

Percutaneous Balloon Plasty for Thoracic Duct Occlusion in a Patient with Chylothorax and Chylous Ascites

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Received: 12 October 2018 / Accepted: 21 December 2018 / Published online: 7 January 2019

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Abstract A patient developed abdominal distension, dyspnea, and nausea due to chylothorax and chylous ascites 1 month after bruising her back. Lymphangiography was unable to identify the site of lymph leakage, and lymphatic duct embolization was impractical. However, lymphangiography showed occlusion of the thoracic duct. Thus, balloon plasty was performed to restore the patency of the thoracic duct, and the chylothorax and chylous ascites improved. Although embolization of the thoracic or lymphatic ducts has been reported as a treatment for lymphorrhea, it is impractical if the lymphatic duct responsible for leakage cannot be identified. In such a case, balloon plasty of the occluded thoracic duct to lower the pressure in the peripheral lymphatic ducts was successfully performed.

Keywords Chylothorax · Chylous ascites · Thoracic duct plasty · Lymphangiography · Balloon

Introduction

The use of thoracic duct and lymphatic duct embolization to treat chylothorax, chylous ascites, and lymphocele has been reported [1–5]. Such treatment works by selectively embolizing the lymphatic duct that is the source of the leakage. However, in some cases, embolization cannot be performed if the lymphatic duct responsible cannot be identified.

In this report, the case of a patient with chylothorax and chylous ascites in whom leakage continued despite conservative treatment and therapeutic lymphangiography, but for whom embolization was impractical because the site of leakage could not be identified is presented. This patient was successfully treated with balloon thoracic duct plasty of the occluded thoracic duct to restore patency.

Case Report

An 82-year-old woman had fallen and hit her back. She subsequently developed chylothorax and chylous ascites, with the appearance of abdominal distension and dyspnea on exertion, as well as nausea as they progressed. The patient had a history of hypertension, atrial fibrillation, and right femoral fracture, but there was no history of thoracoabdominal surgery. The triglyceride levels were 400 mg/

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dL in the pleural effusion and 566 mg/dL in ascites ascitic fluid. Due to abdominal distension and nausea, the patient could eat very little, and conservative treatment with nutritional support through intravenous administration and albumin was given. The pleural effusion and ascites were drained intermittently when abdominal distension and dyspnea became severe. The mean drainage volume of the pleural effusion was 1750 mL/week, and that of the ascites was 1125 mL/week. This conservative therapy was continued for 6 months, but there was no change in chylothorax and chylous ascites. The patient's albumin level decreased to 1.4 g/dL. Lymphoscintigraphy with technetium-99 m-labeled human serum albumin showed the tracer accumulation in the peritoneal space and absence of the thoracic duct (Fig. 1A).

Intranodal lymphangiography was performed with Lipiodol (Lipiodol Ultra Fluide, Laboratoires Guerbet, Aulnay-sous-Bois, France) from bilateral inguinal lymph nodes twice at an interval of 7 days, with the objective of

identifying the leakage site and using the therapeutic effect of Lipiodol. X-ray and computed tomography (CT) following intranodal lymphangiography showed Lipiodol leakage into the peritoneal cavity and reflux to the intestinal lymphatics, but the culprit lymphatic duct for this leakage was unclear (Fig. 1B, C). No Lipiodol was observed in the thoracic cavity. During both procedures, the thoracic duct was occluded at the ninth vertebral level (Fig. 1D). The conservative treatment was continued following lymphangiography, but there was still no improvement in chylothorax and chylous ascites.

