

# MANAGEMENT OF POSTOPERATIVE CHYLOTHORAX IN A PATIENT WITH CARCINOMA OF THYROID AND LYMPHADENOPATHY

- A Case Report -

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## Abstract

Chylothorax is a rare but serious complication following neck dissection with an incidence of 0.5% - 2%<sup>1</sup>. Because of the rarity of chylothorax, surgeons are unfamiliar with its early signs which allow a prompt diagnosis and effective management. Most cases reported in the literature are associated with a concurrent external chyle leakage, occurring either during or after surgery. We report a case of chylothorax without concurrent external chyle leakage, which occurred following neck dissection and mediastinal lymphadenopathy, for thyroid cancer.

## Introduction

Chyle is the lymphatic fluid enriched with fat and its digestive products, absorbed in the intestines, collected and transported by the thoracic duct into the circulation. Chylothorax is characterized by pleural fluid with a turbid or milky white appearance due to a high lipid content, most common source being from disruption of the thoracic duct. Leakage of chyle and lymph leads to significant loss of essential proteins, immunoglobulins, fat, vitamins, electrolytes and water. While therapeutic thoracentesis provides relief from respiratory symptoms, the nutritional deficiency continues to persist or deteriorate unless definitive therapeutic measures are instituted to stop leakage of chyle into the pleural space.

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## Case History

A 17 year female weighing 45 kg presented with swelling at the anterior aspect of neck. She was diagnosed as carcinoma of thyroid and posted for total thyroidectomy with bilateral modified neck dissection and superior mediastinal lymphadenectomy under general anesthesia.

Her medical history was unremarkable. All routine investigations were within normal limits. Her cardiovascular and respiratory systems were unremarkable.

The intraoperative course of six hours was uneventful. She was reversed and extubated on the operating table and after observation overnight in the ICU shifted to ward.

Next morning, she developed sudden respiratory distress with facial and neck puffiness and fall of blood saturation of oxygen to 75 to 80% with central cyanosis. Larygосcopy was done and there was intense oral and tongue edema, An endotracheal tube of 6.0 mm internal diameter could be passed with difficulty. The tube placement was confirmed and connected to an AMBU bag with an oxygen source. At the same time the surgical sutures at side of neck were cut to evacuate around 250 ml of serosanguinous fluid. Pulse rate was 120 per minute and blood pressure 80 mm of Hg systolic with a central venous pressure of 5 cm of water. Respiration was shallow and rapid. Arterial blood gas analysis was a PaO<sub>2</sub> 54 mmHg and PaCO<sub>2</sub> 50 mmHg with a pH of 7.24.

The patient was shifted to the ICU and sedated with morphine and midazolam and connected to the ventilator SIMV mode with a PEEP of 5 and pressure support of 15 cmH<sub>2</sub>O. Inj calcium gluconate 10 cc and hydrocortisone 200 mg were given intravenously. Fluid resuscitation was done with 1000 ml of lactated ringer until the central venous pressure increased to 8 cms and systolic blood pressure to 100 mmHg. Arterial blood gases revealed a PaO<sub>2</sub> 96 mmHg and PaCO<sub>2</sub> 40 mmHg with a FIO<sub>2</sub> of 0.5. Chest radiography revealed mediastinal widening with pleural effusion.

An immediate bilateral chest drainage tube was put with an evacuation of chylous fluid. Patient remained hemodynamically stable throughout and after the procedure. There was constant outpour of serous

discharge from the surgical site. A tracheostomy was done and octreolide 100 mcg subcutaneously 8 hourly started. By evening the edema and facial puffines started to decrease.

By the third day, as the patient was hemodynamically stable, it was decided to go for a surgical exploration of the wound. The thoracic duct leak site could not be identified intraoperatively so the procedure was abandoned and wound closed. On the 4<sup>th</sup> day total parenteral nutrition was started.

Over the next few days the chyle from the chest drains decreased and the facial edema gradually resolved and she could sit up in bed by the seventh day. She was subsequently shifted to the ward and discharged from the hospital on the twelfth day. On follow up she was healthy with no complaints.

## Discussion

The accidental damage of thoracic duct, as happened in this case, leads to leakage of chyle into the pleural space which sometimes presents so acutely as to create a life threatening situation. Trauma to the thoracic duct is the commonest cause of chylothorax. Among traumatic chylothoraces, iatrogenic causes constitute the majority<sup>2</sup>. The commonest cause is thoracic surgery, particularly involving dissection of the mediastinum. In the past, the mortality due to chylothorax was in excess of 50%<sup>3</sup>. Currently, the morbidity and mortality have improved due to the more aggressive management strategies adopted.

Introduction of aggressive therapeutic measures to reverse the adverse effects of chyle loss has led to the lowering of mortality rates for post-traumatic chylothorax<sup>4</sup>. Usually, a latency period of 2-7 days exists between the time of injury and clinical evidence of chylothorax if the injury is not a major one. This is because lymph accumulates in the posterior mediastinum until the mediastinal pleura ruptures, usually on the right side at the base of the inferior pulmonary ligament.

The dissection of mediastinal lymph nodes in relation to the thoracic duct can lead to chylothorax. The mode of injury in our case was clearly iatrogenic. Laceration of the thoracic duct during catheterization of the subclavian vein is another possibility to be

considered in this case. Extensive venous thrombosis complicating central venous catheterization has been reported in bilateral chylothorax and chylopericardium<sup>5</sup>.

Large chylothoraces commonly lead to hypovolemia due to a sudden loss of large volume. The rapidity with which decompensation occurs depends on the amount, rate, and duration of chyle loss. In the early stages, the patient may not demonstrate clinical symptoms or signs of loss of chyle but later may exhibit clinical features of severe malnutrition. Hyponatremia, acidosis, and hypocalcemia are the most commonly electrolyte abnormalities and should always be corrected promptly so as to improve outcome<sup>6</sup>.

Conservative management is currently complemented with various drugs that decrease the chyle leakage (somatostatin and analogs such as octreotide, heparin, and etilefrine). Octreotide in

particular, because of its easy availability and safety in postoperative patients, has been used extensively to reduce intestinal chyle production and secondarily reduce chyle leak<sup>7</sup>.

With conservative management of chylothoraces, mortality after esophagectomy approaches 50%, whereas with active surgical intervention incidence drops to about 10%<sup>8</sup>. It can be further reduced if full knowledge of the process of chylothorax and its metabolic and nutritional complication are available to the clinician. Conservation management has been reported in various previous articles but we recommend an aggressive surgical therapy especially for post-traumatic or post-surgical chylothorax as first line of approach, supplemented with pharmacological measures with prompt correction of the metabolic and nutritional derangements.

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