adenocarcinoma.⁵ Expressions of thyroid transcription factor 1 and epithelial membrane antigen in round and surface cells of SH, accompanied by negative results of round cells for pancytokeratin, provide useful clues for the diagnosis of SH.² Further studies are needed to elucidate the histogenesis and biologic behavior to aid in the diagnosis of SH. In our case, a false frozen-section diagnosis of non-small cell lung cancer was made because of the presence of copious mucoid substances and nuclear atypia. Lobectomy of the right upper lobe with radical lymph node dissection was therefore performed. Pathologic examination of permanent sections revealed pulmonary SH.

The characteristic computed tomographic and magnetic resonance imaging features of SH may help with preoperative diagnosis and assessment.

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Long-term remission of hepatic hydrothorax after OK-432 pleurodesis

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Hepatic hydrothorax, a manifestation of end-stage liver disease, is defined as the presence of more than 500 mL of pleural effusion in patients with liver cirrhosis who do not have cardiopulmonary diseases or malignancies that could explain this effusion.¹ Strauss and Boyer² have reported incidences of 85.4 right-sided, 12.5% left-sided, and 2% bilateral hepatic hydrothoraces. The most likely cause is fluid transfer from the abdomen to the pleural space through diaphragmatic defects.¹ Optimal management remains unclear, and thoracentesis is frequently required for immediate symptom relief. Alternative treatments, including pleurodesis by tube thoracostomy or video-assisted thoracoscopic surgery (VATS), diaphragm repair, transjugular intrahepatic portosystemic shunt, peritoneovenous shunts, or liver transplantation, should be considered when frequent therapeutic thoracentesis is needed.¹ We report here the first case of refractory hepatic hydrothorax in which a 5-year remission was achieved by nonsurgical OK-432 pleurodesis.

CLINICAL SUMMARY

A 78-year-old woman with hepatitis C–related Child–Pugh class C liver cirrhosis came to our hospital with new-onset dyspnea and bilateral lower leg pitting edema. There was a history of refractory right-sided hepatic hydrothorax 5 years previously that was managed with OK-432 pleurodesis through a pigtail after failure of repeated minocycline pleurodesis. Surgical intervention was refused by the patient at that time because of older age and poor liver reserve. The patient had a 5-year symptom-free period with a regimen of oral diuretics and sodium restriction.

The physical examination revealed icteric sclera, right lower lung field dullness with decreased breath sound, and bilateral lower extremity pitting edema. The oxygen saturation was 82% on room air. Results of the neurologic examination were unremarkable. The hemogram was normal, whereas blood biochemistry studies revealed hyponatremia (130 mmol/L), hypoalbuminemia (2.8 g/dL), and abnormal liver function (total bilirubin 3.5 mg/dL, direct bilirubin 1.42 mg/dL, aspartate aminotransferase 58 units/L, alanine aminotransferase 24 U/L).

Chest radiography showed a large amount of right pleural effusion (Figure 1, A). Echocardiography revealed a good left ventricular ejection fraction. Chest computed tomographic scan disclosed right-sided pleural effusion with right lower lobe atelectasis, marked liver cirrhosis, esophageal

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FIGURE 1. A, Initial chest radiograph showing right-sided, massive hepatic hydrothorax. B, Chest radiographs 6 months after second successful pleurodesis with OK-432 reveal stable and loculated pleural effusion.

FIGURE 2. Chest ultrasonography showing loculated pleural effusion (PE) and thickened pleura (PL, arrows) over right hemidiaphragm.

varices, and minimal ascites. Cytologic studies of the pleural effusion and ascites were both negative.

Together with daily pleural effusion drainage of as much as 1000 mL, as well as albumin and diuretic administration, pleurodesis with 800 mg minocycline through a pigtail catheter was performed three times during the 8th to 14th hospital days but failed. Transjugular intrahepatic portosystemic shunt or VATS pleurodesis for the intractable hydrothorax was again refused by the patient. Pleurodesis with OK-432 10 KE was performed on the 17th hospital day, and the amount of daily effusion decreased dramatically to less than 30 mL without complications. The dyspnea improved significantly, and the oxygen saturation could be maintained above 95% on room air. The pigtail catheter was removed on

the 20th hospital day, and the patient was discharged in stable condition with a regimen of oral diuretics.

