INTRODUCTION

Hepatic hydrothorax (HH) is defined as a transudative pleural effusion that is larger than half a liter in a patient with portal hypertension due to liver cirrhosis without any coexisting cardiopulmonary and renal disease [1-3]. Hepatic hydrothorax occurs in 5-11% of patients with liver cirrhosis and leads to increased morbidity and mortality [4-7]. Most cases present as isolated right-sided pleural effusion that is refractory to conventional management [1,2,6]. Indwelling tunneled pleural catheter placement (ITPC) is shown to be a safe and effective therapeutic option particularly when liver transplant or intrahepatic portosystemic shunt (TIPS) are contraindicated or not available.

CASE REPORT

A 66-year-old woman with a history of liver cirrhosis secondary to methotrexate hepatotoxicity, pancreatic mass status post Whipple procedure, psoriatic arthritis and peripheral vascular disease presented with 2 weeks of shortness of breath and bilateral lower extremity swelling. Her physical exam was notable for normal vital signs, 3+ pitting edema in bilateral lower extremities, distended soft abdomen, jugular venous distension and diminished breath sounds in left lung fields. Laboratory analysis was significant for an acute kidney injury with creatinine of 1.8 mg/dL, proBNP of 1050 pg/mL, and liver indices compatible with known mild liver cirrhosis. No significant proteinuria was identified on urinalysis. Initial chest radiography showed a large loculated left pleural effusion (Figure 1). Admission model for end-stage liver disease (MELD) score was 14, indicating a less than 2% 3-month mortality.

Echocardiography demonstrated normal ventricular and valvular function. Abdominal ultrasound with doppler showed cirrhetic liver with patent hepatic veins and reversal of flow within the main and right portal veins (Figure 2). Abdominal CT revealed a large left hydropneumothorax, extensive intra-abdominal varices, small ascites, partial obstruction within the intra- and infra-hepatic inferior vena cava (IVC), thrombosis of main portal vein and limited appearance of right and middle hepatic veins suggesting Budd-Chiari syndrome (Figure 3). The patient required 2 therapeutic thoracenteses 3 weeks apart, both resulted in retrieving 1.5 liter of clear yellow transudative fluid with negative cytology. Portal venous pressure measurement was attempted via the right internal jugular vein however discontinued due to high-grade stenosis of...
superior vena cava. Due to recurrent left sided effusions, the patient had ITPC placed and started on oral anticoagulation for Budd-Chiari syndrome (Figure 4). During the 30-day hospital course, the patient’s respiratory status remained stable without any oxygen requirements. She was stable during a post-discharge follow up and liver transplant evaluation was initiated.

Figure 2: Abdominal ultrasound with cirrhotic appearing liver and reversed flow within main and right portal veins.

Figure 3: Abdominal CT scan showing cirrhotic liver and occluded main portal vein.

Figure 4: Chest radiograph with improvement in left sided pleural effusion after indwelling pleural catheter placement.

3 | DISCUSSION

Hepatic hydrothorax (HH) is a rare complication of portal hypertension defined as transudative pleural effusion that is larger than half a liter in a patient with portal hypertension due to liver cirrhosis without any coexisting cardiopulmonary and renal disease [1-3]. The incidence of HH is 5-11% in cirrhotic patients with right sided HH occurring in 59-80%, left sided in 12-17% and bilateral in 5-24% of cases [1]. More than 90% of HH cases present with concomitant ascites [8,9]. Several theories proposed that HH is resultant of the movement of ascitic fluid through diaphragmatic defects [1,2,7].

The initial management of HH involves sodium restriction for goal < 2 g/day, diuretic therapy with spironolactone and furosemide (100-40 mg/day ratio), and thoracentesis for symptomatic support [1,2,7]. Refractory HH can be managed by serial thoracenteses, however many cases require ITPC placement, pleurodesis, or surgical repair of diaphragmatic defects [1,2]. Liver transplantation is the definitive treatment with positive long-term outcomes [2,5-7]. The next leading treatment for patients who are not candidates for liver transplant or awaiting transplant is TIPS, which is usually used to treat refractory ascites and in secondary prevention of esophageal variceal hemorrhage [2]. However, TIPS showed promising efficacy in cases of portal vein thrombosis, hepatorenal syndrome, Budd-Chiari syndrome and HH [3,10]. TIPS decreases portal pressure by diverting venous blood flow via stent between the intrahepatic portal vein and hepatic vein [1,3]. In a large case series, Dhanasekaran et al. showed the rates of effectiveness of TIPS for refractory HH at 1 and 6 months as 79% and 75% respectively with a 19% 30-day mortality rate [11]. A similar retrospective case series by Campos et al. revealed improvement in patients’ ascites in 73% of cases in 1 month with a 25% 30-day mortality rate [12]. Common adverse effects post-TIPS include hepatic encephalopathy and liver ischemia [2,3,6,13]. TIPS is contraindicated in patients with severe congestive heart failure, pulmonary hypertension, sepsis or severe hepatic dysfunction with MELD scores more than 18 [3].

Our patient underwent ITPC placement for symptomatic treatment of refractory HH. ITPC are traditionally used for treatment of malignant pleural effusions but are now being used more often for non-malignant cases such as HH [1,2,4,5,7,14]. A multicenter retrospective study by Shojaee et al. found that after ITPC placement in 79 patients with refractory HH, 28% of cases achieved spontaneous pleurodesis and average time to removal of ITPC was 55 days [4]. A systematic review by Baig et al. found that 16 patients out of 51 (31%) attained spontaneous pleurodesis after ITPC placement, with duration of catheter placement ranging 73-222 days [6]. Common complications of ITPC placement include pleural fluid infection, electrolyte imbalance and renal failure [1,4,6,7,14]. Mortality data is lacking in most studies, however Chen et al. showed no mortality linked to ITPC infection in their prospective study [7]. Baig et al. found 18% of cases achieved spontaneous pleurodesis and average time to removal of ITPC was 55 days [4]. A systematic review by Baig et al. found that 16 patients out of 51 (31%) attained spontaneous pleurodesis after ITPC placement, with duration of catheter placement ranging 73-222 days [6]. Common complications of ITPC placement include pleural fluid infection, electrolyte imbalance and renal failure [1,4,6,7,14]. Mortality data is lacking in most studies, however Chen et al. showed no mortality linked to ITPC infection in their prospective study [7].

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4 | REFERENCES


