which could be partly or entirely responsible for this symptom. The existence of ascites may help to suggest the origin of hydrothorax. In patients with hydrothorax without ascites, establishing the diagnose is more complicated. We present two hepatic cirrhotic patients with right hydothorax in the absence and presence of ascites using radioisotopic techniques it was demonstrated that in both situations the pleural fluid originated in the abdomen and hence was transported into the pleural cavity. There was no fluid transfer in the opposite direction.

(Key Words: Ascites, Cirrhosis, Hydrothorax, 99m-Technetium tin colloid.)

ÖZET

Karaciğer sirozlu hastalarda özellikle asit gelişmemişse hidrotoraks ender gelişir.

Hidrotoraksın karaciğer orijinli olduğunu kabul etmezden önce akciğer, kalb ve böbrek patolojileri elimine edilmelidir. Asitsiz hastalarda tanı güçleşir. 2 karaciğer sirozlu (biri asitsiz) ve sağ hidrotorakslı hastada radyoizotop tekniğiyle plevral sıvının karın orijinli olduğunu gösterdik. Karşı yönde sıvı transferi yoktu.

(Anahtar Sözcükler: Karaciğer, Siroz, Plöral Effüzyon)

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In liver cirrhosis ascites generated hydrothorax is a well known phenomenon for many years. Later on it has been reported that in chronic liver patients transudatic hydrothorax is also possible in the absence of ascites.

Lieberman et al. (1) have reported an occurence rate of hydrothorax of 6% in 330 cirrhotic patients with ascites. The same rate has been found by Johnston et al. (2) in a series of 200 cirrhotic patients. In the later series none of the 54 patients without ascites had pleural fluid collection.

In hepatic cirrhosis, pleural effusions usually follow the ascites and mostly occur on the right side. Less frequently pleural effusions may appear before ascites arises.

We present two cirrhotic patients with hydrothorax, the first with ascites.

CASE 1

A 67-year-old man with liver cirrhosis, hepatitis-C and hemochromatosis was admitted to the hospital because of jaundice, dyspnoe, severely increasing ascites and a fluid collection in the right hemithorax. A transudatic type fluid was found at pleural and peritoneal ponction. Cardiac, renal, pulmonary and neoplastic pathologies were excluded. As the final diagnosis, right hydrothorax and sever ascites due to advanced hepatic cirrhosis was established. A liver transplantation was temporarily not considered due to chronic osteomyelitis in the left tibia.

To elucidate the mechanism of the pleural fluid formation 185 MBq of tin colloid labeled with 99mTechnetium (Livoscint, Medgenix) were injected intraperitoneally. The reason for using a colloid, a non soluble radiopharmaceutical, was to avoid diffusion. Scintigrams were performed with the gamma camera fitted with a high resolution, parallel-hole collimator and peaked on the 140 keV γ-rays of 99 mTc. Five and 24 hours following the injection, apart from the activity within the abdomen, a distinct homogeneous tracer distribution was observed in the right pleural cavity. A range of mediastinal lymph nodes could also easily be recognized (Fig 1).

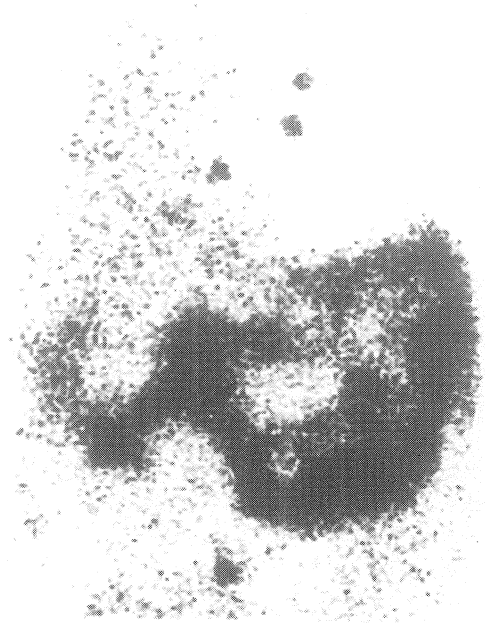

Fig 1: Lying anterior view of abdomen and thorax, five hours after intaperitoneal injection of tin colloid labelled with 99m Tc. Apart from the activity within the abdomen, an homogeneous tracer distribution in the right pleural cavity and clearly delinieated mediastinal lymph nodes are seen.

A possible fluid flow in the opposite direction was investigated by intrapleural injection of 370 MBq of 99 mTc-tin colloid 3 days after the previous examination. No activity whatsoever could be observed in the abdomen even at 24 hours.

