A Contemporary Review of Chylothorax

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ABSTRACT

Objectives. This review will focus on anatomical and aetiological factors as well as the conservative and operative therapy of chylothorax.

Data Source. A Pubmed search for studies pertaining to the aetiology and/or treatment published in the English language from 1960 to 2007.

Study Selection. Studies presenting case reports, series, observational and/or retrospective studies, and those with unique issues pertaining to chylothorax were reviewed independently by both authors. Studies that were selected by both authors contain most clinically relevant data.

Results. Chylothorax is caused by injury or obstruction of the thoracic duct or its main tributaries leading to chyle accumulation in the pleural space. It most commonly occurs from trauma or malignancy, but other causes have been described. Although chylous effusions are rare, they have serious clinical consequences including cachexia and immunodeficiency. There are no evidence-based guidelines to assist in the management of this disease.

Conclusions. A prompt diagnosis is needed to start treatment of the underlying cause. Treatment can be divided into conservative and surgical interventions. There are no evidence-based guidelines to assist in the management of this disease. Initial conservative therapy includes intercostal decompression of the pleural effusion along with nutritional support in the form of total parenteral nutrition, and reduction of chylous formation with somatostatin. Surgical interventions include thoracic duct ligation, pleuroperitoneal shunt and percutaneous embolisation. [Indian J Chest Dis Allied Sci 2008; 50: 343-351]

Key words: Chyle, Thorax, Pleural effusion, Injury, Lymph, Treatment

INTRODUCTION

Chyle in the pleural space was first described by Bartolet in 1633 and since its initial description, numerous causes have been well described.¹ The pleural fluid in chylothorax consists of chylomicrons and very low-density lipoproteins. It results from an anatomical disruption of the thoracic duct and/or a major lymphatic contributory. It was not until 1948 that Lampson² reported the first successful treatment of chylothorax by supradiaphragmatic ligation of the thoracic duct. Although cylothorax accounts for a small proportion of clinical pleural effusions, prompt recognition is needed to avoid malnutrition, immunodeficiency, and fibrothorax. Management requires the physician to individualise the work-up, partly because of diversity in the pathogenesis and anatomy.

ANATOMY

Chyle passes from the intestinal lymphatics to cisterna chyli and then through the thoracic duct to eventually empty into the venous system. The thoracic duct begins at the cisterna chyli near the T-12 vertebra and ascends through the aortic hiatus of the diaphragm on the anterior surface of the vertebral body between the aorta and the azygos vein into the posterior mediastinum. At the level of the fifth thoracic vertebra, it then crosses to the left of the vertebral column and ascends behind the aortic arch to the left of the subclavian artery adjacent to the mediastinal pleura. At the level of the transverse process of the seventh cervical vertebra, the duct turns laterally and runs anterior to the vertebral and thyrocervical arteries and the sympathetic trunk. Passing behind the carotid sheath, it descends anterior to the origin of the left subclavian artery and terminates near the junction of internal jugular and subclavian veins. A bicuspid valve at the lymphovenous junction prevents the reflux of blood into the duct. The duct itself has numerous valves throughout its length. However, in up to 50% of individuals, the route of thoracic duct is anomalous and unpredictable, thus making it more susceptible to damage during surgical...
and chyle flow. In addition to starvation, intestinal acetylcholine all increase thoracic duct contraction splanchnic and vagal stimulation. Serotonin, related to response of the duct wall smooth muscle to over 100 mL/h postprandial. Thoracic duct flow is widely from as low as 14 mL/h in the fasting state to cholesterol. Flow through the thoracic duct can vary high in neutral fats and fatty acids, but low in cellular sediment on the bottom. Chyle is noted to be standing with a fat-rich portion on the top and a specific gravity of greater than 1.012 and will settle on with Sudan III dye. Chyle is also alkaline with a analysis reveals protein and fat globules that stain either extrinsic compression or infiltration of the two major categories—surgical and medical causes. The pathogenesis of chylothorax can be divided into extrinsic compression or infiltration of the mediastinum. Operations on the heart, mediastinum, operations in the neck, such as radical neck dissections, and placement of catheters in the superior vena cava for hemodynamic monitoring may result in chylothorax. Chylothorax is the most common cause of pleural effusion in neonates, and it usually appears spontaneously. Systemic conditions, such as Behcet’s amyloidosis, sarcoidosis, heart failure, and nephritic syndrome, compose another group of miscellaneous conditions associated with chylothorax. Jaffe-Campanacci syndrome, which includes disseminated non-ossifying fibromata has also been reported to be associated with chylothorax. Gorham’s syndrome is a rare disease associated with progressive osteolysis along with intraosseous angiomatosis of lymphatics and blood vessels. Up to 17% of patients develop chylothorax as a complication in this disease. Pulmonary lymphangioleomyomatosis is characterised by proliferation of immature smooth muscle throughout the peribronchial, perivascular, and perilymphatic region. It occurs primarily in women of reproductive age and the perilymphatic proliferation of smooth muscle results in lymphatic obstruction and chylothorax in up to 50% of the patients. This condition may at times be present as a part of the syndrome of pulmonary tuberous sclerosis. A chylothorax may develop acutely without an underlying cause and is referred to as spontaneous chylothorax. Idiopathic chylothorax is uncommon and comprises cases where no underlying cause can be found. A sudden increase in duct pressure from coughing, especially after a heavy meal when the duct is engorged, may be a contributory factor. Despite detailed work-up the cause of chylothorax may not be established in some instances. Most of the cases of chylothorax are unilateral, but bilateral chylothorax has also been reported.

