Clinical Oncology: Case Reports

Case Report

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Thoracic Duct Stent-Graft Decompression in Advanced Colon Cancer Patient with Refractory Chylothorax Caused by Thoracic Duct Obstruction and Associated Hypertension

Masayoshi Yamamoto*, Mitsuhiro Kinoshita, Junya Ichiki, Shuhei Inoue, Hiroshi Kondo, and Hiroshi Oba

Abstract

A 75-year-old man with a history of rectal cancer presented with a chylothorax during chemotherapy. Intranodal lymphangiography showed no leakage, but the thoracic duct was occluded distally, and there was no inflow of contrast medium into his venous angle. We percutaneously punctured the thoracic duct and inserted a micro-catheter. The intra-thoracic duct pressure was over 100 mmHg, and so we reconstructed the thoracic duct with a stent-graft. The pleural effusion decreased immediately after the procedure.

Malignancy-associated chylothorax is a rare condition characterized by the accumulation of chyle within the pleural space leading to respiratory distress, malnutrition, and immunological compromise; it also prevents the continuation of chemotherapy. We report a case of malignancy-associated chylothorax caused by obstruction of the thoracic duct treated with a stent-graft.

Keywords

Refractory chylothorax; Lymphorrhea; Thoracic duct; Thoracic duct hypertension; Thoracic duct decompression

Abbreviation

VA: Venous angle; TD: Thoracic duct; CT: Computed tomography; IL: Intranodal lymphangiography

Introduction

Refractory chylothorax caused by Thoracic Duct (TD) hypertension and obstruction is a relatively rare condition. In many cases, lymphangiography does not show extravasation [1]. Therefore, the leakage mechanism is unclear, and the treatment is often challenging. We present a case treated with thoracic duct recanalization with a stent-graft for refractory chylothorax caused by TD obstruction and associated hypertension.

Case Presentation

A 75-year-old man, after surgery for rectal carcinoma, was undergoing chemotherapy for multiple lung and para-aortic lymph node metastases. The patient developed respiratory distress and right-sided pleuritic chest pain. 18F-Fluorodeoxyglucose positron emission tomography/Computed Tomography (CT) did not show any accumulation in the mediastinum or chest wall. We inserted a chest tube, through which 1200 ml/day of whitish fluid was discharged. Pleural fluid cytology did not show any evidence of malignancy. Biochemical analysis showed that 29mg/dl triglycerides and chylomicrons were strongly positive. Initially, he was treated conservatively with a low-fat diet and two sessions of pleurodesis. These were ineffective. Therefore, we planned Intranodal Lymphangiography (IL).

The procedure was performed in the Angio-CT suite under local anesthesia. IL showed that the cisterna chyli was obstructed due to para-aortic lymph node metastasis, and the TD was revealed as a collateral pathway. It was still blocked at the cephalad portion of the TD (Figure 1a). CT showed no extra-lymphatic leakage. We successfully punctured the TD using a 21G Chiba needle under fluoroscopic guidance. A 0.018-inch micro guidewire (V18-Control, Boston Scientific, Marlborough, Massachusetts) was advanced through the needle into the TD. A 70cm microcatheter (Prominent Raptor, Tokai-medical, Japan) was advanced into the TD over the guidewire. We broke through and successfully reopened the obstructed TD (Figure 1b). Because the blocked TD was tortuous, the guidewire could not cross over. The procedure was completed to confirm the efficacy of the treatment.

After the procedure, the drainage volume decreased to 300 ml/day, but it began to increase within a day.

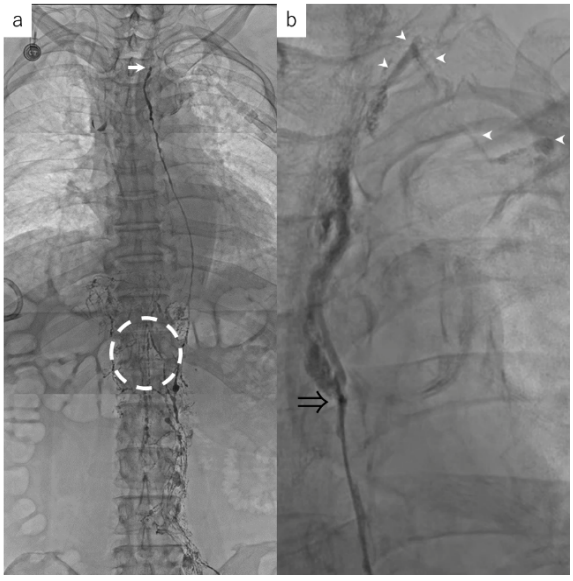

Figure 1: a) Intranodal lymphangiography showed that the thoracic duct is obstructed at the cephalad portion; b) Thoracic ductography after the resumption of lymph flow at the thoracic duct outlet. The venous angle is visualized. Arrow: Occluded thoracic duct; Arrowhead: Venous angle; Dotted circle: Depicted cisterna chyli; Dashed arrow: Tip of the microcatheter.