It was decided to restore the patency of the interrupted thoracic duct. This procedure was performed with the patient under sedation using dexmedetomidine hydrochloride and local anesthesia using lidocaine. Intranodal lymphangiography with Lipiodol was first performed, and the cisterna chyli was identified. The cisterna chyli was punctured percutaneously and transabdominally with a 22-gauge, 20-cm Chiba biopsy needle (Angiotech;

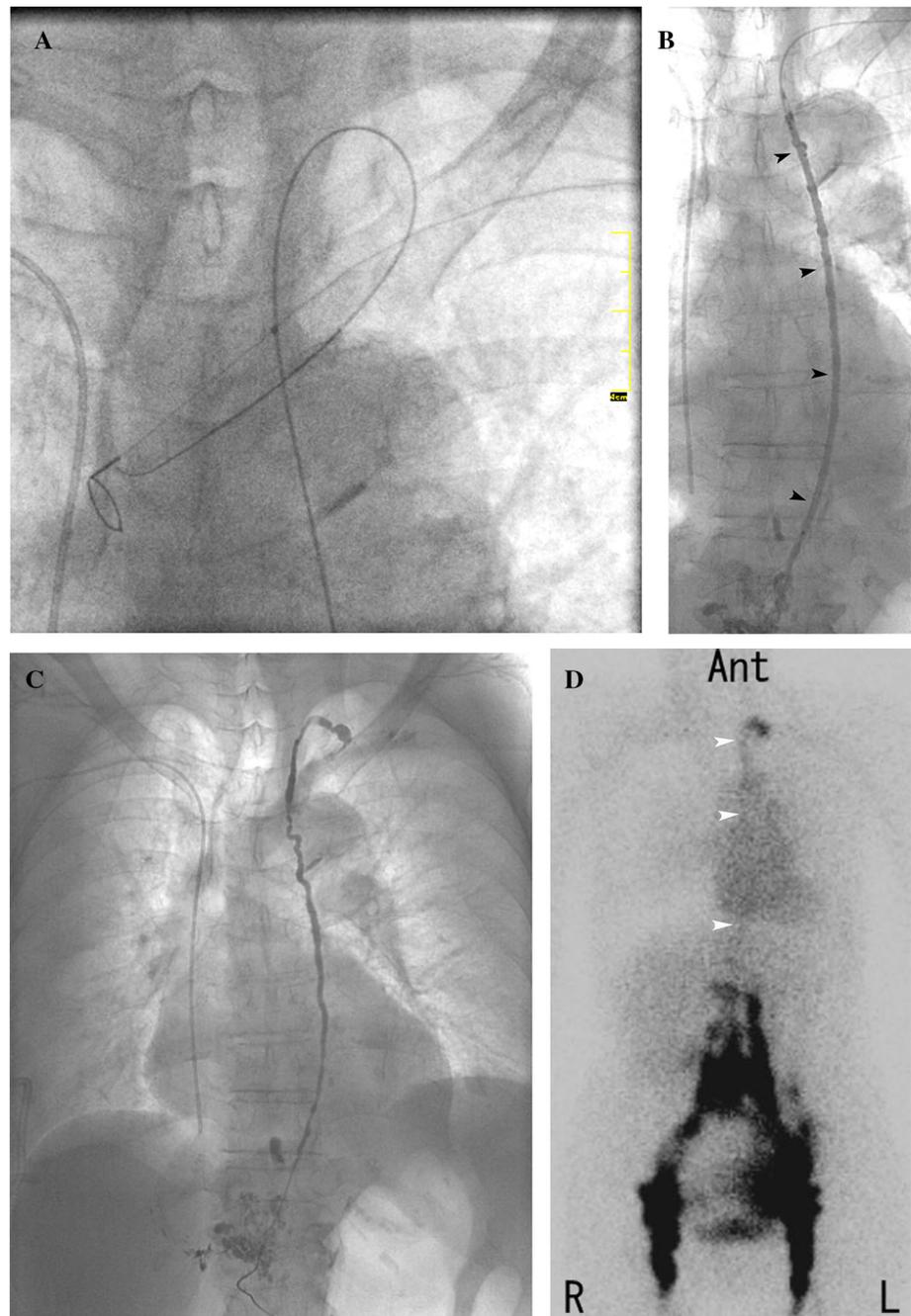
Fig. 1 An 82-year-old woman with chylothorax and chylous ascites **A** Lymphoscintigraphy with technetium-99 m-labeled human serum albumin shows tracer accumulation in the peritoneal space (black arrowheads) and absence of the thoracic duct. **B** Intranodal lymphangiography. Lipiodol leakage is observed (white arrows). **C** Computed tomography following intranodal lymphangiography shows reflux to the intestinal lymphatics (white arrowheads). **D** The thoracic duct is interrupted at the ninth vertebral level (black arrow)



