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Original Article

Evaluation of Hepatic Hydrothorax by ^{99m}Tc-MAA Peritoneal Scintigraphy

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Abstract

Background: Hepatic hydrothorax occurs 6-10% of advance cirrhotic patient. If we can diagnosed quickly and accurately, it can present the patients from unnecessary investigations and early start correct treatment.

Objective: To evaluate the results of using ^{99m}Tc-MAA peritoneal scintigraphy for demonstrate perito-neopleural communication in the patients with suspected hepatic hydrothorax.

Material and Method: Descriptive retrospective study. Collected and analized data of the cirrhotic patients who was investigated by ^{99m}Tc-MAA peritoneal scintigraphy in nuclear medicine section, Rajavithi hospital, Thailand from 2005-2012.

Results: Eleven patients were identified, 7 male and 4 female average age 52.55 years. Most common cause of cirrhosis was viral hepatitis C (45%) and almost all (10/11) were in child class C. 88% of patient who was evaluated by gastroscope had esophageal varies. 91% of patients had ascites which 90% were massive, 20% moderate and 10% small amount. Peritoneal scintigraphy were positive in 7 patients, excluded a patient who was suspected cause of pleural effusion form pulmonary infection, percent of demonstrated peritoneo-pleural communication was 70. A patient who didn't have ascites also had positive scintigraphy. Time since radiotracer administration until it appears in pleural cavity was less than 1 hour in 57% of patients, 2 hours 14.3%, 4 hours 14.3% and 24 hours 14.3%.

Conclusion: ^{99m}Tc-MAA peritoneal scintigraphy is a safe, rapid, cheap, minimally invasive and low radiation method for evaluation of the patients who was suspected hepatic hydrothorax with medium positive results (70%) and high specificity.

Keywords: Hepatic hydrothorax, ^{99m}Tc-MAA, peritoneal scintigraphy

Introduction

Hepatic hydrothorax is explained in detail as the presence of more than 50 ml. pleural effusion in cirrhotic patients without primary lung or heart disease[1-5]. This condition occurs in approximately 6-10% of patients with advance cirrhosis[6,7] and it is thought to be 1%-2% of all pleural effusion[1,8,9]. The incidence of a pleural effusion in cirrhotic patients is much higher with the concomitant presence of ascites fluid.

Although the exact mechanism of hepatic hydrothorax is controversial but the most plausible etiology is direct passage of the ascites fluid in to the pleural space via defects in the diaphragm[1,20].

A rapid and simple method for evaluation of communication between peritoneal and pleural space is peritoneal scintigraphy. Peritoneal scintography is nuclear scan that can be performed by intraperitoneal administration of radiolabled tracer such as ^{99m}Tc-sulfur collid, ^{99m}Tc- human serum albumin, ^{99m}Tc-macroaggregated albumin (MAA). Later scintigraphic images of thoraco-abdominal region are performed by Gamma camera. The migration of radioisotopes from the peritoneal cavity in to pleural space confirms the presence of a communication.

Material and Method

The study is descriptive retrospective study. Collected and analized data of the cirrhotic patients with clinically suspected hepatic hydrothorax who was investigated by ^{99m}Tc-MAA peritoneal scintigraphy in nuclear medicine section, Rajavithi hospital, Thailand, from 2005-2012.

Peritoneal scintigraphic protocol:

1. Injection of ^{99m}Tc-MAA 5 mCi into peritoneal cavity by paracentesis.

2. Image acquisition by GE MPR single-head gamma camera, energy setting

140 keV ± 10% window.

2.1 dynamic 1 hour (60 sec./frame, total 60 frames)

Matrix size 128x128, low energy general purpose collimator (LEGP)

zoom 1.0, anterior view, FOV included chest and upper abdomen.

delay images: preset count 1000 kc.

Matrix size 256x256, LEGP anterior view. delay image every hour or as requested.

Extra views such as RAO, LAO as requested. delay images till 24 hours after the tracer injection are obtained in the patients without radiotracer accumulation in pleural space during the first 6 hours.

Results

Elevent patients were included. Data considering age, gender, etiology of cirrhosis, Child classification, serum albumin, presence or absence of esophageal varices and complication or associated disease of all patients are present in table 1.

Dataconsidering ascites, pleural effusion, findings of peritoneal scintigraphy and time from tracer administration until positive scan are shown in table 2.

All 11 patients were 7 male and 4 female. Average age was 52.55 years (18-78 yr.). The most common etiology of cirrhosis in this study was viral hepatitis C (45%), followed by alcohol (36%), viral hepatitis B (27%), autoimmune (9%) and unknown etiology (9%).

Ten patients (91%) were in Child class C. Serum albumin was 1.2-3 g/dL. Eight patients were evaluated by gastroscope, 7 in 8 (88%) had eso-

phageal varices. Four patients had clinical of GI bleeding. There are 2 patients had hepatomas and 1 patient was suspected.

