HEPATIC HYDROTHORAX
WITH MINIMAL ASCITES

INTRODUCTION

Hepatic hydrothorax is defined as a significant pleural effusion (usually more than 500 ml) that develops in a patient with liver cirrhosis and portal hypertension, in the absence of cardiopulmonary disease. It is right sided in most cases and develops in 6 to 10% of patients with end-stage liver disease. It is commonly seen in conjunction with ascites. Very rarely, it can present in the absence of ascites. We report a case of recurrent right-sided pleural effusion in a known case of cirrhosis and with no clinical evidence of ascites. A comprehensive review of this presentation of hepatic hydrothorax is included.

ABSTRACT

This is the case of a 70-year old male, who was a chronic alcohol drinker and was admitted several times due to complications of liver cirrhosis. He was admitted due to non-productive cough, worsening dyspnea and shortness of breath. Physical examination revealed a patient who was not jaundiced, with decreased breath sounds and dullness to percussion on the right lower lung fields, with normal cardiac findings and abdominal findings significant of the absence of clinical signs of portal hypertension and ascites. A chest radiograph done revealed presence of massive right pleural effusion for which the patient underwent thoracentesis. Due to rapid re-accumulation of pleural fluid, patient underwent CTT insertion and VATS. Unfortunately, despite maximum medical management, the patient developed complications and succumb to the disease.

CASE PRESENTATION

A 70-year old male was admitted with a 3–week history of non-productive cough, worsening dyspnea and shortness of breath associated with fever. The patient went for consult to a private hospital where a chest radiograph was done which revealed
massive pleural effusion on the right. A thoracentesis was done and he was immediately transferred to another institution for further management. Past medical history is significant of a patient who was a chronic alcohol drinker for at least 20 years, was diagnosed to have liver cirrhosis with esophageal varices and underwent rubber band ligation in 2006. In April of last year, he had an upper gastrointestinal bleeding from bleeding esophageal varices. About 3 months after, he presented with massive ascites and underwent paracentesis. A month prior to the admission, he was admitted due to hepatic encephalopathy and was managed accordingly. On that time, the patient was already candidate for liver transplantation. The family history was unremarkable. Aside from being a chronic alcohol drinker, he was also a chronic smoker of 40-pack years.

Physical examination showed a blood pressure of 130/80mmHg, heart rate of 92 beats per minute, respiratory rate of 26 breaths per minute and temperature of 36.8 °C. The patient was not jaundiced, sclerae were anicteric and there was no jugular venous distension. Chest examination revealed a right chest lag, decreased breath sounds, dullness to percussion, decreased vocal & tactile fremiti on the right lower lung fields. Cardiac examination was unremarkable. Abdominal examination showed a non-distended abdomen with normo-active bowel sounds, no fluid wave or shifting dullness noted. No clinical signs of portal hypertension such as caput medusae, spider angioma and splenomegaly were present. There was no pedal edema or clubbing.

