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Hepatic hydrothorax — complication of end-stage hepatic cirrhosis

Abstract

A 62-year-old woman admitted to the hospital with dyspnoea, elevated body temperature, cough for two days. Patient has been treated for alcoholic hepatic sclerosis for 12 years and hepatitis C infection for 5 years. The chest X-ray revealed hydrothorax of right pleura. Continuous drainage of the right pleura was applied. In spite of intensive treatment effusion excuded 1000 ml/day making pleurodesis impossible, and eventually pleuro-peritoneal shunt was performed. Shunt drainage however turned out to be insufficient to evacuate the pleural fluid. The shunt was removed and continuous pleural drainage was reopen. Due to subsequent disseminated intravascular coagulation, patient was treated with fresh frozen plasma, heparin, blood and platelets. The clinical status alleviated, but patient still required chest tube because of the refractory hydrothorax.

Key words: hydrothorax, cirrhosis, chest tube, pleuro-peritoneal shunt

Introduction

Cirrhosis is defined as the histological development of regenerative nodules surrounded by fibrous bands in response to chronic liver injury, which leads to portal hypertension and end-stage liver disease [1]. Patients with cirrhosis can aquire several pulmonary conditions related to portal hypertension, including hepatopulmonary syndrome, portopulmonary hypertension, spontaneous bacterial empyema, and hepatic hydrothorax [2]. The difficult-to-manage hepatic hydrothorax is typically found in advanced stages of cirrhosis [3].

Case report

A 62-year-old Caucasian woman was admitted to the university hospital in Gdansk with dyspnea, elevated body temperature (39°C) and cough persisting for two days. Patient had been treated for alcoholic hepatic sclerosis for 12 years. During this time she has been deteriorating gradually with several severe exacerbations and oesophageal variceal hemorrhages. Five years after the diagnosis of alcoholic hepatic cirrhosis, hepatitis C infection was diagnosed. In spite of poor prognosis and severe clinical course of the disease patient continued to use alcohol.

Physical examination revealed decreased breath sounds over the lower part of the right lung, crecipitations over the upper part of the right lung, ascites and jaundice, body temperature was 39°C. The chest X-ray showed hydrothorax of the right pleura and pneumonia of the right lung (Figure 1). CT examination of the chest showed large quantity of pleural fluid and pneumonia. Laboratory abnormalities included: elevated bilirubin, liver enzymes, ammonia and increased CRP (Table 1).

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Advances in Palliative Medicine 2008, 7, 29–32