Ten patients (91%) had ascites. Seven from ten (70%) had massive ascites, 20% had moderate ascites and 10% had minimal ascites. One patient didn't have ascites (by ultrasonography and CT scan).

The pleural effusion (by CXR) was on the right 8/11 (73%), left 3/11 (27%). Six patients had the results of pleural effusion analysis, 5 were transudate, 1 was exudate.

There was compatibility between CXR and scintigraphic results in 7 patients (63.64%). However if exclude a patient with negative scan who had exudative pleural effusion and right middle lung infiltration from CXR (because pleural effusion may be from pulmonary infection), the positive result will increase to be 70% (7 in 10 patients). Time since radiotracer administration until positive scan is less than 1 hour in 4 patients (57%), 2 hours 1 case (14.3%), 4 hours 1 case and 24 hours 1 case.

From all of scan positive patients, there were 5 positive on right side (71%), 2 positive on left side (29%)

All of scan result negative patients (4) had massive ascites. Three had right pleural effusion on

Table 1 Information of the cirrhotic patients.

No	age	sex	cause of cirrhosis	child	serum albumin	Esophageal varices	Complication/ Associated disease
1	59	m	Viral hepatitis B	C	2.3	NE	Hepatocellular carcinoma
2	74	f	unknown	B	3	present	UGI bleed (DU & erosive gastritis), CKD, DM, HT
3	45	m	Alcoholic, Viral hepatitis C	C	2.1	NE	Acute renal failure, Spontaneous bacterial peritonitis
4	54	m	Alcoholic, Heptitis B virus carrier	C	2.5	NE	End stage renal disease (from Membranoproliferative glomerulonephritis)
5	44	m	Alcoholic	C	1.2	absent	
6	40	m	Alcoholic, Viral hepatitis B	C	2.4	present	UGIB (mucosal tear at EG junction), hepatic encephalopathy
7	65	f	Viral hepatitis C	C	1.7	present	Spontaneous bacterial peritonitis
8	18	m	AIH+PSC	C	2.1	present	LGIB (5colonic polyps), Spontaneous bacterial peritonitis
9	44	f	Viral hepatitis C	C	2.7	present	R/O small hepatocellular carcinoma 1 cm.
10	57	m	Viral hepatitis C	C	2.6	present	Hepatocellular carcinoma, UGIB
11	78	f	Viral hepatitis C	C	2.4	present	UGIB, erosive gastritis, DU, DM

AIH = autoimmune hepatitis, PSC = primary sclerosing cholangitis, NE = non evaluated, CKD = chronic kidney disease, UGIB = upper gastrointestinal bleeding, LGIB = lower gastrointestinal bleeding, DU = duodenal ulcer

Table 2 Information of ascites, pleural effusion and scintigraphic findings.

No	ascites	pleural effusion on CXR (amount/side)	pleural tapping	Scan findings	time of positive (hours)
1	medium positive	massive right	transudate	positive right	less than 1
2	minimal positive	massive right	NE	positive right	less than 2
3	massive positive	modearte left	NE	positive left	4
4	massive positive	moderate right	transudate	negative	
5	massive positive	massive right	NE	negative	
6	medium positive	massive right	NE	positive right	less than 1
7	massive positive	massive right	transudate	positive right	less than 1
8	massive positive	minimal right, minimal infiltraion in RML	exudate	negative	
9	negative	moderate right	NE	positive right	less than 1
10	massive positive	small left	transudate	negative	
11	massive positive	massive left	transudate	positive left	24

CXR, the other had on the left. Two had small amount of effusion, one had moderate and one had massive effusion.

Discussion

Emerson was the first who describe the diaphragmatic defects (post-mortem) in a patient with hepatic hydrothorax in 1955. These defects can be demonstrated not only grossly, but also microscopically in these patients[17]. These defects may also be visualized using thoracoscopy[13,18,19].

Microscopic examinations of these defects have revealed discontinuities or gaps in the collagen bundles that make up the tendinous portion of the diaphragm. When ascitic fluid collects within the peritoneal cavity, it raises the intra-abdominal pressure and tends to stretch the diaphragm, creating or enlarging these microscopic defects. Then herniation of peritoneum through these gaps into the pleural cavity can be occurred. This leads to the formation of pleuro-peritoneal blebs. These blebs aretypically less than 1 cm. in diameter and tend to rupture, thus providing free communication between the peritoneal and pleural cavities. These blebs tend to occur more commonly in the right hemidiaphragm, may be related to embryonic development, the left hemidiaphragm is more muscular and relatively resistant to bleb formation[6,13]. The fluid in peritoneal cavity will be passed into pleural space via these blebs by negative intrathoracic pressure.