The initial chest x-ray showed a homogenous opacification of right hemithorax with contralateral shift of the mediastinum. Initial chemistry revealed prothrombin activity of 66% with International normalized ratio (INR) of 1.33, SGOT of 64.0 U/L, SGPT of 62.0 U/L, total protein of 63.4 g/L, albumin of 20.0 g/L, globulin of 43.4 g/L and A/G ratio of 0.5. Complete blood count showed anemia with platelet count of 156. Renal function was within normal limits with blood urea nitrogen of 5.8 mmol/L and creatinine of 111 umol/L. A cardiology work up revealed normal left ventricular dimensions with preserved systolic function (ejection fraction of 78%) and mild aortic stenosis on echocardiogram.
An ultrasound of right hemithorax revealed free-flowing fluid with estimated amount of 1500 ml and passive atelectasis of right lower lung. The patient underwent thoracentesis with return of 1.8 liters of yellow-cloudy fluid, which on analysis revealed sugar of 6.40 mmol/L, protein of 20 g/L, LDH of 224 u/L, RBC of 123x10^6 /L, WBC of 29x10^6 /L with differential count of 11% polymorphonuclear cells with lymphocytic predominance of 89%. Bacterial and fungal cultures of the pleural fluid were negative, as was the cytology and TB-PCR. Pleural fluid analysis also showed cholesterol of 0.20 mmol/L and triglyceride of 1.58 mmol/L. The pleural fluid to serum protein ratio of 0.315 and pleural fluid to serum LDH ratio of 0.366 points out to a transudative type of effusion. A repeat chest x-ray done revealed a significant reduction in the pleural fluid on the right (Figure 1). However, 2 days later, the patient became severely dyspneic again. A repeat chest radiograph was done which showed rapid re-accumulation of right sided pleural effusion (Figure 2). He was referred to TCVS service for CTT insertion and with plans for VATS and pleurodesis. A follow up chest radiograph revealed an interval decrease of the right-sided pleural effusion (Figure 3). Pleural fluid analysis still showed a transudative type of effusion.

Figure 1. Chest radiograph (PA view) showed significant reduction of the right-sided pleural effusion and cardiomegaly.

Figure 2. Chest radiograph (AP view) showed re-accumulation of right-sided pleural effusion.
On the 7th hospital day, a high resolution CT scan of the chest done revealed bilateral upper and lower lobe fibrosis with emphysematous changes, left lower lobe calcific granuloma and cardiomegaly. There was an incidental note of liver cirrhosis with signs of portal hypertension, splenomegaly and abdominal ascites (Figure 4).

Figure 3. Chest radiograph (AP) showed an interval decrease of the right-sided pleural effusion.

A Video-assisted thoracoscopy was done which showed smooth visceral and parietal pleura and the diaphragm showed no visible underlying diaphragmatic defects. A post VATS x-ray showed resolution of effusion and re-expansion of the lungs (Figure 5). Pleural biopsy result revealed presence chronic inflammation & hemorrhage and it was negative for malignancy and TB-PCR. The pleural effusion later developed multiloculations and required a separate pleural catheter insertion in a major locule.

Figure 4. High-resolution CT of the chest showed bilateral upper and lower lobe fibrosis with emphysematous changes, left lower lobe calcific granuloma and cardiomegaly. An incidental note of liver cirrhosis with signs of portal hypertension, splenomegaly and abdominal ascites.

Despite VATS and mechanical pleurodesis, there was still persistent significant pleural fluid output of 900 – 1400 ml/day. Peritoneal scintigraphy was considered but was deferred because of technical difficulty and high complication risk for the patient.

The patient developed complications including hospital-acquired pneumonia and secondary
empyema which were managed & treated accordingly. This resulted to subsequent resolution of the empyema, reduction of pleural fluid output and eventual CTT and pleural catheter removal.

Throughout the course in the hospital, medical management was maximized and this included sodium restriction, the use of diuretics, albumin-furosemide infusion and paracentesis. However, the patient developed hepatic encephalopathy with seizures and succumb to the disease.

DISCUSSION

Hepatic hydrothorax by definition is a transudative pleural effusion associated with portal hypertension without cardiopulmonary and pleural disease. Portal HTN is a requirement for its evolution and presence of 500cc of fluid in the pleural spa is included in the definition by many authors. It is uncommon. The incidence varies depending on the method used in detecting pleural effusion. According to an evidence based review by Singh et.al. of hepatic hydrothorax, CXR can detect 4-6% of these pleural effusions and, in a series of 862 patients with cirrhosis in china, where ultrasound was used, incidence was found to be ~15%. Hepatic hydrothorax accounts for 2-3% of all pleural effusion in this study. In the review by Camron et.al. in 2008, it was reported that effusion may affect one or both of the hemithoraces but largely affects the right hemithorax.