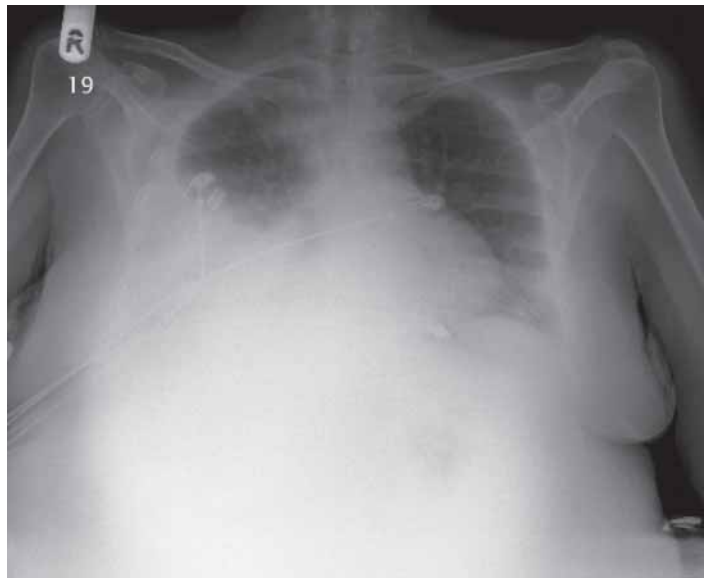

Figure 1. Chest X-ray

Table 1. Laboratory test results

Biochemical screen		**Full blood count**		**Urinalysis**	
Na [mEq/l]	138	HGB [g/dl]	6.9	Color	Yellow
K [mEq/l]	3.1	RBC [T/L]	1,8	Specific gravity [kg/l]	1.010
Glucose post prandial [mg/dl]	438	HCT (%)	19	Glucosa	Neg
Creatinine [mg/dl]	1.59	MCV [fl]	110	Bilirubin	Neg
BUN [mg/dl]	41	MCH [pg]	39	Proteine	Neg
AspAT [U/l]	38	MCHC [g/dl]	35	Red cell	4–10
AlAT [U/l]	18	PLT [G/l]	61	Leukocyte count	Few
Total bilirubin [mg/dl]	3,8	WBC [G/l]	3.74		
GGTP [U/L]	164	Neutrophilic granulocytes (%)	77.4		
LDH [U/L]	356	Lymphocytes (%)	8.8		
Amonia [μmol/l]	137	Monocytes (%)	10,2		
ALP [U/l]	134	Eosinophilic granulocytes (%)	3.2		
Total protein [g/l]	48	Basophilic granulocytes (%)	0.31		
Albumin [g/l]	15				
		CRP	31.9	Coagulation tests	
Capilar blood gases				Protrombin ratio (%)	69
pH	7,41	**CA 125** U/ml	408.5	INR	1.54
pCO_2 [mm Hg]	29,2			APTT [s]	44.8
pO_2 [mm Hg]	54.3			Fibrynogen [g/l]	2.81
HCO_3^- [mmo I/L]	18.2			D-dimers	755
HCO_3 std [mmo I/L]	19.9			AT III %	33.2
BEecf [mmo I/L]	-5.6				
BE (B) [mmo I/L]	-5.3				
SO_2c (%)	89				

The diagnosis was right pleuropneumonia, hepatic cirrhosis, hepatorenal syndrome.

Continuous drainage of the right pleura was applied as well as antibiotics (amoxycycline, metronidazole). Pleural fluid and blood cultures were negative. However *Pseudomonas aeruginosa* was found in sputum thus the antibiotic was changed to ceftazidime. The examination of fluid showed transduate, amylase was normal, CA125 was insignificantly elevated. In spite of intensive treatment effusuion excuded 1000 ml/day making pleurodesis impossible, and eventually the pleuro-peritoneal shunt was performed. Pleuroscopic examination of pleura showed no abnormalities. The postoperative period was complicated with renal, hepatic insufficiency and cardiogenic shock. Shunt drainage turned out to be insufficient to evacuate the pleural fluid, and eventually shunt was removed and the continuous pleural drainage with Heimlich valve was reopen. The bacteriological examination of the pleural fluid revealed *Corynebacterium jejunium*. Treatment with vancomycin and albumin supplementation was introduced. Subsequently disseminated intravascular coagulation (DIC) occurred, and was treated with fresh frozen plasma, heparin, blood and platelets. The clinical status became alleviated, but patient still required with chest tube because of refractory hydrothorax.

Discussion

Liver transplantation remains the only curative option for a selected group of patients, however pharmacological treatments that could halt progression to decompensated cirrhosis are needed [1]. The disease may lead eventualy to liver failure and may become complicated with ascites.

Hepatic hydrothorax is a relatively uncommon complication of end-stage liver insufficiency with an estimated prevalence of 5–10%. The pathophysiology includes the direct movement of ascetic fluid from the peritoneal cavity into the pleural space through diaphragmatic defects [4]. The effusion is found most commonly in the right hemithorax, less frequently on the left side and very rarely — bilaterally [4, 5]. The effusion is usually transudate. Hepatic hydrothorax can accumulate slowly, but on rare occasions patients present with acute tension hydrothorax which can fill the whole pleural cavity [6]. The first-line therapy includes sodium restriction and administration of diuretics (spironolactone up to 400 mg/day and furosemide up to 160 mg/day). Thoracocentesis is a simple and effective procedure indicated in recurrent hydrothorax and for relief of dyspnea. It is recommended to remove no more than 2l at a time because there is a risk of re-expansion pulmonary oedema or hypotension [7]. When thoracocentesis is required every 2–3 weeks alternative strategies should be considered [4]. Chest tube should be avoided as associated with complication such as hypovolemic shock or empyema. Pleurodesis is not routinely recommended as well. Refractory hydrothorax can be managed with transjugular intrahepatic portosystemic shunt (TIPS) in selected cases [4]. Good response to TIPS placement is found in 70–80% of patients. TIPS may help as a bridge to liver transplantation [8]. For those patients that cannot undergo TIPS placement, consideration for pleurodesis or diaphragmatic repair by thoracoscopy should be considered [4].

Our patient cannot undergo liver transplantation and TIPS, so chest tube was recommended for pleurodesis. Although she was treated with diuretics, albumin and pleuro-peritoneal shunt unfortunatelly the talc pleurodesis cannot be perform becouse of persistent drainage of pleural effusion. Every third of all cirrhotic patients with hydrothorax dies with chest tube still in the place. Failure to remove the chest tube increases mortality in patients with increasing severity of liver disease [3].

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