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A microcatheter was inserted into the TD as before, and a 5F sheath was added to the left brachial vein. The TD pressure measured from the microcatheter was 109/99 mmHg (mean 103 mmHg), and that of the subclavian vein was 2/1 mmHg (mean 0 mmHg). Even in the resting state, the backflow of chylous fluid was confirmed through the microcatheter. We tried to approach the occluded Venous Angle (VA) from the TD side, but could not break through the occlusion again, and the guidewire penetrated the TD wall. Therefore, we planned to reinsert the guidewire into the subclavian vein by drilling it from outside of the vessel from the TD side (Figure 2). A microcatheter was advanced along the extravascular guidewire to the vicinity of the subclavian vein wall. It was confirmed with a small amount of iodine contrast agent that was injected through the microcatheter and distributed along the wall of the subclavian vein. We successfully penetrated the subclavian vein drilling with the guidewire and using the microcatheter as a backup. A 10 mm Amplatz GooseNeck Snare (ev3, Irvine, CA, USA) was inserted into the subclavian vein from a sheath. The guidewire was captured by this snare and pulled out of the sheath. We inserted a 6F guiding sheath (Destination, Terumo, Japan) into the TD using a through-and-through wire technique from the left brachial vein side. We deployed a 6 mm × 10 mm VIABAHN (W.L. Gore, Newark, DE) into the occluded TD by inserting it through the sheath. The stent-graft was dilated with a 5mm x 4cm balloon (Mustang, Boston, US), and TD-graphy showed good flow. Following treatment, the previously high TD pressure was decreased to 16/12 mmHg (mean 14 mmHg).

The patient developed no complications postoperatively, and the pleural fluid decreased immediately (Figure 3). After five days, the drainage volume was 50 ml/day, and pleurodesis was performed using OK-432 (Picibanil Chugai Pharmaceutical Co. Ltd. Tokyo, Japan) solution (5 KE/2 ml). The patient was discharged after removal of the chest tube three days later (Figure 3). Clopidogrel sulfate 75 mg was administered to prevent stent occlusion.