The underlying mechanisms of this entity is similar to those leading to other forms of fluid accumulation in cirrhotic patients. Several mechanisms have been proposed by several case
reports and series. The most popular and most accepted mechanism is the so-called porous diaphragm syndrome with direct shift of ascitic fluid from the peritoneal cavity to the pleural space. Raised azygous venous pressure with plasma leakage across transdiaphragmatic lymphatics and alteration in splanchnic and renal circulation abnormalities leading to water retention, are the other possible process leading to hepatic hydrothorax.

Historically, hypoalbuminemia resulting in decreased oncotic pressure was thought to be the primary mechanism involved in these cases. However recent case reports and scientific reviews acknowledge hypoalbuminemia as a contributory but not the primary factor in the evolution of hepatic hydrothorax. The pressure gradient between the peritoneal and pleural cavities is altered in patients with ascites mainly due to the increased intraabdominal pressure, and diaphragmatic thinning due to malnutrition and marked abdominal distention from massive ascites. This mechanism is particularly evident in patients with considerable and significant clinical ascites.

However, hepatic hydrothorax can be a consequence of chronic liver disease or cirrhosis even without or with minimal ascites. The exact mechanism in its evolution is not well-understood, although it is believed to be similar to the development of hepatic hydrothorax in patients with overt clinical ascites. In these cases however, virtually all of the ascitic fluid rapidly crosses the diaphragm to the pleural space. A transudative pleural fluid is required with demonstration of diaphragmatic defects when possible. The absence of ascites should not prevent the inclusion of cirrhosis as a differential diagnosis.

A thorough reinvestigation of the history and a comprehensive physical examination are necessary. These patients may preset with worsening symptoms related to the pleural but clinical presentation is dominated by signs and symptoms of liver decompensation the severity of the pulmonary symptoms depend on the volume of the accumulated fluid. Although, some patients may be asymptomatic, in whom pleural effusion can be an incidental finding. Diagnosis of hepatic hydrothorax is usually suspected in patients with advanced
cirrhosis or chronic liver disease and unilateral pleural effusion when presented with a case of recurrent pleural effusion, as in our patient, the patient should be reanalyzed.

Additional diagnostic modalities that may be helpful in ruling in or out hepatic hydrothorax include, CT scan of the chest to rule out pleural or parenchymal-based pathology not evident on a plain chest radiograph. Bronchoscopy is rarely helpful in these cases unless an endobronchial pathology is suspected. Pleural biopsy is of greatest value in diagnosing granulomatous and malignant diseases. Scintigraphic studies using injection of Tc human albumin or Tc sulphur colloid is the best modality to confirm the existence of a communication between the pleura and the peritoneal space. This involves the migration of radioisotopes into the pleural cavity and is most useful in the absence of ascites. Thoracoscopy is an alternative diagnostic option for direct visualization of the diaphragm for defects. The findings might be considered in decisions for surgical treatment. Direct demonstration of these abnormalities with non invasive techniques is extremely rare. MRI was however used successfully in one case report by Zenda et.al., showing hypointense jet flow on both T1 and T2 weighted sagittal scans color Doppler ultrasonography was used in another case study. It is considered as the easiest method and has benefits of real time diagnosis. The communication was marked by jet flow from the peritoneal to the pleural space. The defects are due to discontinuities in the collagen bundles of the tendinous portion of the diaphragm, which occur mostly on the right side because of the close proximity of the liver with the diaphragm. It should be stressed however, that although the diagnosis may require exclusion of alternative diagnosis in some cases, demonstration of a peritoneal-pleural communication is not necessary, unless a surgical repair is contemplated.

