Recurrence Chylothorax Caused by a Retained Guidewire in a Central Vein: A Case Report

Young-Do Jeon1,2, Eun Kyoung Kim1, Ji-Won Hwang1, Il Park1, Nam-Joon Kim1

1Division of Cardiology, Department of Medicine, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, 2Division of Cardiology, Department of Medicine, Iksan Hospital, Iksan, Korea

INTRODUCTION

A chylothorax results from leakage of lymphatic fluid, and is caused by obstruction or injury of the thoracic duct1). Nontraumatic chylothorax results from increased superior vena cava pressure due to malignancy, lymphangioleiomyomatosis, sarcoidosis, cirrhosis, tuberculosis, or thrombosis causing venous obstruction2,3). As the correction of underlying causes is the first and best treatment option for nontraumatic chylothorax, the causative factors should be evaluated.

The loss of a guidewire occurs very rarely during central venous catheterization (CVC)4-6). The guidewire may remain unnoticed in the vessel for a long time and may not induce specific symptoms6). However, it may cause vascular injury, cardiac injury, arrhythmia, or chronic venous thrombosis7). The development of a chylothorax following venous thrombosis caused by a retained guidewire after catheterization has not been reported. We report a case of recurrent chylothorax caused by chronic total central venous obstruction due to a retained guidewire after CVC.

CASE REPORT

A 74-year-old woman was admitted because of a femoral fracture. A large left pleural effusion was incidentally detected on preoperative chest radiography (Fig. 1). We performed diagnostic thoracentesis and drained milky-chylous fluid. Pleural fluid analysis revealed a white blood cell count of >1,000/µL (87% lymphocyte), glucose 124 mg/dL, protein 3,522 mg/dL, amylase 32 U/L, adenosine deaminase 11.5 IU/L, and triglyceride 295 mg/dL. The pleural fluid culture showed no growth. The pleural effusion was diagnosed as a chylothorax, although there was no history of chest trauma.

Chest computed tomography revealed the presence of a wire and chronic thrombus from the right internal jugular vein. Chest computed tomography revealed a guidewire that had remained in a central vein for 3 years, and the patient had extensive central venous thrombosis and a chylothorax. The guidewire and venous thrombus could not be removed because of high perioperative risk due to adhesions and chronic atrophic changes of the intravascular layer. The chylothorax was resistant to conservative treatment. Therefore, the patient was subjected to thoracic duct ligation and embolization. Subsequently, the chylothorax disappeared and the patient was discharged. Physicians should be especially aware of the adverse effects caused by a remnant guidewire and the need for simple chest radiography after CVC.

Key Words: Chylothorax, Central venous catheterization, Venous thrombosis

Fig. 1. Initial chest radiograph. Left pleural effusion and linear structure across the right lung were observed.
Fig. 2. Chest computed tomographic angiography. The retained wire and diffusely collapsed central vein with thrombus were observed. (A) The white arrows indicate the guidewire along the central vein. (B) Maximal intensity projection view: The black arrow indicates the chest tube. The white arrow indicates the guidewire and the black arrow indicates the chest tube.

vein to a lower extremity vein (Fig. 2A, B). Review of the initial chest radiographic image also confirmed the wire in the central vein. Two years prior, she was hospitalized in the intensive care unit of another hospital with ischemic colitis. At that time, CVC was performed via the subclavian vein, but the guidewire was not removed after the procedure. The wire remained in the vessel for 2 years between the right internal jugular and femoral vein. We determined that the chylothorax was caused by obstruction of a central vein by chronic thrombus. However, removal of the retained wire was dangerous because the thrombus chronically and totally obstructed a long segment of the central venous structure and was adherent to the vessel wall. Therefore, after conservative treatment for chylothorax, the patient was placed on anticoagulation therapy and discharged.

One year later, she revisited the Emergency Department with new-onset dyspnea. Physical examination was unremarkable, except for decreased breath sounds in the bilateral lower lobes. Chest radiography showed recurrent pleural effusion (Fig. 3). Examination of the pleural fluid also revealed a chylothorax (total white blood cell count >1,000/µL (78% lymphocytes), glucose 123 mg/dL, protein 3,518 mg/dL, amylase 36 U/L, cholesterol 59 mg/dL, and triglyceride 635 mg/dL). Octreotide was administered to reduce lymph flow, and total parenteral nutrition and water restriction were started. Percutaneous catheter drainage was continued for 2 weeks, but the amount of chylothorax did not decrease. Magnetic resona-
nce lymphangiography revealed collapse of the thoracic duct and central lymphatic system. Leakage of lymphatic fluid was also suspected (Fig. 4A). In addition, imaging showed obstruction of the distal thoracic duct with abnormal collateral channels in both the hilum and apex of the lung (Fig. 4B, C). She underwent thorascopic-thoracic duct ligation and embolization of the thoracic duct and collateral channels. The guidewire and venous thrombus were not removed because of high perioperative risk from adhesions and chronic atrophic intravascular changes. The chylothorax was not observed at the 6-month follow-up after discharge, and the patient is followed as an outpatient in good general condition.

**DISCUSSION**

Chylothorax is a life-threatening complication of chronic venous obstruction. When chylothorax is diagnosed and is resistant to conservative treatment, the possibility of extensive venous occlusion should be suspected, and surgical treatment should be considered. Here, we present a case of recurrent chylothorax caused by chronic total obstruction of the long segment of the central vein due to a retained guidewire. Loss of guidewire occurs very rarely during CVC. However, it can cause severe complications similar to those reported in our case, unless the wire is removed immediately. Remnant wire not only occupies the vessel space but also obstructs venous circulation, leading to complications including arrhythmia, cardiac or vascular perforation, looping of wire, and thrombus. If thrombus in the central vein is left untreated, unexpected adverse events may occur, including chylothorax. Early detection of a retained wire following thrombus formation is key to complete resolution after wire removal and anticoagulation.

The clinical success of chylothorax treatment depends on the underlying cause. Tamai et al. reported a case of chylothorax associated with thrombosis in the superior vena cava that was resistant to thoracic duct ligation. A stent was implanted in the superior vena cava, after which the chylothorax disappeared. Manghat et al. reported a case of chylothorax secondary to extensive central venous thrombosis. They performed catheter-directed thrombolysis. In these cases, the venous thrombus was discovered in a relatively acute phase compared with that in our case. In our case, conservative medical treatment failed to prevent a chylothorax. Therefore, thoracic duct ligation and embolization were selected as the first treatment option. However, because the retained guidewire and chronic venous thrombosis from the jugular vein to the femoral vein could not be removed, thoracic duct ligation and embolization were only palliative for prevention of recurrent chylothorax.

By reviewing this case, we hope that physicians will gain sufficient awareness of the long-term adverse events of guidewire placement and the need for early detection of this complication using simple chest radiography after CVC.

**Conflicts of Interest Disclosures:** The researchers claim no conflicts of interest.

**REFERENCES**