# Successful Management of Chylothorax in Cirrhosis of the Liver using Trans-jugular Intrahepatic Portosystemic Shunt (TIPS)

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Chylothorax is an uncommon condition recognized by the accumulation of lymphatic fluid (chyle) which is rich in cholesterol and triglycerides in the pleural cavity. In more than 95% of cases, chylothorax or chylous ascites is secondary to malignancy, trauma, tuberculosis or surgery. In about 1% of cases, chylothorax with or without chylous ascites is secondary to liver cirrhosis.[1,2]

## Case Report

A 64-year old non-alcoholic female was diagnosed to have liver cirrhosis about two years back and was on regular follow up without any ascites or hepatic encephalopathy till a month ago. This time she presented to her family physician with symptoms of increasing breathlessness, abdominal distension and swelling over feet for the last one month. Further evaluation by the family physician revealed tachypnoea with oxygen saturation of 91% on 2 litres per minute of oxygen by nasal prongs. She was found to have a right-sided pleural effusion which was further confirmed by a chest x-ray. The x-ray showed complete opacification of the right hemithorax (**Figure 1**). She also had mild to moderate ascites. She had no history of any trauma or malignancy. She was managed with repeated therapeutic thoracentesis and intra-venous albumin along with diuretics in the last one month. However, each time symptomatic improvement did not last for more than 4-5 days. Further evaluation revealed total serum protein was 8.4 gm/dL, albumin 3.5gm/dL, bilirubin 2.25 gm/dL, and prothrombin time 16.4 sec with control 13.3 sec and INR 1.32. Contrast enhanced computed tomography (CECT) of the abdomen revealed shrunken nodular liver, splenomegaly and moderate ascites which were suggestive of cirrhosis with portal hypertension. CECT of the chest revealed massive right-sided hydrothorax. The pleural fluid analysis revealed a total white blood cell count of 40/cu cm (polymorphs 10% lymphocytes 90 %), albumin 2.6gm %, SAAG (serum ascitic fluid albumin gradient): 0.9, sugar 209 mg%, fluid ADA (Adenosine deaminase) 5 U/L, fluid lactate dehydrogenase (LDH) 105 mg% and fluid triglycerides 690 mg%, Fluid acid fast bacilli (AFB) staining was negative and culture showed no growth. The cytological examination did not show any malignant cells. In view of recurrent rapid filling of fluid in the right pleural cavity, there was suspicion of a malignant pleural effusion, and therefore a pleural biopsy was done. However, it was unremarkable. Her ascitic

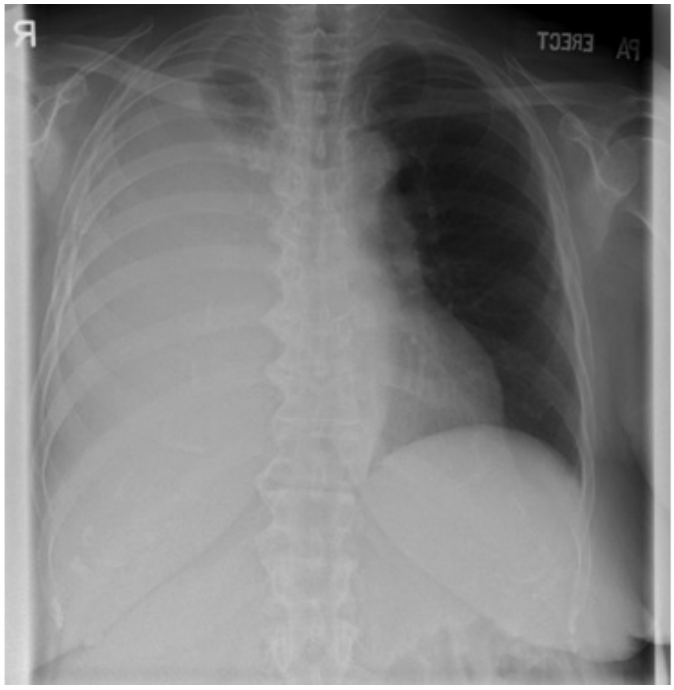

**Figure 1: Initial Chest x-ray showing massive right pleural effusion.**

fluid examination was similar to the pleural fluid and was suggestive of chylous ascites. Her aetiological workup was already done during previous visits to the hospital. Her workup for hepatitis B, hepatitis C, autoimmune hepatitis and Wilson's disease was negative. 2D Echo revealed left ventricular (LV) ejection fraction of 77% with grade I diastolic dysfunction. Gastroscopy revealed nonbleeding small oesophageal varices and portal hypertensive gastropathy.She had no prior episodes of hepatic encephalopathy with Model for End-stage Liver Disease (MELD) score of 13. She was started with a salt-restricted diet, diuretics, intravenous albumin infusion. She could not tolerate prolonged increases in her diuretics due to worsening renal function that normalized on withdrawal of said diuretics. The maximum tolerated doses of diuretics were spironolactone 100 mg and furosemide 40 mg per day. Given repeated refilling following therapeutic thoracentesis leading to breathlessness with underlying cirrhosis of the liver with portal hypertension, treatment options were limited to either intercostal drainage with pleurodesis or trans-jugular intrahepatic portosystemic shunt (TIPS). The patient family members opted for TIPS. Post-TIPS her hepatic venous pressure gradient (HVPG) reduced from 14 mmHg to 9 mmHg (**Figure 2**). Despite the decrease in portal pressure over the next seven days, there was no significant reduction inright-sidedchylothorax, and the patient continued to have increasing breathlessness. Hence intercostal catheter drainage (ICD) was done. She started draining 1 to 1.5 L white milky appearing pleural fluid daily. She was placed on total parenteral nutrition (TPN) with no oral intake along with subcutaneous injection octreotide. In the next seven days, pleural effusion and ascites resolved completely allowing discontinuation of diuretics over the next few days. She did not develop any signs of hepatic encephalopathy. She was discharged on a salt-restricted high protein diet and supportive treatment to prevent hepatic encephalopathy. For the next two years, she remained encephalopathy free without any requirement for diuretics and was able to carry on all her routine activities.