The main goal of treatment is relief of symptoms and prevention of complications. Unfortunately no large clinical trial or study has been done on this exceptional manifestation of liver decompensation. A report done by Umino et.al. in 2004 reviewed 25 cases from the 1969 to 2003. It was observed that 89% of the patients had serum
albumin levels of less than 3.5 mg/dl and a total bilirubin levels within the range of 0.5 to 13.2 mg/dl. Etiologies of cirrhosis were found to be heterogenous. Treatment strategies included diuretics, medical and surgical pleurodesis, diuretics with medical pleurodesis or TIPS. 48% improved with diuretics alone or in combination with medical pleurodesis and TIPS. 40% died with similar treatment strategies, although half of the patients in this group were not given diuretics.

The same principles for the management of ascites are applicable to hepatic hydrothorax. Most authors agree that the Principles of management should tackle 4 goal one is, to reduce the formation of ascites, to prevent the transfer of ascitic fluid across the diaphragm, to drain the pleural space and to obliterate the pleural space. Initial treatment of choice should be diuretics and strict Na restriction similar to ascites. However, medical treatment has proved to be effective in only a third of the patients with hepatic hydrothorax but must be maximized if tolerated.

Thoracentesis serves to be the most effective procedure in reducing large effusions. It is indicated for the immediate relief of symptoms of dyspnea. Repeated thoracentesis may be an option for patients responding to medical management and with slow fluid re-accumulation. A combination of therapeutic thoracentesis followed by diuresis may lead to complete resolution of the fluid accumulation. If thoracentesis is required every 2-3 weeks despite maximal medical tx, procedure related complications increase. Paracentesis in patients with significant ascites can provide some relief of dyspnea by significantly increasing TLC and FRC. for rapidly accumulating HH, such as in our patient, one might consider the insertion of a chest tube. CTT however in many studies has been associated with high rates of complications that contribute to mortality and secondary empyema. A new modality is insertion of a small bore tunneled pleural catheter like the pleurX. This can be a safe and effective minimally invasive tool for the management of hepatic hydrothorax, and can be done on an outpatient basis with little complications. Video assisted thoracoscopy is best done alongside pleurodesis. Immediate efficacy is found
to be 47%, increasing to 60% if suturing of defects is done. VATS has diagnostic and therapeutic indications and allows direct visualization of the pleura and diaphragm to look for defects, and to do direct biopsies of abnormal pleura. Pleurodesis is a least aggressive technique for patients with poor general condition to undergo more invasive procedures. Various techniques have been described, with varying success rates. However failure rate can be as high as 33%. This may be due to high rate of reaccumulation of the pleural fluid. NIPPV is another therapeutic option and was used effectively in one case study by Takahashi et al. this addresses the shift of fluid from the peritoneum by converting the negative intrathoracic pressure to more positive, thus, halting the unidirectional flow of ascitic fluid to the pleural cavity.

Transjugular intrahepatic portosystemic shunt or TIPS decompresses the portal system artificially by placing a metallic stent under fluoroscopy. In one study, the overall favorable response was as high as 73%. It serves as bridging therapy of patients for liver transplantation. The major procedural complication is hemoperitoneum and the most frequent early complication was the development of new or worsening hepatic encephalopathy. Liver transplantation is the treatment of choice for refractory hepatic hydrothorax with poor liver function and after an SBEM episode. The challenge is to determine the appropriate treatment to bridge patients to liver transplantation when TIPS is not a good option.

In summary, hepatic hydrothorax should be suspected in cases of pleural effusion in the presence of chronic liver disease. Early diagnosis is critical and thorough evaluation is key. Complications increases mortality and hepatic hydrothorax without ascites or with minimal ascites like in our patient is possible. Lastly, therapeutic options are meant to alleviate symptoms, improve the quality of life, prevent complications and most importantly, bridge patients successfully and safely to liver transplantation.
CONCLUSION

In summary, hepatic hydrothorax should be suspected in cases of pleural effusion in the presence of chronic liver disease. Early diagnosis is critical and thorough evaluation is key. Complications increase mortality and hepatic hydrothorax without ascites or with minimal ascites like in our patient is possible! Lastly, therapeutic options are meant to alleviate symptoms, improve the quality of life, prevent complications and most importantly, bridge patients successfully and safely to liver transplantation.

