Pulmonary Pressure Necrosis due to Chronic Pleural Effusion after Heart Transplantation: A Case Report

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Abstract
A 51-year-old woman had severe restrictive cardiomyopathy with heart failure. The first symptoms had started 12 years ago, and her symptoms gradually worsened. She was treated with diuretics, intermittent but repetitive thoracentesis, and paracentesis due to recurrent pleural effusion and ascites. Consequently, a collapse of the right lower lobe (RLL) was noted. We stopped thoracentesis and paracentesis and added continuous intravenous diuretics for 3 months before heart transplantation (HT). Finally, she underwent HT. However, her RLL remained collapsed and chest tube drainage persisted. We performed a RLL lobectomy with video-assisted thoracic surgery. No specific pathologic findings were noted except pulmonary necrotic lesions. We report a rare case of pulmonary necrosis caused by mechanical compression due to chronic pleural effusion after HT.

Keywords
► transplantation
► heart
► pleural disease
► pathology

Introduction
Patients with congestive heart failure (CHF) generally develop pleural effusion and ascites, and a lung may collapse passively. The primary mechanism for pleural fluid formation in patients with heart failure is fluid entering the pleural space from the lung interstitium.1 Pleural fluid in a patient with CHF is most likely the result of cardiac cirrhosis with ascites and transdiaphragmatic movement into the pleural space or possibly paradoxical motion of the intraventricular septum causing left ventricular diastolic dysfunction.2 Diuretics (particularly loop diuretics) remain the most effective medication for relieving symptoms and improving the pathophysiologic status of fluid overload, including heart failure.3 However, if symptoms (dyspnea, orthopnea, etc.) are not relieved despite using appropriate diuretics, then the thoracentesis or paracentesis should be considered. Although both therapeutic methods can improve symptoms rapidly, repeated procedures may cause side effects such as infection, fibrin formation, and so on. Therefore, avoiding repeated attempts is better if the patient remains tolerable.4 Usually, these conditions resolve after heart transplantation (HT) and a collapsed lung will expand to near-normal size after clearance of the effusion and ascites. However, our patient’s RLL of the lung remained collapsed after HT.

Case Report
A 51-year-old woman had severe restrictive cardiomyopathy, and her first symptoms started 12 years ago. Despite appropriate medical therapy, her symptoms gradually worsened. She was treated with diuretics, intermittent thoracentesis, and paracentesis due to recurrent pleural effusion and ascites. After the thoracentesis, the right lower lobe (RLL) of the lung expanded; however, it collapsed repeatedly during the follow-up period (►Fig. 1A). We stopped thoracentesis and paracentesis and added continuous intravenous diuretics for 3 months before HT because of the possibility of infection and
trauma caused by the repetitive procedure. She finally underwent HT, but her RLL remained collapsed, and chest tube drainage persisted. We failed to find the cause of restrictive cardiomyopathy, a pathology finding showed just an interstitial fibrosis, and the Congo red stain was negative for amyloidosis. After rechecking the computed tomographic (CT) scan (Fig. 1B), we performed video-assisted thoracic surgery (VATS)-assisted RLL lobectomy through a mini-

Fig. 1 Chest X-ray and computed tomography scan. (A) Before heart transplantation. Both images show right pleural effusion and collapsed right lower lobe (RLL) of a lung. (B) The RLL remains collapsed after heart transplantation (B-1). A large cystic lesion was observed in the in RLL (B-2). (C) All lesions were resolved with minimal pleural effusion after the RLL lobectomy.