No further thoracocentesis was required for symptom relief. Follow-up chest radiographs (Figure 1, B) 6 months later showed a right loculated pleural effusion. Chest ultrasonography (Figure 2) revealed thickened pleura of up to 5 mm over the right hemidiaphragm. The patient has remained free of symptoms and is leading an uneventful life.

DISCUSSION

Medical treatments of salt restriction and diuretics are mainstays of therapy for hepatic hydrothorax but have only a temporary effect in a third of cases.² Although peritoneovenous shunt and transjugular intrahepatic portosystemic shunt have been reported to be useful, frequently encountered complications, such as hepatic encephalopathy and infection, limit their clinical applications. These procedures are thus better reserved as a bridge to liver transplantation.¹

Successful pleurodesis with tetracycline through a tube thoracostomy for hepatic hydrothorax was first reported in 1977.² Repair of diaphragmatic defects with sutures and biologic glue by VATS was successfully applied for this condition in 1996. Since then, VATS pleurodesis with or without diaphragmatic repair has been considered the technique of choice.³ The chemical agents used included tetracycline, talc, minocycline, and OK-432.^{2,4,5}

OK-432 is an immune modulator purified from the SU strain of *Streptococcus pyogenes* A3 that has been proved effective in controlling malignant ascites and pleural effusion.⁶ In Japan, it is also used in pleurodesis for pneumothorax and chylothorax.⁷⁻⁸ The experience of OK-432 pleurodesis for hepatic hydrothorax through a tube thoracostomy is limited to a small number of Japanese patients.⁹

Previous reports on hepatic hydrothorax have used different definitions of successful pleurodesis. Radiologic evidence of recurrence is frequently observed within 2 months, even after VATS pleurodesis.^{3,5} Our patient is the first reported to undergo OK-432 pleurodesis that resulted in a 5-year remission. The second pleurodesis with OK-432 was once again successful in terms of dramatic resolution of the recurrent hepatic hydrothorax after failure of repeated minocycline pleurodesis.

In conclusion, patients with end-stage liver cirrhosis and refractory hepatic hydrothorax have few therapeutic options. Our case demonstrates that OK-432 pleurodesis through a pigtail catheter is safe and can provide an effective alternative for patients for whom surgical intervention is not suitable.

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Perioperative nesiritide and possible renal protection in patients with moderate to severe kidney dysfunction

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Postoperative renal dysfunction develops in approximately 5% to 30% of patients undergoing cardiac surgery with cardiopulmonary bypass, is a potent predictor of poor outcomes, and is especially prevalent among patients with preexisting renal dysfunction.^{1,2} Strategies to protect kidney function during cardiac surgery have included the use of low-dose dopamine, fenoldopam mesylate (INN fenoldopam), bicarbonate infusion, and diuretics, yet efficacy of any treatment has been difficult to demonstrate.

Nesiritide, a recombinant form of human B-type natriuretic peptide, has been suggested to be renally protective

for patients with end-stage heart failure.³ Additionally, in the exploratory Nesiritide Administered Peri-Anesthesia (NAPA) trial, patients with congestive heart failure undergoing cardiac surgery who received nesiritide had a significantly lower incidence of postoperative renal dysfunction.⁴ The goal of this post hoc evaluation of the NAPA trial was to assess the impact of perioperative nesiritide on patients with preexisting, non-hemodialysis dependent, moderate-to-severe renal dysfunction.

MATERIALS AND METHODS

The design and methodology of the NAPA trial has been reported previously.⁴ In brief, patients with chronic left ventricular dysfunction underwent cardiac surgery utilizing cardiopulmonary bypass. Patients were randomly assigned to treatment with intravenous infusion of nesiritide (0.01 µg/[kg · min] without bolus) or placebo in addition to usual care. Study drug infusion was initiated after induction of anesthesia and continued for 24 to 96 hours after surgery. Inotropes and vasopressor medications were used without restriction in accordance with institutional practice. In the NAPA trial, 303 patients were randomly assigned, of whom 272 (nesiritide n = 137, placebo n = 135) ultimately underwent surgery. Of these 272 patients, 266 (nesiritide n = 133, placebo n = 133) had a baseline serum creatinine measurement and at least one postoperative serum creatinine measurement, and these patients form the basis for this evaluation.

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