Lieberman FL et al, study 330 patients with cirrhosis and ascites, 6% also had a pleural effusion[1,10]. In another study, Johnston et al reported that 6% of 200 patients with cirrhosis had a pleural effusion but none of 54 without ascites had demonstrable pleural fluid[1,11]. However absence of ascites cannot exclude the cirrhotic etiology of pleural effusion[12].

Regarding the reported case of hepatic hydrothorax, 85% have been right sided, 13% left sided and 2% bilateral[13]. But Alberts WM et al reported bilateral pleural effusion up to 15-30%[14].

The effusion is transudative but the protein content of the pleural effusion is usually slightly higher than that of ascitic fluid since the pleura has a higher absorptive capacity[14].

There are many diagnostic modalities to prove peritoneo-pleural communication included injection of air, contrast agents or radiopharmaceutical in to peritoneal cavity, MRI and surgical exploration by thoracoscopy[20].

We used ^{99m}Tc-MAA peritoneal scintigraphy for evaluation of communication between peritoneal and pleural spaces because it is a safe, rapid, cheap, minimally invasive method and low radiation to the patient. In a case without ascites we performed abdominal tapping under CT guide and introduction of 500 ml. 0.9% NSS in to peritoneal cavity before injection of ^{99m}Tc-MAA 5 mCi. as shown in Figure 1.

The peritoneal scintigraphy has been considered the “gold standard” for identification of hepatic

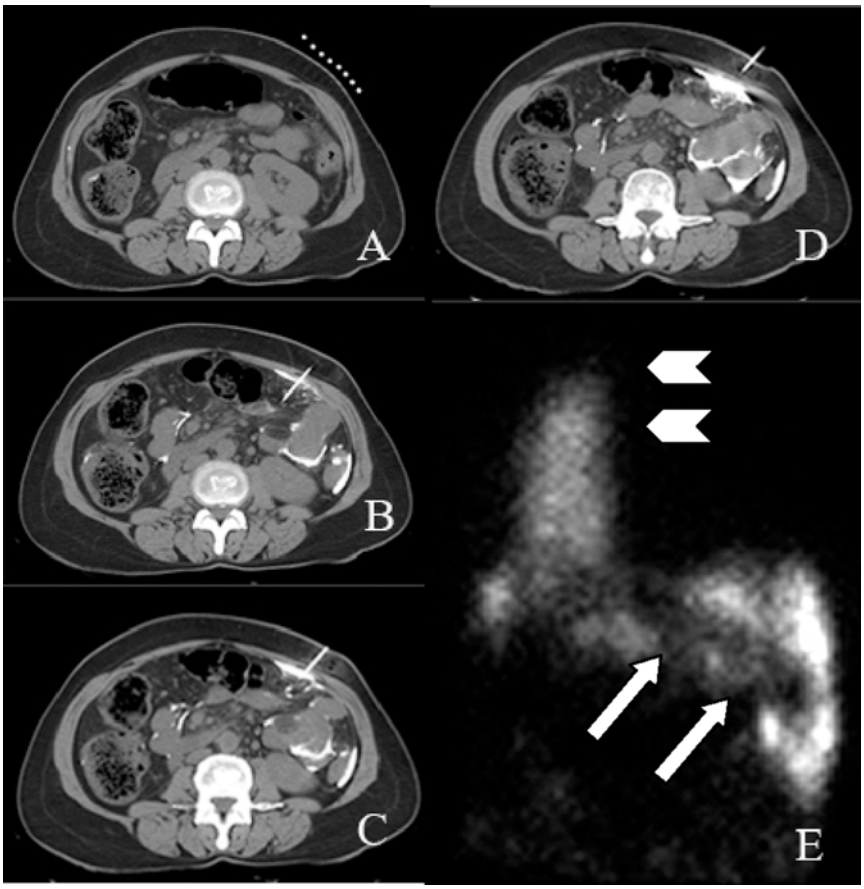

Fig.1 (A-D) CT scan images show CT guided abdominal tapping in a patient without ascites. Small amount of contrast media was injected to test position of the needle and (E) a ^{99m}Tc-MAA peritoneal scintigraphic image shows radiotracer activity in upper abdomen (arrows) and tracer migration into right sided pleural cavity (arrow heads), confirm condition of hepatic hydrothorax.

hydrothorax because of very high specificity (upto 100%) and its sensitivity is about 71%. However its sensitivity can be improved by performing a thoracocentesis before administration of radiopharmaceutical in order to reduce pressure in pleural cavity[6].

Demonstration of peritoneo-pleural communication is importance in patient management because it can prevent unnecessary investigation and cause start correct treatment quickly.

Conclusion

^{99m}Tc-MAA peritoneal scintigraphy can be used for demonstrate peritoneo-pleural communication in cases of hepatic hydrothorax even if in case of absent ascites with fair sensitivity and very good specificity.

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