Fig. 2 (A) Gross examination shows a relatively demarcated cystic lesion filled with semisolid hemorrhagic material. This cystic lesion was confined within lung parenchyma, and did not involve the bronchial system. (B) The cystic wall shows a fibrotic capsule overlying fibrinoid material without any epithelial lining (hematoxylin and eosin [H&E], ×40). (C) The fibrotic capsule consisted of fibroblasts intermingled with hemosiderin-laden macrophages, suggestive of an old hemorrhage (H&E, ×200).
posterolateral thoracotomy (15-cm incision). Intraoperative
dfindings revealed no effusion and the lung could not be
deflated due to the cystic cavity (►Fig. 2A). No specific
pathological findings were revealed except RLL necrotic
lesions. A large cavitory lesion with a thick capsule was found
(►Fig. 2A, B). The capsule of the cavity showed diffuse fibrosis
with intermingled hemosiderin-laden macrophages. The lu-
men of the cavitory lesion was filled with inflammatory cells,
preponderantly neutrophils, in the background of fibrinoid
material (►Fig. 2C). She had not shown any complications
(►Fig. 1C). She has no dyspnea, infection, or exercise limi-
tations for the past 4 years.

Discussion

Diuretic therapy is recommended to restore and maintain
normal volume status in patients with clinical evidence of
fluid overload, generally manifested by congestive symptoms
(orthopnea, edema, and shortness of breath), or signs of
elevated filling pressure (jugular venous distention, periph-
eral edema, pulsatile hepatomegaly, and rales). Loop diuretics
rather than thiazide-type diuretics are typically necessary to
restore normal volume status in patients with HF. However,
if symptoms are not relieved despite appropriate diuretics,
thoracentesis or paracentesis should be considered. Although
thoracentesis can improve symptoms rapidly, repeated pro-
cedures may cause side effects such as infection, fibrin
formation, pneumothorax, and so on. Chung et al reported
that repeated thoracentesis increases vascular endothelial
growth factor, proinflammatory cytokines (interleukin 1β,
tumor necrosis factor-α) and plasminogen activator inhib-
itors in pleural fluid. These increase fibrin formation and
interrupt reabsorption of effusion, which may result in fibrin
deposition and impaired resolution of pleural transudates.
The patient received a multiple series of thoracentesis, but
pleural effusion recurred repeatedly. Therefore, we stopped
the procedure and infused intravenous dobutamine and loop
diuretics on admission. She received a new heart after 3 months. But against our expectations, the RLL of the lung
did not expand and pleural drainage > 150 mL/day was
continuously produced. We found cystic lesions in the RLL
on follow-up CT scan (►Fig. 1C) and performed a VATS-
assisted RLL lobectomy.

The common causes of pulmonary necrosis are bacterial
pneumonia and pulmonary tuberculosis, and they both cause
vascular and bronchial obstructions. No signs of vascular or
bronchial obstructions were observed in our case.

In this case, the pulmonary necrosis was caused by me-
chanical compression without bronchial or vascular obstruc-
tion and infection. We may consider pleural effusion more
actively (preoperative bronchoscopy or opening the pleura to
check effusion status during HT) if we encounter a similar
patient in the future. We report a rare case of pulmonary
pressure necrosis on the RLL of the lung caused by mechanical
compression due to chronic pleural effusion after HT.

References

1 Kinasewitz GT, Jones KR. Effusions from cardiac diseases. In: Light
RW, Gary Lee YC, eds. Textbook of Pleural Diseases. 2nd ed.
London: Hodder Arnold; 2008:315–321
2 Porcel JM. Pleural effusions from congestive heart failure. Semin
Respir Crit Care Med 2010;31(6):689–697
3 Bellomo R, Prowle JR, Echeverri JE. Diuretic therapy in fluid-
overloaded and heart failure patients. Contrib Nephrol 2010;
164:153–163
4 Chung CL, Yeh CY, Sheu JR, Chen YC, Chang SC. Repeated thora-
centeses affect proinflammatory cytokines, vascular endothelial
growth factor, and fibrinolytic activity in pleural transudates. Am J
Med Sci 2007;334(6):452–457
5 Heart Failure Society of America. Executive Summary: HFSA 2006
Comprehensive heart failure practice guideline. J Card Fail 2006;
12:10–38