CASE REPORT

Bilateral indwelling pleural catheter for hepatic hydrothorax

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SUMMARY

A 68-year-old man with alcoholic liver cirrhosis and portal hypertension was admitted due to ascites. Bilateral severe hepatic hydrothorax did not resolve despite sodium restriction, and diuretic treatment, requiring repeated thoracentesis for relief of dyspnoea. Curative options, as transjugular intrahepatic portosystemic shunt and liver transplantation were both contraindicated. An attempt of pleurodesis was ineffective. Bilateral insertion of tunnelled pleural catheter allowed symptom control and home discharge. After 6 months, bilateral stable pleurodesis has been achieved and patient is still free from dyspnoea.

BACKGROUND

Tunnelled indwelling pleural catheter is a validated treatment option for malignant pleural effusion. However, its role in recurrent non-malignant pleural effusion is less established. Hepatic hydrothorax (HH) is described in up to 15% of patients with end-stage liver disease,¹ with significant impact on morbidity and quality of life in patients with end-stage liver disease. Options for patients who are not candidate to curative treatments, that is, liver transplantation, are limited and often ineffective. We present a case of HH who was successfully treated with bilateral indwelling pleural catheter, allowing symptoms control and management in the outpatient setting.

CASE PRESENTATION

A 68-year-old man diagnosed with alcoholic liver cirrhosis, with portal hypertension-related complications: grade F3 esophageal non-bleeding varices and moderate gastropathy, reported an increase in abdominal circumference and shortness of breath in the previous 2 weeks. Physical examination indicated decreased breath sounds in the left hemithorax, with dullness on percussion; vascular spiders and a positive puddle sign were observed.

Laboratory test showed sodium level 129 mmol/L, creatinine 1.34 mg/dL, alkaline phosphatase 76 U/L, γ -glutamyl transferase 88 U/L; haemoglobin was 12.5 g/dL with a mean corpuscular volume of 102 fL; his white cell count and platelet count as well as aspartate aminotransferase and alanine aminotransferase were within normal limits.

A chest radiography (CXR) confirmed the clinical suspicion of a submassive left pleural effusion (PE) (figure 1A). Left thoracentesis was performed, draining 1500 cc of clear serous pleural fluid. Laboratory analysis revealed a transudative PE, with negative microbiological cultures and benign

cytology. A spontaneous bacterial peritonitis was ruled out by paracentesis. CT scan showed left severe PE, cirrhotic liver without any focal lesion, portal vein thrombosis and severe ascites. CT scan findings, along with a normal bronchoscopic exploration of the airway, definitely ruled out lung and liver malignancy. On the basis of these findings, we confirmed a diagnosis of HH.

A combination of furosemide and spironolactone was started and progressively increased in a step-wise manner, in order to achieve a renal excretion of at least 120 mEq sodium/day. In the following days, despite sodium restriction and diuretic treatment, he developed bilateral PE, predominantly left-sided, requiring repeated left thoracentesis for relief of dyspnoea; furthermore, regardless of timely albumin infusions, he experienced deterioration in his renal function, which further limited volume depletion treatment.

TREATMENT

Owing to refractory HH, we considered the option of transjugular intrahepatic portosystemic shunt (TIPS), but the finding of portal vein thrombosis contraindicated the procedure. Liver transplantation and repair of diaphragmatic defects through video-assisted thoracoscopic surgery were contraindicated because of the high comorbidity-based surgical risk. In the end, we settled on insertion of a tunnelled pleural catheter (TPC; PleurX System) on the left side.

After few days, the patient experienced progressive dyspnoea, in spite of daily drainages (~700 mL/day) through the left TPC; a CXR showed worsening severe right PE and complete re-expansion of the left lung (figure 1B). Talc slurry pleurodesis, through an 8F chest tube in the right pleural cavity, was initially effective in reducing right PE, allowing chest tube removal and patient discharge from the hospital.

