Resistant hepatic hydrothorax: a successful case with treatment by nCPAP

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Summary Hepatic hydrothorax is defined as pleural effusion with liver cirrhosis but no primary cardiopulmonary disease. Hepatic hydrothorax is often resistant to various therapeutic interventions. The most likely cause is the transfer of ascites fluid from the abdomen to the pleural space via the diaphragm because of a negative intrathoracic pressure gradient. A 62-year-old man was diagnosed with hepatoma and cirrhosis. After a partial hepatectomy, he suffered with hepatic hydrothorax. He had snoring without obvious sleep apnea. The patient's hepatic hydrothorax markedly improved following nasal continuous positive airway pressure (nCPAP) treatment during sleep. The mechanism for the improvement may have been the intrathoracic positive pressure during sleep induced by the nCPAP treatment during sleep. nCPAP treatment may provide a new therapy for resistant hepatic hydrothorax.

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Introduction

About 6\% of cirrhotic patients have a significant pleural effusion.\textsuperscript{1,2} The effusion in a patient with liver cirrhosis but no primary cardiac or pulmonary disease is called hepatic hydrothorax (HH). Ascites exists to some degree in most patients with progressive liver cirrhosis, and it permeates the diaphragm, moving into the pleural space.\textsuperscript{3} The conventional management of this condition consists of sodium restriction, diuretics, thoracentesis, and pleurodesis. But the effusion is difficult to eliminate because it continuously shifts from the abdomen into the pleural cavity as a result of the
pressure gradient. We used nasal continuous positive airway pressure (nCPAP) treatment in an HH patient who had habitual snoring, and his HH markedly improved. nCPAP converts the negative intrathoracic pressure to positive and may prevent the shifts of fluid.

Case report

A 62-year-old man, who was a carrier of the hepatitis type B virus, was diagnosed with hepatoma. He underwent a partial hepatectomy in Kyoto University Hospital. Pathological examination showed severe liver cirrhosis and poorly differentiated hepatocellular carcinoma. The patient made good progress after the operation but had right-sided pleural effusion and passive atelectasis. After release from the hospital, two months of careful observation and sodium restriction with diuretics could not reduce the pleural effusion.

His chest radiograph and computed tomography on re-admission showed pleural effusion and passive atelectasis. After release from the hospital, two months of careful observation and sodium restriction with diuretics could not reduce the pleural effusion.

His chest radiograph and computed tomography on re-admission showed pleural effusion and slight atelectasis on the right side (Fig. 1A–C). His body mass index was $24.7 \text{ kg/m}^2$. Electrocardiogram and cardioechography showed no cardiac failure. Abdominal echography revealed slight ascites. Laboratory tests showed mild liver damage (aspartate aminotransferase $96 \text{ IU/L}$, alanine aminotransferase $104 \text{ IU/L}$) and hypothyroidism (95 $\times$ 10$^4$/L). Neither inflammation nor renal dysfunction were detected. Effusion was transudative and there was no sign of malignancy or infection. Bronchoscopy showed no apparent airway obstruction causing the local lung collapse. The first step of treatment for pleural effusion is to restrict daily sodium intake and to administer diuretics. His weight reduced slightly (63.0–60.5 kg in a week) but his effusion was not reduced by the standard therapy.

The patient had habitual snoring without daytime sleepiness. His night oximetry had a >3% desaturation index of 4.6 per hour. Since it has been reported that negative intrathoracic pressure during snoring is high, 5 cm H$_2$O of nCPAP (ResMed, NSW, Australia) was used at night to convert the negative intrathoracic pressure to positive. The >3% desaturation index with nCPAP decreased to 1.7 per hour and his snoring ceased. One week later, we found the effusion had decreased remarkably and the lung collapse disappeared on chest radiograph but his weight did not change (60.5–60.0 kg). After the near elimination of the effusion (Fig. 1D), the patient left the hospital.

Figure 1 (A) Chest radiography on admission shows right pleural effusion. (B) Chest radiography in the right decubitus position shows massive effusion. (C) Chest computed tomography shows right pleural effusion and lung collapse. (D) Chest radiography on day 50 after usage of nCPAP shows no pleural effusion.
Following release, the patient discontinued nCPAP use at night. After one month, the pleural effusion returned. He resumed using nCPAP at night and the HH again improved.

Discussion

This habitual snorer with resistant HH improved with nCPAP treatment during sleep. Snoring in middle-aged patients is very common. Therefore, the incidence of concurring HH and snoring may be considerable. nCPAP treatment may provide a new therapy for resistant HH in addition to the usual medical care.

The most likely cause of HH is a direct transfer of fluid from the abdomen to the pleural space via defects in the diaphragm. The diaphragm can have defects both grossly and microscopically. Surgical procedures on the liver, of course, result in additional injuries to the diaphragm. The pathophysiology of ascites formation involves fluid leaking from the liver into the peritoneal cavity. The ascites collects around the liver, which is closely applied to the undersurface of the right hemidiaphragm. The negative pressure of the pleural space relative to the peritoneal cavity favors the one-way transfer of fluid across these defects and its subsequent trapping within the pleural space. In this case, hepatectomy may have induced additional defects in the diaphragm and further decreased the liver function.

The first step of treatment is to restrict daily sodium intake and to administer diuretics. But this therapy is often ineffective. Thoracentesis is effective for pulmonary symptoms but this therapy is temporary because of the inexhaustible supply of ascites fluid. Moreover it increases the risk for hypoproteinaemia, dehydration, infection, and other complications. Pleurodesis is usually unsuccessful because of the rapid re-supply of the effusion from the abdominal cavity. Other invasive therapies, such as repair of the diaphragm defects by videothoracoscopy and peritoneovenous shunts have also been tried but with only marginal success. Our findings suggest that conversion of the intrathoracic pressure from negative to positive might offer a new strategy to control HH.

The prevalence of snoring is reported to be high, sometimes estimated to be as high as 83%. Therefore, the prevalence of HH patients with snoring would also be expected to be high. It has been reported that intrathoracic negative pressure become worse during snoring, which would exacerbate the HH. Although this is only the report of a single case, the pleural effusion returned after stopping the nCPAP, and improved after nCPAP re-treatment. Further clinical research would need to establish nCPAP therapy as the appropriate therapy for HH, but we recommend this treatment for resistant HH in addition to the usual medical treatment because nCPAP treatment is noninvasive for almost all patients.

References