Gainesville, FL, USA) under X-ray fluoroscopy. A 0.014-inch wire (Chevalier 14 Universal; FMD, Saitama, Japan) was passed via the needle through the cisterna chyli and advanced into the thoracic duct until it reached the site of occlusion. The wire was turned back at the occlusion site, but it was passed through the occlusion site by maneuvering the tip. The wire was advanced centrally into the thoracic duct and through the opening into the left venous angle until it reached the left brachiocephalic vein. The needle was then withdrawn, and a microcatheter (1.8-Fr Prominent Raptor, Tokai Medical Products, Aichi, Japan)

was inserted over the wire. A 4F 10-mm-diameter loop snare (Amplatz Goose Neck™, e3, Plymouth, MN, USA) was inserted via the left brachial vein. The tip of the wire that was advanced into the vein from the thoracic duct was trapped with the loop snare and drawn out of the body via a sheath deployed in the left brachial vein (Fig. 2A). A pull-through was thus formed between the percutaneous transabdominal route and the left brachial vein. A balloon catheter with diameter of 2.5 mm and length of 150 mm (Saberx, Cardinal Health Japan, Tokyo, Japan) was deployed from the left brachial vein into the thoracic duct,

Fig. 2 Percutaneous balloon thoracic duct plasty. **A** The micro-guidewire is passed through the obstruction in the thoracic duct and advanced from the thoracic duct into the vein. The wire is trapped by a loop snare and drawn out of the body through a sheath deployed in the left brachial vein, forming a pull-through between the percutaneous transabdominal route and the left brachial vein. **B** A balloon catheter of diameter 2.5 mm and length 150 mm (black arrowheads) is inserted from the left brachial vein into the thoracic duct, and balloon dilation of the stenotic region is performed at 15 atm. **C** Post-dilation transcatheter thoracic ductography. The thoracic duct is patent from the cisterna chyli to the venous opening. **D** Six months after thoracic duct plasty, lymphoscintigraphy with technetium-99 m-labeled human serum albumin shows that the thoracic duct (white arrowheads) is still patent, and there is no tracer accumulation in the peritoneal space



and the stenotic region was dilated at 15 atm (Fig. 2B). Post-dilation thoracic ductography showed that the thoracic duct was patent from the cisterna chyli to the venous opening (Fig. 2C).

From the day after thoracic duct plasty, it was no longer necessary to drain the pleural effusion or ascites because the abdominal distension and respiratory distress disappeared, and the volumes of pleural effusion and ascites decreased. Parenteral nutrition was stopped 10 days later, and the patient became capable of ingesting sufficient nutrition by mouth without fat restriction. On day 20 after thoracic duct plasty, the albumin level was improved to 3.0 g/dL. Six months later, lymphoscintigraphy with technetium-99 m-labeled human serum albumin showed that the thoracic duct was still patent, and there was no tracer accumulation in the peritoneal space (Fig. 2D). Although some pleural effusion and ascites persisted, drainage was not performed, and there was no recurrence of symptoms.

Discussion

A previous study reported that after thoracic duct embolization, abdominal swelling occurred in 6% of patients, and when lower-extremity edema and diarrhea were included, then 14.3% of patients were symptomatic [6]. Thoracic duct occlusion may cause lymph congestion. In this case, reflux to the intestinal lymphatics was confirmed. We therefore postulated that thoracic duct occlusion might have raised the internal pressure of the peripheral lymph ducts, causing refractory lymph leakage, and that restoring the patency of the occluded thoracic duct might reduce the internal pressure of the thoracic and abdominal lymphatic ducts, reducing the amount of leakage.

