INTRODUCTION
Hepatic hydrothorax is defined as pleural effusion of patients with hepatic cirrhosis and portal hypertension without a primary cardiac, pulmonary or pleural disease. Ascites may also cause abdominal distension that could result in dyspnea, cough, discomfort and pain. On reviewing literature, it is noted that 1% to 20% of patients with hepatic cirrhosis could develop hydrothorax. It usually occurs on the right side of the pleural space and is probably caused because the ascitic liquid passes through due to: a structural defect of the diaphragm, fragile diaphragmatic muscles, a defect in the tendinous part of the diaphragm, 0.03 to 1.2 mm orifices at the tendinous center of the diaphragm, transdiaphragmatic lymphatic migration, hypoalbuminemia, hypertension of the portal vein, hypertension of theazygous vein, hyperaldosteronism, hypertension of the pulmonary veins, intrathoracic lymphatic stasis following hypertension of the thoracic duct and splenomegaly.

*Corresponding Author
Dr. A S Pandey
A103, Teaching Staff quarters, Smimer hospital campus, Sahara darwaja, Surat,
Pin Code: 395010
E mail: aspandey72@yahoo.co.in

CASE REPORT
A 35 year old male patient working as a labourer, an alcoholic for 20 years, tobacco chewer for 15 years, presented with complain of cough with expectoration for 1 month, abdominal distension since 20 days, bilateral lower limb swelling for 15 days and chest pain for 2-3 days. He was previously hospitalized elsewhere four times for ascites and on two occasions pleural aspiration with abdominal paracentesis in last 2 years. Patient presented to us with hydropneumothorax, ascites, cirrhosis and pulmonary Tuberculosis. Patient was managed with ICD, anti TB treatment, diuretics and chemical pleurodesis. After pleurodesis, there was no recurrence of pleural effusion.
Complicated Hepatic Hydrothorax

Figure 1: Pneumothorax

Left sided hydropneumothorax

Partially expanded lung after 7 days of ICD insertion, with thin strip of pneumothorax

The pleural fluid protein was 1.0 gm%, albumin 0.8 gm%, cell count was 260 with 90% lymphocytes. Serum protein was 5.4% and serum albumin was 2.7%. So the serum to pleural fluid albumin gradient was greater than 1.1 and fluid was transudative. Post ICD chest X-ray chest revealed partial expansion of lung with a cavity in left mid zone. Sputum for acid fast bacilli were positive. Pleural fluid for AFB smear and culture was negative. As the patient was having abdominal distension, abdominal paracentesis was done on next day and 1200ml ascitic fluid was drain out. The ascitic fluid protein was 0.4gm%, sugar 148mg%, 10cells/cu.mm with lymphocytic predominance with absence of coagulum. USG abdomen revealed changes of cirrhosis, raised portal vein pressure and moderate ascites. 2D Echo was carried out to rule out cardiac cause but it was normal. Liver function and Complete blood count were normal.

The patient was put on of Cat-I anti tuberculosis treatment, antibiotics and supportive treatment for pulmonary Tuberculosis with left hydropneumothorax. Injection human albumin (20%) was given for 7 days, but the edema did not resolve and the pleural fluid drain was consistently present (500-700 cc) daily for consecutive 15 days. As there was no decrease in the drain output and the pleural fluid was transudative, we thought about the possibility of hepatic hydrothorax. Than we added diuretics in form of aldactone (50 mg 2BD) and furosemide (40 mg 1 BD). After 5 days of adding diuretics drain significantly reduced to 30 ml and the lung expanded. As the patient had recurrent history of ascites, pleural effusions, the fluid was transudative and even the patient well responded to diuretics, we attempted for chemical pleurodesis with oxytetracycline 20 mg per kg.

Figure 2: Expanded lung

Expanded lung with left mid zone cavity after 7 days of diuretic therapy. After 14 days of ICDT

After pleurodesis ICD was removed after 2 days. After 6 months follow up patient has no recurrence of pleural effusion.

DISCUSSION

The patient in the above report presented left pleural effusion, while in most cases it occurs on the right.7,9 There are more chances of complication like empyema, pleural TB, adenocarcinoma and parapneumonic effusion if the hepatic hydrothorax is on left side (65%).12 Our case was complicated with pulmonary TB. The probable mechanism of hepatic hydrothorax is the defect in the tendinous part of the diaphragm. When intra abdominal pressure increases gaps can develop between the muscle fibers of the diaphragm and lead to small herniation of the peritoneum into the pleural space (pleuroperitoneal blebs). These blebs can rupture resulting in passage of fluid to pleural space.6,13 The negative intrathoracic pressure favors the one way transfer of fluids across these defects.6,14-15 In our case, biochemical examination of the pleural fluid showed the presence of a transudate, which was in keeping with the reports in literature.8 The pleural fluid in hepatic hydrothorax has serum to pleural fluid gradient greater than 1.1 as is found in ascites secondary to portal hypertension which was same as in our case.16,17 The polymorphonuclear count is usually less than 500cells per mm³, the total protein concentration is less than 2.5 gm/dl.16,17 The scintigraphic examination of this patient was not done due to unavailability. Pleural drainage was used to rapidly improve the symptoms and discomfort caused by hydropneumothorax.19 If the pleural effusion persists, than some procedures can be adopted such as: thoracic...
drainage, chemical pleurodesis (most frequently used) using tetracycline powder, bleomycin, radioactive phosphorus, gold, traumatic pleurodesis, thoracotomy or thoracoscopy in repairing the diaphragm, pleurectomy, more recently TIPS - Transjugular intrahepatic percutaneous portosystemic shunt (this is a palliative) and an orthotopic liver transplant. There was a decrease in drain output following diuretics and the lung expanded. Chemical pleurodesis with oxytetracycline was carried out to prevent further recurrence. A liver transplant could not be carried out in this case even after treating hydropneumothorax, ascites and malnutrition because the patient is still on alcohol. After 6 months follow up patient had no recurrence of pleural effusion and pulmonary TB was cured.

REFERENCES