Hence a functional, transdiaphragmatic, unidirectional communication between the peritoneal and the right pleural cavities was established.

CASE 2

A 63-year -old man with postnecrotic liver cirrhosis and hepatitis-C was evaluated in view of a possible hepatic graft. Jaundice, dyspnoe and severe denutrition were observed but there were no clinical signs of ascites. A massive fluid accumulation was found in the right hemithorax. Thoracocentesis yielded nearly 200 ml of yellow fluid with a low protein content. One day after the thoracocentesis a rapid reaccumulation of the pleural fluid was observed. Complementary examinations excluded cardiac, renal, pulmonary and neoplastic pathologies as the possible cause of the fluid effusion and established the final diagnosis as advanced hepatic cirrhosis with right hydrothorax without ascites.

To elucidate the mechanism of the pleural fluid formation, 1 L of glucose 5 % and 185 MBq of tin colloid labeled with 99mTc (Livoscint, Medgenix) were injected in the abdominal cavity. Scintigrams were performed with the gamma camera fitted with a high-resolution, parallel-hole collimator and peaked on the 140 keV γ-rays of 99mTc.

After 4 hours most of the tracer was found between the liver dome and the right hemidiaphragm and an homogeneous activity distribution was seen over the right hemithorax when the patient was supine. Additional images made when the patient was sitting or lying on his right side showed the activity to be freely movable a long with the pleural fluid. Lymph nodes were also clearly delineated in the mediastinum and along the diaphragm (Fig. 2).

To look for a possible flow in the opposite direction, one week later 370 MBq of 99mTc-tin colloid were injected into the pleural cavity. Even after 24 hours no activity was observed within the abdomen.

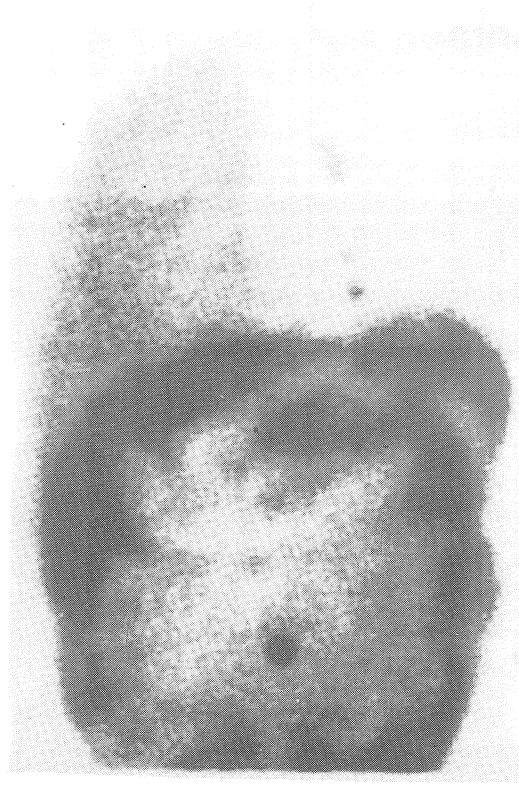

Fig 2: Sitting anterior view of thorax and abdomen 5 hours after intraperitoneal injection of 99m Tc-tin colloid. diffuse activity in the right hemithorax and a pronounced activity in mediastinal lymph nodes are visualized.

On the basis of these scintigraphic observations functional transdiaphragmatic unidirectional fluid transportation between the peritoneum and the right pleural cavities was detected.

DISCUSSION

In advanced liver cirrhosis abdominal fluid transudation is easily understandable in view of the increased intrahepatic capillary pressure. The explanation for an in loco fluid effusion in the pleural cavity however is less obvious, since su-

pradiaphragmatic hydrostatic pressure is not expected to be elevated. Low plasma protein concentration which causes reduced osmotic pressure might only be a minimal contributory factor, considering the poor correlation between the development of effusion and the plasma protein level (3). On the other hand, a plausible answer is provided if the pleural fluid is proven to come from the peritoneal cavity, as has been demonstrated in our two patients.

Some mechanisms have been suggested in order to explain the transdiaphragmatically occuring hydrothorax.

It is known that the diaphragm may contain small congenital holes through which the peritoneum may herniae and open into the pleura because of the intrapleural negative pressure. If the peritoneal fluid production is higher than the continuous lymphatic drain of the abdomen, intrapleural negative pressure, in combination with intraabdominal pressure during inspiration, may cause the passage of ascites through the diaphragma either via small defects (1) or via its lymphatics (2). Even if, due to the increasing fluid accumulation, the original intrapleural negative pressure may have become less evident, the combined thoracic and abdominal respiratory movements may result in a pumping up of the abdominal fluid towards the pleural cavity.