**PHYSIOLOGY**

The word “chyle” comes from the Latin word meaning “juice” and is applied to the lymph of intestinal origin. Ingested fats are transported by chyle into the venous blood via the thoracic duct. In the fasting state, chyle is usually clear, owing to a low fat content. Both the protein content and the volume are also diminished. After ingestion of a fatty meal, chyle assumes its characteristic “milky” appearance. It is estimated that up to 60% of ingested fat passes into the lymphatics.

Between 1,500 to 2,500 milliliters of chyle is normally emptied into the venous system daily. The protein content of chyle is more than 3 gm/dL, and the electrolyte composition of chyle is similar to that of serum. Significant losses of chyle may result in severe nutritional depletion, especially hypoproteinemia. The chyle formed in the cisterna chyli is an odourless, white, opaque liquid. Analysis of chyle reveals approximately 90% lymphocytes and a white cell count of 2,000 to 10,000 cells per cubic millimeter, which generally renders this fluid bacteriostatic. It is believed that most of the lymphocytes in chyle are T-lymphocytes. Chemical analysis reveals protein and fat globules that stain with Sudan III dye. Chyle is also alkaline with a specific gravity of greater than 1.012 and will settle on with Sudan III dye. Chyle is also alkaline with a specific gravity of greater than 1.012 and will settle on with Sudan III dye.

**PATHOGENESIS**

The pathogenesis of chylothorax can be divided into two major categories—surgical and medical causes (Table). Chylothorax from medical causes results from either extrinsic compression or infiltration of the thoracic duct, that causes an increase in intraductal pressure. This increased pressure promotes the formation of dilated collateral channels that eventually drain into the pleural space. The most common cause of chylothorax is neoplasm, which is responsible for more than 50% of cases. The most common malignancy leading to this condition is lymphoma, which leads to chylothorax by compressing or invading the thoracic duct or obliterating the lymphatics after radiation therapy. Obstruction of the thoracic duct by lymphoma or bronchogenic carcinoma tends to cause a right-sided chylothorax when the lower portion of the duct is involved, whereas a left-sided chylothorax results when the upper portion of the thoracic duct is involved. Chylothorax in the setting of malignancy below the diaphragm invariably indicates metastasis.

The other leading cause of chylothorax are surgery and trauma. Although any penetrating injury that disrupts the thoracic duct would produce chylothorax, even severe coughing and straining have reportedly caused this condition. Surgical procedures on the heart, mediastinum, operations in the neck, such as radical neck dissections, and placement of catheters in the superior vena cava for hemodynamic monitoring may result in chylothorax. Chylothorax is the most common cause of pleural effusion in neonates, and it usually appears spontaneously. Systemic conditions, such as Behcet’s amyloidosis, sarcoidosis, heart failure, and nephritic syndrome, comprise another group of miscellaneous conditions associated with chylothorax.