References:

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Good evening, Dr Bautista has presented a difficult case of pleural effusion in a patient with liver cirrhosis.

Hepatic hydrothorax by definition is a transudative pleural effusion associated with portal hypertension without cardiopulmonary and pleural disease. Portal HTN is a requirement for its evolution and presence of 500cc of fluid in the pleural spa is included in the definition by many authors. It is uncommon. The incidence varies depending on the method used in detecting pleural effusion. According to an evidence based review by singhetal of hepatic hydrothorax, CXR can detect 4-6% of these pleural effusions and, in a series of 862 patients with cirrhosis in china, where ultrasound was used, incidence was found to be ~15%. Hepatic hydrothorax accounts for 2-3% of all pleural effusion in this study. In the review by Camronetal in 2008, it was reported that effusion may affect one or both of the hemithoraces but largely affects the right hemithorax. Why do patients with cirrhosis develop pleural effusion? The
underlying mechanisms of this entity is similar to those leading to other forms of fluid accumulation in cirrhotic patients. Several mechanisms have been proposed by several case reports and series. The most popular and most accepted mechanism is the so-called porous diaphragm syndrome with direct shift of ascitic fluid from the peritoneal cavity to the pleural space. Raised azygous venous pressure with plasma leakage across transdiaphragmatic lymphatics and alteration in splanchnic and renal circulation abnormalities leading to water retention, are the other possible processes leading to hepatic hydrothorax. Historically, hypoalbuminemia resulting in decreased oncotic pressure was thought to be the primary mechanism involved in these cases. However, recent case reports and scientific reviews acknowledge hypoalbuminemia as a contributory but not the primary factor in the evolution of hepatic hydrothorax. But how does this exactly happen? The innate negative intrathoracic pressure coupled with the positive intrabdominal pressure creates a pressure gradient that allows the unidirectional flow of ascitic fluid across the diaphragm to the pleural cavity. The pressure gradient between the peritoneal and pleural cavities is altered in patients with ascites mainly due to the increased intraabdominal pressure, and diaphragmatic thinning due to malnutrition and marked
abdominal distention from massive ascites. This mechanism is particularly evident in patients with considerable and significant clinical ascites. However, hepatic hydrothorax can be a consequence of chronic liver disease or cirrhosis even without ascites! Minuscule amount of ascites or even the absence of it is rare and provides diagnostic confusion. It is a manifestation of decompensated chronic liver disease, similar to the advent of ascites, hepatic encephalopathy or variceal hemorrhage. The exact mechanism in its evolution is not well-understood, although it is believed to be similar to the development of hepatic hydrothorax in patients with overt clinical ascites. In these cases however, virtually all of the ascitic fluid rapidly crosses the diaphragm to the pleural space. Pleural effusion in the absence of ascites or with minimal ascites is commonly associated with cardiopulmonary, tuberculous, malignant, infectious or renal cause, and thus these need to be ruled out.

Conversely, hepatic hydrothorax may be the only pointer to an underlying cirrhosis. To reiterate, diagnosing hepatic hydrothorax in this subset of patients, all cardiopulmonary or renal cause must be excluded. A transudative pleural fluid is required with demonstration of diaphragmatic defects when possible. The absence of ascites should not prevent the inclusion of cirrhosis as a differential diagnosis. A life-
threatening but an underdiagnosed pleural complication of cirrhosis is Spontaneous bacterial empyema or SBEM. It is distinct consequence of hepatic hydrothorax involving spontaneous infection of the pleural fluid Chih yen-tuetalin 2012 hypothesized that SBEM may be due to either one of 2 process: one is the spontaneous bacteremia leading to empyema and the 2\textsuperscript{nd} is that SBEM is a complication of SBP when infected abdominal fluid transfers from the abdomen to the pleural space. Paracentesis and thoracentesis should be performed when an infection is suspected. Pleural infection in SBEM is usually monomicrobial with E.coli and K. pneumoniae as the most common etiologic agents.

