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Lung: Case Report

Improved Pulmonary Hypertension and Heart Failure by Diaphragmatic Plication and Tracheal Resection

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The patient was a 66-year-old man who developed exacerbation of respiratory and heart failure that necessitated tracheal intubation. The exacerbation was considered to be largely caused by asthma-chronic obstructive pulmonary disease overlap syndrome and type 3 pulmonary hypertension. However, left diaphragmatic eventration and tracheal stenosis were also found. We hypothesized that diaphragmatic eventration and tracheal stenosis surgeries would improve the patient's pulmonary function, pulmonary hypertension, and cardiac function. Postoperatively, he recovered well and was discharged home on room air, with a good performance status. He also showed improved pulmonary hypertension on echocardiography and improved pulmonary function test results.

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Diaphragmatic eventration and tracheal stenosis can cause chronic respiratory failure.[1-5] However, their effect on pulmonary hypertension and cardiac function is unclear. We herein describe successful sequential surgical repair of diaphragmatic eventration and tracheal stenosis, which improved the patient's pulmonary function, pulmonary hypertension, and cardiac function.

A 66-year-old man developed exacerbation of respiratory and heart failure that necessitated tracheal intubation. This exacerbation was mostly driven by asthma-chronic obstructive pulmonary disease (COPD) overlap syndrome and type 3 pulmonary hypertension. Asthma and COPD overlap syndrome affect approximately one quarter to one third of patients with COPD or asthma. This syndrome leads to worse respiratory symptoms and a higher risk of exacerbations and hospital admissions compared with COPD or asthma alone.[6] The patient also had left diaphragmatic eventration since childhood and tracheal stenosis over the second to fourth tracheal rings. The tracheal stenosis appeared to have been caused by previous tracheostomy for COPD exacerbation 2 years prior to presentation at our hospital (Figures 1A, 2A-2C). The diaphragmatic eventration was considered diaphragmatic paralysis because there was no clear movement of the patient's left diaphragm during ultrasonography; however, a fluoroscopic sniff test was not performed.

In addition to the patient's diminished respiratory function, cardiac function was a major concern. Right heart catheterization 1 year previously demonstrated pulmonary hypertension, with a mean pulmonary artery pressure of 34 mm Hg and left ventricular ejection fraction of 43% on echocardiography. Angiography revealed no coronary artery stenosis.

Retracheostomy was performed in addition to the use of diuretics and antibiotics, which were ongoing therapies for right heart failure and suspected bacterial pneumonia, respectively. Inotropic support and ventilator assistance were gradually weaned over several days. The patient subsequently became ambulatory on room air, but remained on the ventilator at night because of slight hypercapnia (maximum partial pressure of carbon dioxide, 64 mm Hg). To evaluate whether the degree of right heart failure was tolerable for the patient to undergo surgery, follow-up right heart catheterization was performed. This procedure revealed improved pulmonary artery pressure (mean, 22 mm Hg). We

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speculated that retracheostomy relieved the central airway obstruction and resulted in improvement of the secondary pulmonary hypertension. The patient was recovering well but suddenly developed acute cholecystitis, which required percutaneous transhepatic gallbladder drainage and antibiotics. The treatment options for the cholecystitis were to maintain permanent percutaneous transhepatic gallbladder drainage or perform cholecystectomy, as per a gastroenterologist's recommendations.

The surgical strategy was difficult to plan because of the multiple surgical targets and the patient's comorbidities: diaphragmatic eventration, tracheal stenosis, cholecystitis, acute respiratory failure, and pulmonary hypertension. We waited for 1 month, and, fortunately, the patient recovered well with initial therapy and the right heart failure improved sufficiently to permit moderate-risk procedures. Therefore, starting with diaphragmatic plication seemed reasonable, because this procedure is relatively low-risk and can achieve functional improvement. Lung expansion after diaphragmatic plication would help the patient's respiratory function and further reduce the pulmonary artery pressure, potentially benefitting cardiac function.

The patient was placed in the right hemirecumbent position to allow simultaneous abdominal and thoracic procedures. Laparoscopy confirmed adhesions between the transverse colon, spleen, omentum, and left diaphragm (Figure 1B). Once the abdominal adhesions were released, the operation table was rotated to the right decubitus position, and thoracoscopy was initiated. After the thoracic adhesions were released, the thin left diaphragm was rolled up using forceps, and the excess diaphragm was stapled anteroposteriorly (Video 1). While this procedure was being performed thoracoscopically, laparoscopy was also performed to avoid injury to the abdominal organs by the stapler (Figure 1C). The staple lines were reinforced with horizontal mattress sutures using 2-0 monofilament suture. The operation table was then rotated to the supine position, and laparoscopic cholecystectomy was performed. Postoperative radiographs showed excellent expansion of the left lung (Figure 1D). The patient was extubated to a tracheostomy cannula in the operation room and was ambulatory on postoperative day 1. The length of the postoperative intensive care unit stay was 2 days, and the patient recovered well.