Jaffe-Campanacci syndrome, which includes disseminated non-ossifying fibromata has also been reported to be associated with chylothorax. Gorham’s syndrome is a rare disease associated with progressive osteolysis along with intraosseous angiomatosis of lymphatics and blood vessels. Up to 17% of patients develop chylothorax as a complication in this disease. Pulmonary lymphangioleomyomatosis is characterised by proliferation of immature smooth muscle throughout the peribronchial, perivascular, and perilymphatic region. It occurs primarily in women of reproductive age and the perilymphatic proliferation of smooth muscle results in lymphatic obstruction and chylothorax in up to 50% of the patients. This condition may at times be present as a part of the syndrome of pulmonary tuberous sclerosis. A chylothorax may develop acutely without an underlying cause and is referred to as spontaneous chylothorax. Idiopathic chylothorax is uncommon and comprises cases where no underlying cause can be found. A sudden increase in duct pressure from coughing, especially after a heavy meal when the duct is engorged, may be a contributory factor. Despite detailed work-up the cause of chylothorax may not be established in some instances. Most of the cases of chylothorax are unilateral, but bilateral chylothorax has also been reported.
for example, Kaposi’s sarcoma has been reported to result in bilateral chylothorax. Chylothorax and chyloperitoneum have also been reported in patients with nephritic syndrome, malignancies such as Wilms’ tumor, gall-bladder carcinoma, uterine cancer, stomach cancer, lymphoma, retroperitoneal surgery, hypothyroidism, sarcoidosis, yellow nail syndrome, idiopathic, primary lymphatic dysplasia, lymphangiomatosis, and pancreatitis.

CLINICAL FEATURES AND DIAGNOSIS

The usual presenting symptom is dyspnoea due to accumulation of pleural fluid. Chest pain and fever are uncommon because chyle is not irritating to the pleural surface. Traumatic chylothorax usually develops within two to ten days post injury. In non-traumatic chylothorax, the onset of symptoms is more insidious. Spontaneous chylothorax may rarely present as a sudden neck mass. The severity of symptoms is related to the rate of accumulation of chyle and the size of the pleural effusion.

The more serious sequelae of chylothorax are malnutrition, weakness, dehydration, metabolic acidosis, and compromised immunologic status due to the loss of chyle, which is rich in proteins, fats, electrolytes, bicarbonate, lymphocytes, and fatsoluble vitamins. Prolonged chylothorax may be associated with reversible T-cell deficiency. Hypoalbuminemia and lymphopenia secondary to prolonged loss of chyle increase the risk of systemic bacterial and viral infections. There is a good correlation between rate of chyle loss, operative intervention, and survival.

Chyle is often suspected only after a thoracentesis. Chyle is distinctively white, odourless, and milky in appearance. The diagnosis of chylothorax is established by measuring triglyceride levels in the pleural fluid. If triglycerides are greater than 110 mg/dL, the diagnosis is probably chylothorax; conversely, if the level is less than 50 mg/dL, chylothorax is unlikely. When levels are between 50 to 110 mg/dL, lipoprotein analysis should be performed. Chylomicrons in the fluid establish the diagnosis of chylothorax. Milky or creamy pleural fluid can also be associated with a condition called pseudochylothorax. In this condition, usually associated with chronic diseases, such as tuberculosis, turbidity is the result of high levels of cholesterol or lecithin-globulin complexes and not chylomicrons. The effusion is usually of long-standing duration and may cause thickened and even calcified pleura. The presence of cholesterol crystals in the fluid is diagnostic of pseudochylothorax, and chylomicrons are never present on lipoprotein analysis. Both chylous and pseudochylous fluid remain opaque after centrifugation, but the turbidity of chylous fluid clears out on addition of two millilitres of ethyl ether. Thus, whenever there is any doubt, the fluid should be analysed for chylomicrons.

Another useful test is the ingestion of lipophilic dye or radio-labeled triglyceride (131I-triolein). Presence of the dye colour in the fluid within one hour or detection of high radioactivity in the pleural fluid after 48 hours confirms the presence of chylothorax. Lymphangiography may be helpful in defining the site of chyle leak or obstruction or penetrating trauma in spontaneous chylothorax and in lymphangiomatous malformations. Lymphangiography has also demonstrated a therapeutic role in assisting occlusion of the post-operatively damaged lymphatic vessel. This may occur from an inflammatory granulomatous reaction by the lipiodol dye during extravasation.

MANAGEMENT

Initial management is determining the aetiology. Surgical causes are more obvious. As lymphoma is the most common cause of chylothorax in the nonsurgical setting, a computed tomography (CT) of the chest and abdomen should be performed to evaluate mediastinal and para-aortic lymph nodes. Primary treatment must be directed at the underlying cause. In most instances surgical therapy should be pursued only after failure of conservative therapy. However, the timing of surgical management does not have consensus.