The existence of diaphragmatic defects have been shown in several studies. Lieberman et al. (1) noticed that following the intraperitoneal administration of 500-1000 ml of air, in all 5 patients with cirrhosis, ascites and pleural effusion, a pneumothorax developed within 48 hours. They were able to observe air bubbles coming through, an otherwise undetectable diaphragmatic defect at thoracoscopy in one of their patients.

Johnston and Loo (2). demonstrated that after the intravenous injection of radiolabeled albumin, radioactivity first appeared in the peritoneal fluid and then in the pleural fluid. In their continuous ambulatory peritoneal dialysis (CAPD) pediatric group, Bjerke et al. (4) have demonstrated that 2 % of the patients develop unilateral massive hydrothorax; this complication is fully treated by the repair of diaphragmatic eventration.

The diaphragmatic defects have also been proven by autopsy findings (2,5).

Another possible explanation for the peritoneo-pleural fluid transport might be lymphatic drainage. The transfer of the fluid from the peritoneal cavity towards the supradiaphragmatic region is realized by the well developed supra and infra diaphragmatic lymphatic plexus. The cavity surface of the parietal and visceral pleura is covered with a single layer of mesothelial cells. Electron microscopy shows that the parietal pleura contains stomata and areas of loose connective tissue covering lymphatic spaces (membrana cribriformis). Though these stomata the fluid reaches the pleural cavity and areas of loose connective tissue covering lymphatic spaces (membrana cribriformis). Through these stomata the fluid reaches the pleural cavity and the visceral mesothelium. Under this layer the subpleural lymphatics can drain the fluid towards the deeper lymphatics in the hilus. Excess fluid, proteins and particles such as cells can be removed thorught these openings (3). Animal experiments have shown that the diaphragmatic lymphatic plexus is better developed on the right than on the left which may account for the very much higher incidence of right-sided effusions(6).

Even if the exact mechanism of the abdomino-pleural fluid translocation can not be ascertained, the immediate fluid reaccumulation in the pleura following the thoracic drainage and the rapid peritoneopleural transport as shown in our second patient, makes one think that the transport via the lymphatic plexus plays only a secondary role. We think that in the development of transdiaphragmatic hydrothorax, some congenital bleb like structurally veak areas in

the diaphragm are perforated by increared intraabdominal pressure and this mechanism plays the most important role. On our case 2 (hydrothorax without ascites), 4 hours after the intraperitoneal tracer injection a high activity was already found in the right pleural cavity. An increased abdominal pressure hardly could have been a causative factor due to the lack of detectable ascites. In hydrothorax without detectable ascites only the high aspiration capacity of the negative pleural pressure offers a satisfactory explanation. In these cases one may suppose that, as soon as fluid transudation in the abdomen takes place, the liquid is continously aspirated towards the pleural cavity. The predominant pumping direction from the peritoneal to the pleural cavity probobly also explains why, in spite of gravity, no displacement of pleural fluid towards the abdomen could be observed.

In cirrhotic patients with hydrothorax in the absence of ascites the differential diagnosis to elucidate the underlying primary pathology is sometimes not so easy. In these cases, if the pleural effusion shows the characteristics of a transudate, congestive left ventricular failure, chronic hepatic failure or nephrotic syndrome has to be considered (7). In cirrhosis without ascites, the transudatic hydrothorax is frequently caused by transdiaphragmatic fluid translocation. In this situation scintigraphic studies may help to establish the diagnosis and explain the possible mechanisms of the fluid accumulation (1,5,8,9,10,11,12). This is not a matter of merely academic interest, since Runyon et al. have reported that patients with cirrhosis and massive right sided pleural effusions may have congenital diaphragmatic defects that predispose them to life-threatening fluid depletion when chest tubes are inserted (13).

The scintigraphic demonstration of peritoneo-pleural transdiaphragmatic fluid passage is not a new technique. However, although it has well established benefits it is still not routinely used. Herefore we wanted to emphasize the importance of scintigraphy once more on the basis of two examples of unilateral right sided hydrothorax in cirrhotic patients with and without ascites. In these two patients the existence of a peritoneo-pleural unidirectional transdiaphragmatic fluid transport was proven by 99mTc-tin colloid scintigraphy.

In conclusion, in cirrhotic patients with hydrothorax in the absence or presence of ascites, scintigraphic methods are among the primary techniques to be considered. These non-invasive methods will hep the physician to find out the origin of the hydrothorax in order to establish the diagnosis and appropriate treatment (5,8,9,11,14).

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