Diagnosis of SBEM is based on the following: a positive culture and a PMN count of more than 250 cells/mm if however, culture comes out negative, the PMN count should be more than 500 cells/mm there should be no evidence of pneumonia and a transudative pleural effusion in the presence of infection may be diagnostic. Initial antibiotic course is usually started without delay and 3\textsuperscript{rd} generation cephalosporin is the antibiotic of choice, similar to SBP. In our patient, the major problem was symptomatic recurrent pleural effusion despite insignificant ascites! How do we evaluate and manage these patients?We have performed an extensive review of the literature and as far as we are aware,
There are no clinical trial on this subject and the clinical utility of diagnostic and therapeutic options is mainly based on case reports, case series and small cohorts. We did find a systematic review published on April this year, in respiration, and I will be citing much of its results, but please note that the best level of evidence is based on pooling of results of case series. First let me tackle the evaluation hepatic hydrothorax. A thorough reinvestigation of the history and a comprehensive physical examination are necessary. These patients may present with worsening symptoms related to the pleural but clinical presentation is dominated by signs and symptoms of liver decompensation. The severity of the pulmonary symptoms depend on the volume of the accumulated fluid. Although, some patients may be asymptomatic, in whom pleural effusion can be an incidental finding. Diagnosis of hepatic hydrothorax is usually suspected in patients with advanced cirrhosis or chronic liver disease and unilateral pleural effusion when presented with a case of recurrent pleural effusion, as in our patient, the patient should be reanalyzed. Some pointers to note are the following: transudative pleural fluids from patients receiving diuretics may have an elevated protein level and can be mistakenly classified as an exudate; proBNP levels in the pleural fluid may be useful in diagnosing a congestive heart failure etiology especially with levels higher than
1500pg/ml. echocardiography should be done also to rule in or out a cardiac pathology. The British thoracic society guidelines for the investigation of unilateral effusion, recommend doing a 2\textsuperscript{nd} thoracentesis, if the first tap is non-diagnostic, to increase the diagnostic yield. The following is the diagnostic algorithm recommended in the evaluation of HH according to the recent evidence based review. Aside from the basic battery of tests and exclusion of other causes, thoracentesis is done and analysed.

The pleural fluid is classified as either exudative or transudative using the light’s criteria. Absolute neutrophil count is determines the diagnosis of HH or SBEM as mentioned earlier. Typical pleural fluid features of uncomplicated hepatic hydrothorax as shown in this slide are the usual transudative finding based on light’s criteria. On the other hand, if the fluid seems to suggest an exudative character, but HH is still highly entertained, pleural fluid gradient of $>1.1\text{g/dl}$ will point to the latter. It should be noted that in patients with cirrhosis, 18% of patients can have an alternative diagnosis. (click) Still, despite extensive work-up, the cause of 15% to 20% of all pleural effusions will remain unknown.

What other diagnostics tests can be done?
Imaging such as CT scan may be useful in ruling out pleural or parenchymal-based pathology not evident on a plain chest radiograph.

Bronchoscopy is rarely helpful in these case unless an endobronchial pathology is suspected.

Pleural biopsy is of greatest value in diagnosing granulomatous and malignant diseases. Scintigraphic studies using injection of Tc human albumin or Tc sulphur colloid is the best modality to confirm the existence of a communication between the pleura and the peritoneal space. This involves the migration of radioisotopes into the pleural cavity and is most useful in the absence of ascites. Thoracoscopy is an alternative diagnostic option for direct visualization of the diaphragm for defects. The findings might be considered in decisions for surgical treatment.

Diaphragmatic defects are classified under one of four types. Type I shows no obvious defects, type II defects shows blebs on the surface, type III shows broken defects with fenestrations and numerous bead-like rough surface, multiple gaps are seen in type IV.