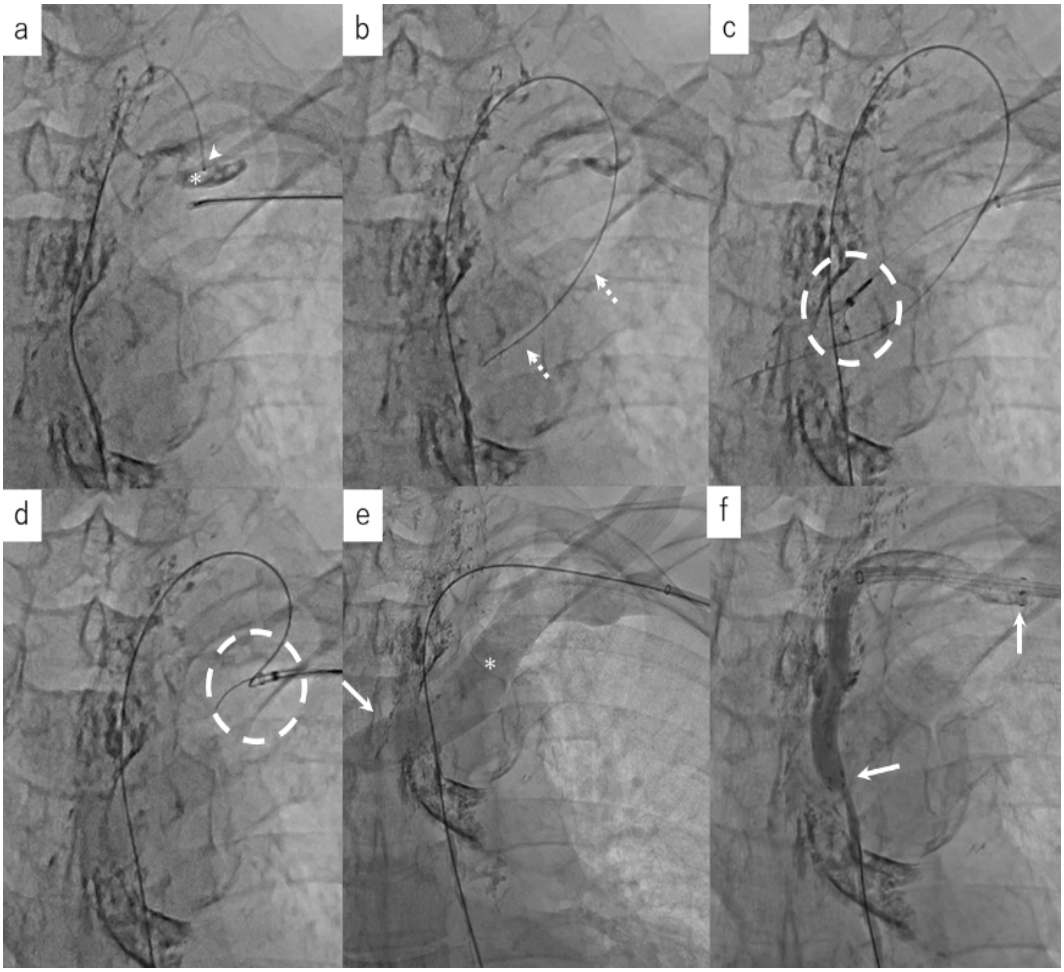

Figure 2: Method of thoracic duct decompression; a) A small amount of contrast agent is injected through a microcatheter near the venous angle to confirm the location of the subclavian vein. A 5F catheter is inserted from the brachial vein into the subclavian vein; b) The micro guidewire has penetrated the vein wall and is advancing into the subclavian vein; c) The micro guidewire was placed in the snare loop advanced from the sheath indwelling in the left subclavian vein; d) The micro guidewire, which was grasped by the snare, was taken out of the body; e) Subclavian vein angiography is performed from the guiding sheath advanced along the micro guidewire. After this, the guiding sheath is advanced from the subclavian vein side into the thoracic duct; f) The covered stent should be deployed so that the proximal side of the stent is the thoracic duct, and the distal side is the subclavian vein. Lymphangiography performed through a microcatheter shows good flow in the stent; Arrowhead: Tip of the microcatheter; Dashed arrow: Micro guidewire; Dotted circle: A micro guidewire is passed through the snare loop; Arrow; Covered stent.

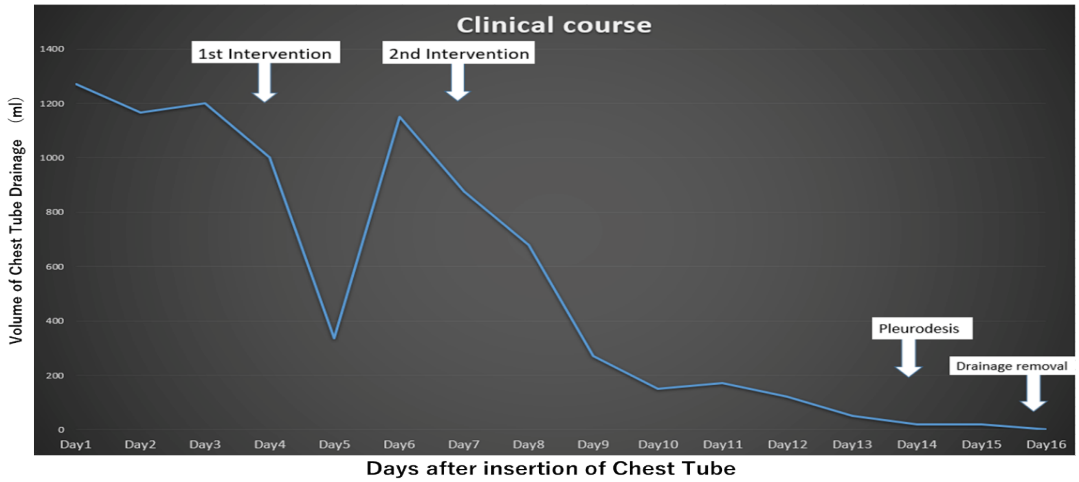

Figure 3: The clinical course of our case. After chest tube placement, the patient was treated on days 4 and 7; drainage improved quickly after thoracic duct decompression, pleural adhesions were performed on day 14, and he had the chest tube removed on day 16.

After six months, there was no recurrence of either the pleural effusion or ascites. Patient consent was obtained for publication of this case report.

Discussion

The mechanism underlying refractory chylothorax caused by TD hypertension and obstruction is unclear. A few reports have described TD decompression for lymphatic congestion [2-4], but none have described TD decompression for malignancy-related chylothorax like in the present case. We assume the mechanism is as follows: lymphatic fluid in the TD is advanced by the beating of the thoracic aorta [5]; there are valves every few millimeters in the lymphatics, which interfere with the backflow of lymphatic fluid; only the cephalad portion of the TD was occluded and the TD accompanying the descending aorta was not occluded; pulsation of the descending aorta was transmitted to the accompanying TD [5], and the existence of the valve structure promoted hypertensive like portal hypertension; the high pressure of TD spreads throughout the lymphatic system, rupturing peripheral lymph ducts and causing lymphorrhea. We speculate that this phenomenon may occur in some cases in which the site of the leak cannot be identified by lymphangiography.

Thus, we measured the TD pressure and treated TD hypertension by reconstructing the occluded TD with a stent-graft to reduce the pressure in the TD, thereby leading to the reduction of pressure throughout the entire lymphatic system.

Conclusion

Stent-graft decompression may help to clarify the leakage mechanism and may by itself be a sufficient treatment option for refractory malignancy-associated chylothorax due to TD obstruction and associated TD hypertension.

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