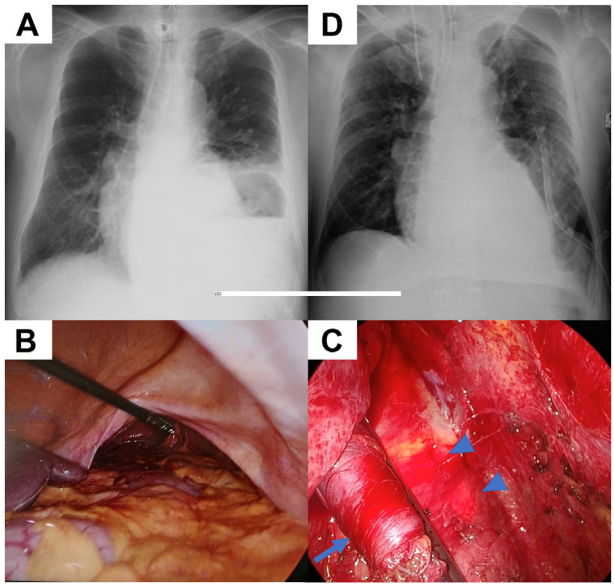

FIGURE 1 Preoperative and postoperative radiographs and surgical findings for the diaphragmatic plication. (A) Preoperative radiograph showing a markedly elevated left diaphragm. (B) Intraoperative laparoscopic findings. The stomach, spleen, transverse colon, and omentum were adhered to the diaphragm. (C) Thoracoscopic diaphragmatic plication. The thinned diaphragm was rolled up and stapled with simultaneous laparoscopic observation to avoid injuring the abdominal organs. (Arrow, rolled-up diaphragm; arrowhead, laparoscope light, which is recognizable through the thin diaphragm.) (D) Postoperative radiograph showing the expanded left lung.

Two weeks later, tracheal resection was performed. Mobilization of the thoracic trachea was highly restricted because of severe fibrotic adhesions and necrotic anterior tracheal cartilages from the second to fourth rings, possibly caused by repeat tracheostomy. Given the poor mobilization of the trachea and estimated strong tension at the anastomosis site, we abandoned segmental resection of the tracheal rings. Instead, bevel-shaped resection of the anterior cartilage from the second to fourth rings was performed. End-to-end anastomosis was performed with 4-0 polydioxanone running sutures for the back wall and 3-0 absorbable multifilament interrupted sutures for the front wall (Video 2).

The patient was extubated on postoperative day 4 because it was considered safer to wait until the in-out balance resolved given the underlying pulmonary hypertension. He was ambulatory on postoperative day 5. There was no reintubation or readmission to the intensive care unit through the subsequent course of therapy. The length of the intensive care unit stay was 6 days after the tracheal resection. Although his postoperative

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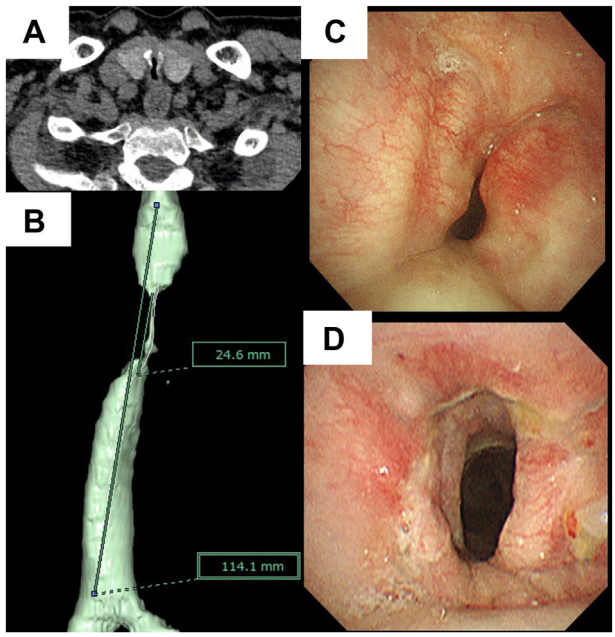

FIGURE 2 Preoperative and postoperative findings for the tracheal stenosis. (A) Preoperative computed tomography showing the tracheal stenosis. (B) Preoperative 3-dimensional reconstruction of the tracheal stenosis. (C) Preoperative bronchoscopy showing marked tracheal stenosis. (D) Postoperative bronchoscopy showing good opening of the anastomosis, with structural narrowing.

course was uneventful, the relatively strong tension at the anastomosis site was of concern. Therefore, we planned to hospitalize the patient for longer time for close monitoring. Postoperative bronchoscopy performed at 3 weeks revealed structural narrowing of the anastomosis without dehiscence or stenosis. This narrowing formed because of the bevel-shaped resection of the tracheal cartilages and tension at the anastomosis site, which did not hamper air flow (Figure 2D). This finding remained when follow-up bronchoscopies were performed at 4 weeks, 3 months, and 6 months postoperatively. The length of hospital stay after the tracheal resection was 4 weeks. His performance status was 1 when he was discharged. There were no complications following the first or second operation.

Pulmonary function tests revealed 23% and 56% improvement of forced vital capacity (preoperative, 1.73 L; postoperative, 2.12 L) and forced expiratory volume in 1 second (preoperative, 0.86 L; postoperative, 1.34 L), respectively. Transthoracic echocardiography also showed improvement: left ventricular ejection fraction +12% (preoperative, 43%; postoperative, 55%); right ventricular systolic pressure −20 mmHg (preoperative, 61 mm Hg; postoperative, 41 mm Hg). The patient was discharged home without oxygen and remained clinically well.

COMMENT

This patient presented with acute-on-chronic respiratory and cardiac failure associated with COPD, asthma, and secondary pulmonary hypertension, which were complicated by tracheal stenosis, diaphragmatic eventration, and cholecystitis. A previous study reported improved pulmonary hypertension after surgical repair of a Bochdalek hernia, in which the underlying mechanism of pulmonary hypertension improvement was quite similar to that in our case.[7] Theoretically, diaphragmatic plication expands an affected lung and increases the vascular bed, which should improve oxygenation and attenuate pulmonary artery pressure. The improved pulmonary hypertension can lead to improved cardiac function.

The Videos can be viewed in the online version of this article [https://doi.org/10.1016/j.atssr.2024.05.016] on http://www.annalsthoracicsurgery.org.

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