During the following weeks, a progressive decrease in the amount of drainage from the left catheter was observed; after ruling out drain obstruction or dislocation, spontaneous pleurodesis was confirmed (20 days after TPC insertion); nevertheless, right PE relapsed, hence we decided to insert a TPC in the right side too (figure 2A).

OUTCOME AND FOLLOW-UP

At present, 6 months after the insertion of the second TPC, patient is still free from left PE, and the amount of drainage from the right side is progressively reducing (figure 2B).

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Figure 1 (A) Posteroanterior projection of chest X-ray showing complete white-out of left hemithorax, with slight mediastinal shift. CT scan confirmed severe left pleural effusion. (B) New onset right-sided pleural effusion. After pleural catheter insertion (arrow), left lung is fully expanded and only mild residual effusion is observed in the costophrenic angle.

Figure 2 (A) Bilateral tunnelled pleural catheter. (B) Chest X-ray showing tunnelled pleural catheters (arrows) without residual pleural effusion.

DISCUSSION

Nearly 20% of HH are refractory to salt restriction and diuretics, requiring repeated thoracentesis for relief of symptoms that expose the patient to the risk of complications such as pneumothorax, haemothorax or re-expansion pulmonary oedema. In patients with refractory HH, liver transplant is the curative treatment; for patients who are not candidate or as bridge to transplant, TIPS can be performed as alternative, with high success rate reported in the literature.² Surgical repair of diaphragmatic defects prevent the transfer of ascitic fluid across the diaphragm, but data concerning feasibility are scarce. TPC seems to be a reasonable option for patients who are not eligible for the latter strategies. It has been reported to be effective in the management of malignant recurrent PE, although scientific evidence is weaker in non-malignant conditions.^{3–5} One of the most notable point in its favour is the possibility for early hospital discharge and home care management; however, this option should imply a great effort in order to improve education of patients and their caregivers. In this view, concerns exist regarding the risk of TPC-related pleural infection, which has been reported up to 12% of patients.⁶ Pathogenesis of this latter phenomenon has not been clarified; it seems related to bacterial migration from the skin, as *Staphylococcus aureus* is the most represented pathogen in pleural fluid culture and, notably, could be mainly associated with inadequate catheter aftercare and deficient immune system of the host, rather than to the insertion procedure.⁶ Prolonged drainage through a chest tube has been associated with electrolyte loss, protein depletion and impaired immunological function. Some authors suggest that the intermittent removal of small amounts of fluid via the TPC significantly reduces the chance of renal dysfunction, commonly associated

with the massive fluid loss through a conventional chest tube. Currently, data reporting the effect of IPC drainage, with respect to conventional chest tube, on nutrition and immunology are scarce. In our patient, we did not perform any systematic pleural fluid culture as we did not observe any sign of infection; on the other hand, an oral protein supplementation with liver-adapted solution was provided, as a mild drop in albumin level was observed. While the success rate of chemical pleurodesis in this context remains low,⁷ spontaneous pleurodesis after TPC placement has been frequently described in case series.^{4, 8} Interestingly, patients with non-malignant pleural effusion seem to have significantly higher time to pleurodesis compared with malignant aetiology (110.8+41 days vs 36+12 days), and patients with HH have shorter time to spontaneous pleurodesis compared to congestive heart failure. Increased levels of circulating inflammatory mediators in persons with cirrhosis could be responsible for rapid pleural symphysis.⁴

Patient's perspective

Patient definitely appreciated the option of home care management. After a brief education course on how to drain at home with the PleurX system, the patient and his family felt confident with the drainage system.

Learning points

- ▶ Hepatic hydrothorax (HH) contributes to increase morbidity in patients with end-stage liver disease.
- ▶ This case, in line with recent literature, provides evidence about effectiveness and safety of tunnelled pleural catheter (TPC) in HH.
- ▶ In patients not eligible for curative treatment, TPC insertion should be considered as alternative strategy to provide symptoms control and management in the home care setting.

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