Laboratory Note

The purpose of this newsletter is to update our readers with the evidence-based management of certain Head & Neck disease presentations. In this issue we shall focus on chyle fistula.

The Yale Larynx Laboratory was founded by John A. Kirchner in 1967. Since 1975 this laboratory has been in continuous operation under the direction of Clarence T. Sasaki, the Charles W. Ohse Professor and has been funded by the National Institutes of Health and by endowments of grateful patients.

Chyle Fistula

Clarence T. Sasaki MD, Jacob Tower MD

Case Presentation

A 45-year-old man presented with a history of papillary thyroid cancer that had been treated in the past with a total thyroidectomy, central neck dissection, and radioactive iodine. He was found on post treatment surveillance to have a suspicious lymph node adjacent to the left carotid artery. After an equivocal FNA, he elected to undergo a redo left neck dissection for purposes of definitive diagnosis and treatment. In the course of the left neck dissection, the contents of the carotid sheath were identified and preserved. Suspicious, firm, tissue was discovered posterior to the carotid artery and extending inferior and deep to the clavicle. In the course of excision, an intraoperative chyle leak was encountered. The area was oversewn, and with a Valsalva maneuver there was no additional flow of chyle. A suction drain was placed at the end of the case, and the patient was discharged home.

On postoperative day 2, the patient presented with milky white output from his neck drain, concerning for a chyle fistula. His drain output had been increasing over time as well, initially with 100cc on postoperative day 1, but increasing to 210cc total for postoperative day 2. The milky drain fluid was sent for a triglyceride level which was found to be 1,380 mg/dl. He was admitted to the hospital and started on a zero fat diet; however, by postoperative day 4 the patient’s drain was now putting out 1,645cc of chyle over a 24-hour period (Fig. 1). At this time he also suffered a syncopal episode, likely as a result of new hyponatremia and hypovolemia. He was started on IV fluids and 100 mcg of octreotide, three times daily. His electrolyte disturbances improved, but his chyle fistula showed no signs of resolution with conservative management.
On postoperative day 6, the patient was taken back to the OR for left neck exploration and closure of the chyle fistula. After opening the neck, chyle was found to be draining from the left upper mediastinum, behind the carotid artery and deep below the clavicle. The area of the leak was oversewn, and a sternothyroid muscle flap was rotated over the area, inset with vicryl suture, and then covered with Evicel fibrin sealant. Again, a drain was left in place at the conclusion of the case. Despite this procedure, the patient continued to have a high volume chyle leak with maximum 24 hour output of 2,000cc. On postoperative day 2 from the neck exploration, the patient underwent an attempted thoracic duct embolization with Interventional Radiology, but this was unsuccessful. At this time, Thoracic Surgery was consulted for VATS ligation of the thoracic duct, and the following day (postoperative day 3 from neck exploration, and postoperative day 8 from the neck dissection) the patient underwent bilateral chest tube placement for drainage of a left chylothorax, and right VATS thoracic duct ligation. The patient did well postoperatively and over the remainder of his hospitalization his neck drain and chest tubes were removed. He was discharged home on a low fat diet.

Discussion

Chyle is composed of lymphatic fluid and chylomicrons from the gastrointestinal system. The chylomicrons are formed from the breakdown products of long-chain fatty acids and absorbed into the lymphatic system in the intestines. By comparison, short- and medium-chain fatty acids are absorbed directly by the intestinal mucosa, thus bypassing the lymphatic system. Chyle is transported into the venous system via the thoracic duct, a large lymphatic channel that ascends from the abdomen, and through the thorax, before terminating in the neck (Fig. 2). Chyle fistula is caused by inadvertent injury to the thoracic duct, usually as a complication of neck dissection. The thoracic duct is a thin-walled structure, typically 2-5 mm in diameter, and terminates in a cervical lympho-venous junction. There is significant variability around the termination of the thoracic duct with regard to the point of entry into the venous system. Most frequently, the duct terminates in the left internal jugular vein, or in the angle between the IJV and subclavian vein. Less commonly, the duct drains directly into the subclavian vein. It is estimated that approximately 1-5% of the population has a right-sided termination.
The delicacy of the thoracic duct and significant variation in its anatomy puts it at risk for injury during lower neck dissection. Intraoperative dissection of the thoracic duct is not recommended because the risk of iatrogenic injury is high.

In one study of 472 patients undergoing neck dissection involving the level IV compartment, the incidence of chyle fistula was 4.7%. The presence of a metastatic lesion around the junction of the internal jugular and subclavian increased the likelihood of a chyle fistula complication.

A chyle leak may be detected intraoperatively or postoperatively. When discovered intraoperatively, the thoracic duct should be identified and ligated. This may be difficult due to patient fasting in preparation for surgery and thoracic duct collapse. A Valsalva maneuver or manual abdominal compression may assist in locating the leaking thoracic duct. Postoperatively, high increases in drain output, creamy or milky appearance, or a palpable supraclavicular fluid collection raises suspicion for a chyle leak. In equivocal cases, sending the drain fluid for triglyceride levels will aid in the diagnosis (triglyceride level >100 mg/dl or greater than the serum triglyceride level is suggestive of a chyle leak). Prompt diagnosis and treatment is essential to prevent further known complications including electrolyte imbalances, malnutrition, and wound healing complications such as skin flap necrosis.

Conventional treatment of chyle fistula begins with conservative medical management including pressure dressings, closed suction drainage, and nutritional modification (Fig. 3). Such modifications include zero fat diet, low-fat diet, medium chain fatty acid diet, or total parenteral nutrition, which bypasses the lymphatic system completely. If the chyle fistula does not respond to these measures, chyle production can be decreased with adjunctive medications. Octreotide decreases chyle production via reduction of gastric, pancreatic, and intestinal secretions, and constricts smooth muscle in splanchnic and lymphatic vessels to decrease lymph production and flow, respectively. Octreotide is given subcutaneously and has been demonstrated to decrease morbidity, length of stay, and need for surgical intervention related to chyle fistula. From initiation of therapy to chyle leak cessation, octreotide treatment duration ranged in one prospective study from 5 days for low output leaks (<500cc/day) to 7 days for high output leaks (>500cc/day).
Surgical exploration should be considered if initial measures are ineffective, and especially when fistula outputs are >500cc/day to 1000cc/day, usually after a trial of up to 5 days of medical management. Some authors advocate for having a patient ingest a fatty diet prior to surgery to stimulate chyle production and identification of the leak intraoperatively. The leaking duct can be ligated, covered with a muscle flap, or treated with sclerosing or adhesive agents. A low suction drain must be placed at the conclusion of the case.

If surgical exploration fails, cannulation and selective embolization of the thoracic duct can be performed with the assistance of interventional radiology. Alternatively, thoracoscopic ligation can also effectively address the leaking fistula via a right-sided approach through which the thoracic duct is ligated between the aorta andazygous vein at the level of the supradiaphragmatic hiatus (Fig 4).

References