Direct demonstration of these abnormalities with non invasive techniques is extremely rare. MRI was however used successfully in one case report by Zenda et al., showing
hypointense jet flow on both T1 and T2 weighted sagittal scans color Doppler ultrasonography was used in another case study. It is considered as the easiest method and has benefits of real time diagnosis. The communication was marked by jet flow from the peritoneal to the pleural space. The defects are due to discontinuities in the collagen bundles of the tendinous portion of the diaphragm, which occur mostly on the right side because of the close proximity of the liver with the diaphragm. It should be stressed however, that although the diagnosis may require exclusion of alternative diagnosis in some cases, demonstration of a peritoneal-pleural communication is not necessary, unless a surgical repair is contemplated.

After discussion of diagnostic tests, allow me now to talk about management.

The main goal of treatment is relief of symptoms and prevention of complications. Unfortunately no large clinical trial or study has been done on this exceptional manifestation of liver decompensation. However, from our extensive research, we have unearthed a literature review by Umino et al in 2004. Allow me to breakdown and summarize this busy slide for you! This review included 25 cases from years 1969 to 2003, mean age is 53yo with the youngest patient at 29 years old. The 25 patients comprised
of 13 females and 12 males. It was observed that 89% of the patients had serum albumin levels of less than 3.5 mg/dl and a total bilirubin levels within the range of 0.5 to 13.2 mg/dl. Etiologies of cirrhosis were found to be heterogenous. Treatment strategies included diuretics, medical and surgical pleurodesis, diuretics with medical pleurodesis or TIPS. 48% improved with diuretics alone or in combination with medical pleurodesis and TIPS. 40% died with similar treatment strategies, although half of the patients in this group were not given diuretics. It is necessary that patients be evaluated for liver transplantation. The same principles for the management of ascites are applicable to hepatic hydrothorax. Most authors agree that the Principles of management should tackle 4 goals: one is, to reduce the formation of ascites, to prevent the transfer of ascitic fluid across the diaphragm, to drain the pleural space and to obliterate the pleural space. This simple algorithm published in seminar of respiratory and critical medicine last year, summarizes the treatment of hepatic hydrothorax. Let me run you through the different modalities. Initial tx of choice should be diuretics and strict Na restriction similar to ascites. However, medical treatment has proved to be effective in only a third of the patients with hepatic hydrothorax but must be maximized if tolerated. We will learn more about this thru our expert reactor.
Thoracentesis serves to be the most effective procedure in reducing large effusions. It is indicated for the immediate relief of symptoms of dyspnea. Repeated thoracentesis may be an option for patients responding to medical management and with slow fluid reaccumulation. A combination of therapeutic thoracentesis followed by diuresis may lead to complete resolution of the fluid accumulation. If thoracentesis is required every 2-3 weeks despite maximal medical tx, procedure related complications increase.

The next modality is paracentesis. Paracentesis in patients with significant ascites can provide some relief of dyspnea by significantly increasing TLC and FRC. For rapidly accumulating HH, such as in our patient, one might consider the insertion of a chest tube. In one case series, successful resolution was noted in the majority of patients, at least in the short term. It is often done when VATS and pleurodesis are contemplated. CTT however in many studies has been associated with high rates of cx that contribute to mortality and secondary empyema. Regulation of fluid drainage is crucial to avoid rapid shifts in pressures and self-perpetuating PE. Most authors recommend avoiding this when possible. A new modality is insertion of a small bore tunneled pleural catheter like the pleurx. This can be a safe and effective
minimally invasive tool for the management of hepatic hydrothorax, and can be done on an outpatient basis with little complications. The next modality is Video assisted thoracoscopy is best done alongside pleurodesis. Immediate efficacy is 47% increasing to 60% if suturing of defects is done. VATS Has diagnostic and therapeutic indications and Allow direct visualization of the pleura and diaphragm to look for defects, and to do direct biopsies of abnormal pleura. It is usually done alongside mechanical/chemical pleurodesis.