Two studies reported stent-graft placement in the thoracic duct [7, 8]. In both studies, placement was performed as treatment to block the flow of lymph into the lymphatic duct supplying the site of leakage by grafting. Unlike those previous reports, the objective in performing thoracic duct plasty in the present case was to release a blockage in the thoracic duct to reduce pressure in the peripheral lymphatic ducts, reducing the amount of leakage from these ducts.

A limitation of this case report is that the internal pressure of the thoracic duct was not measured before and after the procedure. One can thus only infer that thoracic duct plasty reduced the pressure in the lymphatic ducts and that the chylothorax and chylous ascites improved as a result. Even when surgical thoracic duct ligation is performed, chylous ascites and chylothorax are usually rare complications. Therefore, rupture of the lymph duct due to the blunt trauma may have been involved in the chylothorax and chylous ascites. Chylothorax is known to

occur as a result of blunt trauma, but, in the present patient, it was not possible to demonstrate a causal relationship between the bruising and the chylothorax and chylous ascites [9, 10].

Conclusions

Refractory chylothorax and chylous ascites improved with percutaneous balloon thoracic duct plasty to restore the patency of the occluded thoracic duct.

Compliance with Ethical Standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent This case report was approved by our institutional review board, and the requirement to obtain informed consent for inclusion in this study was waived. Informed consent for percutaneous balloon thoracic duct plasty was obtained from the patient before the procedure.

References

1. Itkin M, Kucharczuk JC, Kwak A, Trerotola SO, Kaiser LR. Nonoperative thoracic duct embolization for traumatic thoracic duct leak: experience in 109 patients. *J Thorac Cardiovasc Surg.* 2010;139(3):584–9 **discussion 9–90, Epub 2010/01/01.**
2. Nadolski GJ, Itkin M. Thoracic duct embolization for nontraumatic chylous effusion: experience in 34 patients. *Chest.* 2013;143(1):158–63 **Epub 2012/07/17.**
3. Baek Y, Won JH, Chang SJ, Ryu HS, Song SY, Yim B, et al. Lymphatic embolization for the treatment of pelvic lymphoceles: preliminary experience in five patients. *J Vasc Interv Radiol.* 2016;27(8):1170–6.
4. Hur S, Shin JH, Lee JJ, Min SK, Min SI, Ahn S, et al. Early experience in the management of postoperative lymphatic leakage using lipiodol lymphangiography and adjunctive glue embolization. *J Vasc Interv Radiol.* 2016;27(8):1177–86 **Epub 2016/07/01.**
5. Nadolski GJ, Chauhan NR, Itkin M. Lymphangiography and lymphatic embolization for the treatment of refractory chylous ascites. *Cardiovasc Intervent Radiol.* 2018;41(3):415–23 **Epub 2017/12/15.**
6. Laslett D, Trerotola SO, Itkin M. Delayed complications following technically successful thoracic duct embolization. *J Vasc Interv Radiol.* 2012;23(1):76–9 **Epub 2011/11/26.**
7. Dori Y, Keller MS, Rome JJ, Gillespie MJ, Glatz AC, Dodds K, et al. Percutaneous lymphatic embolization of abnormal pulmonary lymphatic flow as treatment of plastic bronchitis in patients with congenital heart disease. *Circulation.* 2016;133(12):1160–70 **Epub 2016/02/13.**
8. Chick JFB, Reddy SN, Murrey DA, Castle JC, Gemmete JJ, Saad WE, et al. Single-session endolymphatic thoracic duct stent-graft

- placement for recurrent idiopathic chylothorax. *J Vasc Interv Radiol.* 2017;28(7):1063–7 **Epub 2017/06/25.**
9. Pillay TG, Singh B. A review of traumatic chylothorax. *Injury.* 2016;47(3):545–50 **Epub 2016/01/19.**
 10. Apostolakis E, Akinosoglou K, Koletsis E, Dougenis D. Traumatic chylothorax following blunt thoracic trauma: two conservatively treated cases. *J Cardiac Surg.* 2009;24(2):220–2 **Epub 2009/03/10.**