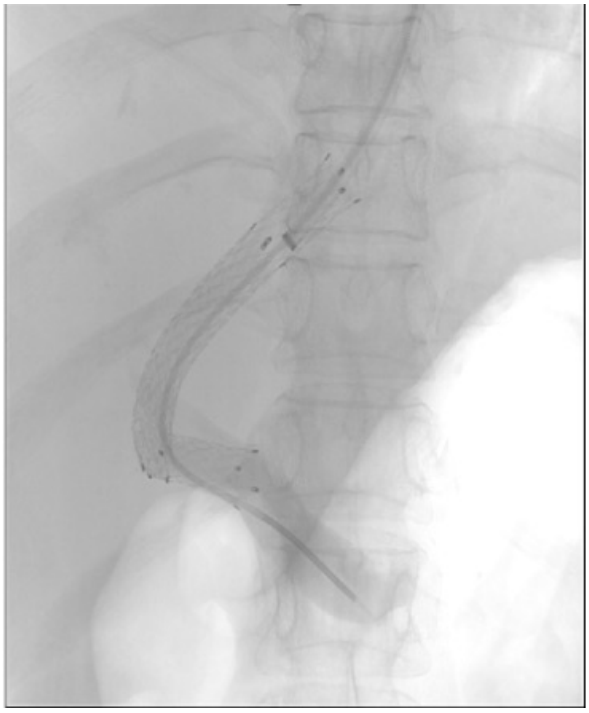

**Figure 2: TIPS, The stent is seen bridging hepatic vein to portal vein.**

## Discussion

A majority of the patients with chylothorax are treated conservatively unless the patient has shortness of breath due to fluid in the pleural space. Most of these patients are treated with intermittent thoracentesis for symptomatic relief of the dyspnoea. It has been seen that patients developing chylothorax after surgery or trauma respond to complete bowel rest and total parenteral nutrition with octreotide. Those patients who do not respond to conservative treatment are then managed with surgical procedures such as pleurodesis or ligation/embolization of the thoracic duct. The success of chemical pleurodesis depends on adequate drainage of the lymphatic fluid from the pleural space before the instillation of the talc. The surgical approach is successful in patients having chylothorax secondary to trauma or tumours.[3] It is believed that chylothorax in cirrhosis is secondary to portal hypertension, which leads to increased formation of hepatic lymph. The increased lymph formation ultimately leads to increased pressure in the central lymphatic vessels, which leads to spontaneous rupture of lymphatic channels and accumulation of chyle in ascitic fluid. Because of the negative intrathoracic pressures, the chylous ascitic fluid migrates into the pleural cavity through a defect in the diaphragm leading to chylothorax.[4] Surgical procedures done for treatment of chylothorax secondary to trauma or tumours such as ligation, disruption or embolization of

**Table 1: Summary of four patients treated with TIPS for chylothorax and chylous ascites by Kikolski SG *et al*[2].**

| Etiology of Chylothorax/ Chylous Ascites | Triglyceride level (mg/dl) in pleural fluid | Triglyceride level(mg/dl) in ascitic fluid | PPG before TIPS mm/Hg | PPG after TIPS mm/Hg | Improvement in Chylothorax/ Chylous ascites | Follow up (mo) |
|---|---|---|---|---|---|---|
| Cirrhosis | 495 | 585 | 16 | 11 | Improved | 4.1 |
| Cirrhosis | 258 | 271 | 16 | 9 | Improved | 16.4 |
| Cirrhosis | 213 | - | 12 | 3 | Improved | 0.6 |
| Cirrhosis | 154 | 186 | 27 | 8 | Improved | 6 |

thoracic duct do not address portal-hypertension, which is believed to be one of the underlying causes in patients of chylothorax with or without chylous ascites and liver cirrhosis. In these patients, ascites and TIPS may be more effective as it decreases post sinusoidal and portal pressure significantly, thereby reducing the hepatic lymph formation. In a retrospective study of chylothorax with chylous ascites in patients with cirrhosis of the liver, who were treated by TIPS, there was a significant decrease in portosystemic pressure gradient after TIPS (**Table 1**)[2]. This decrease in gradient leads to a reduction of hepatic and gastrointestinal lymph flow, which decreases pressure in lymphatic channels, thereby improving chylothorax and chylous ascites. Recently a case report has described that TIPS has been successful in controlling chylothorax completely.[5] It is possible that in our patient, repeated refilling after thoracentesis was due to a spontaneous leak which might have persisted because of portal hypertension.

In summary, this case highlights the growing evidence about massive chylothorax in patients with cirrhosis of the liver with portal hypertension. It is suspected to be due to increase lymph formation secondary to an increase in portosystemic pressure gradient. It further suggests that TIPS may be an effective treatment option in such patients with other supportive treatments like intercostal drainage, octreotide and TPN, which help in reducing lymph formation, with excellent long term outcomes.

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# Irreducible Rectal Prolapse in a Patient with Anorectal Malformation: Arduous to Reduce


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