Suturing of the diaphragmatic defects is also possible. Pleurodesis is a least aggressive technique for patients with poor general condition to undergo more invasive procedures. Various techniques have been described, with varying success rates. However, failure rate can be as high as 33%. This may be due to high rate of reaccumulation of the pleural fluid. In one study Somatostatin can be combined with pleurodesis to decrease the portosystemic gradient, thereby decreasing reaccumulation of pleural fluid. Various have been tried with most studies employing talc. Average of chest tube placement with talc pleurodesis was 12.6 days. Many patients require to attempts. There is one study that compares VATs + talc and CTT + talc showing higher success rate in the former. Another study also showed higher
success rate in combined mechanical and chemical pleurodesis.

NIPPV is another therapeutic option and was used effectively in one case study by Takahashi et al. This addresses the shift of fluid from the peritoneum by converting the negative intrathoracic pressure to more positive, thus, halting the unidirectional flow of ascitic fluid to the pleural cavity. Transjugular intrahepatic portosystemic shunt or TIPS decompresses the portal system artificially by placing a metallic stent under fluoroscopy. In one study, the overall favorable response was as high as 73%. It serves as bridging therapy of patients for liver transplantation. The major procedural complication is hemoperitoneum and the most frequent early complication was the development of new or worsening hepatic encephalopathy. Allow me to break down this slide, from the study done by Singh et al. This review included 8 studies from 1994-2010, with a total of 332 patients. The overall response rate was observed to be as high as 73%, hepatic encephalopathy happened in 26.7%. The 30-day mortality is 18.6%, and a 1-year survival rate of 52.3%. TIPS can be effective in with less severe liver disease based on clinical scoring such as MELD and CTP, in young patients and is an effective treatment for refractory Hepatic Hydrothorax.
Contraindications include heart failure, severe pulmonary and severe liver disease.

Liver transplantation is the treatment of choice for refractory hepatic hydrothorax with poor liver function and after an SBEM episode. The challenge is to determine the appropriate treatment to bridge patients to liver transplantation when TIPS is not a good option. In the last 30 mins, I have showed you the diagnostic approach and the therapeutic options for patients with refractory hepatic hydrothorax. In summary, Hepatic hydrothorax should be suspected in cases of pleural effusion in the presence of chronic liver disease. Early diagnosis is critical and thorough evaluation is key. Complications increase mortality and hepatic hydrothorax without ascites or with minimal ascites like in our patient is possible! Lastly, therapeutic options are meant to alleviate symptoms, improve the quality of life, prevent complications and most importantly, bridge patients successfully and safely to liver transplantation.

In the words of Missy Higgins:

Dr. Grace Arellano, Chief Pulmonary Fellow-in-Training at UP-PGH, congratulates the presentors and MDH consultants for presenting such an interesting topic. Dr. CamiloRoa, a well known physiologist and exceptional pulmonologist,
enlightened the everyone of the physiology behind hepatic hydrothorax and the differences and interplay of pressures in the abdominal and pleural cavities. Dr. Janus Ong, a well-known hepatologist/liver specialist, gave his insights and experience, and the updates in management of hepatic hydrothorax and the different treatment modalities available for this uncommon complication of end stage liver disease. Dr Luminardo Ramos, thoracovascular surgeon discussed the value of early diagnosis using invasive procedures and the yield of accurate results using these modalities.

Resource persons were given tokens of appreciation, which was awarded by Dr. Aileen Wang, section chief and Dr. Dennis Teo, the training officer. Dr. Gregorio Ocampo, PCCP board member ended the program with his closing remarks.

Overall, the event is a success and hoping that all the Pulmonary Fellows-in-Training bring home in their perspective institution the knowledge that we shared with them.