Conservative Therapy [Figure 1]

The initial approach to management of chylothorax involves chest tube drainage of the pleural space. Continuous suction drainage helps to relieve the pressure of chyle on the lungs, re-expands the partially collapsed lungs, obliterates the pleural space, and permits an accurate measurement of chyle production. Lung re-expansion is sometimes hindered by formation of a fibrinous membrane around the lung, which may necessitate surgical intervention. The rate of chyle leakage can be actually measured and recorded, if the chest tube is in place. Chest tube drainage of less than 500 mL during the first 24 hours after complete oral intake cessation and total parenteral nutrition may predict successful conservative treatment.

Because up to three liters of chyle may drain daily, large amounts of fluid, electrolytes, fat, protein, and lymphocytes may be lost leading to severe nutritional depletion and an immunodeficiency state. Careful monitoring of chyle output and replacement of daily losses are essential, as is monitoring of the patient’s weight, serum albumin, total protein, absolute lymphocyte count, and electrolyte levels. A non-fat, high-protein, high-calorie diet will produce some reduction of chyle flow. Administration of medium-chain triglycerides (MCTs) as a source of fat is invaluable. The MCTs are absorbed directly into the portal system rather than the intestinal and thoracic...
lymphatics. With the use of MCTs, not only is the nutrition satisfactorily maintained, but thoracic duct flow is minimised to promote healing of the leak. It has been suggested that in the selection of MCTs, trioctanoin (C8:0) may be the preferable MCT substrate for these patients.\(^{55}\) The MCT diets have met with variable success in the treatment of chylothorax. This is because: (i) any oral enteral feeding increases lymph flow,\(^{56}\) and (ii) intestinal triglycerides are derived from both endogenous and exogenous sources.\(^{57}\) If drainage remains unchanged, parenteral alimentation should be started. Others have recommended stopping oral feeding and initiating total parenteral nutrition (TPN) at the time of diagnosis.\(^{5}\) Comparison of enteral versus parenteral nutrition in the setting of chylothorax shows that the thoracic duct closure occurs faster with TPN.\(^{58}\)

If there is imminent nutritional deterioration, surgical intervention is indicated. If a patient is on mechanical ventilation, adding a positive end-expiratory pressure would lead to an increase in intrathoracic pressure, help approximate the two intrapleural surface and potentially decrease chyle leakage.\(^{59}\) Thomson and Simms\(^{60}\) have reported successful reinfusion of chyle drained from the chest tube using blood filters and a volumetric pump, but anaphylactic reactions have been reported after intravenous transfusion of chyle.\(^{61}\) Somatostatin is an inhibitor of gastric, pancreatic, and intestinal secretions, thereby helping to keep the gastrointestinal tract empty, which in turn decreases the chyle production.\(^{62}\)

Octreotide is an analog of somatostatin and has been used in conjunction with other modalities (i.e., TPN, effusion drainage) in conservative management of various aetiologies of chylothorax.\(^{63-65}\) Because of octreotide’s longer half-life, it offers a subcutaneous as well as intravenous route of administration. The support for octreotide is limited to case reports and small series, making the therapeutic effectiveness of octreotide difficult to differentiate from spontaneous improvement or other concurrent treatments. Due to the lack of existing evidence, we cannot recommend octreotide as a first-line agent at this time.

Treatment of the underlying cause may be helpful, especially in patients where chylothorax is secondary to lymphoma, where radiotherapy to the mediastinum is given to resolve the tumour mass. Mediastinal tumours associated with chylothorax, such as lymphangiomas, also respond to radiotherapy.\(^{66}\)

The duration of the conservative management is
not firmly established and must be related to pathogenesis, underlying comorbid illness, and institutional experience. Operative intervention in chylothorax should be considered when: (i) average daily loss has exceeded 1,500 milliliters per year of age in children for a five-day period; \(^5\) (ii) chyle flow has not diminished over 14 days; (iii) nutritional complications appear imminent; or (iv) accumulation of chyle is continuous despite chest tube drainage.

Chylothorax associated with sarcoidosis will respond very well if the patient is treated with steroids. Patients with Gorham’s syndrome and chylothorax should be treated with a pleuroperitoneal shunt or thoracic duct ligation.\(^2\) Pulmonary lymphangiomatosis can be treated with hormonal manipulation targeting high-affinity, low-capacity progestin-binding sites.\(^{67, 68}\) In a meta-analysis of 30 cases of lymphangioleiomyomatosis treated with various hormonal manipulations, it was concluded that the administration of progesterone, oophorectomy, or both results in improvement or stabilisation of the disease.\(^6\) Lung transplantation may be the ultimate answer to this problem.\(^7\)

**Surgical Therapy [Figure 2]**

Two weeks is often used as the limit for resolution by conservative management,\(^45,52\) but there are no large controlled studies to confirm this recommendation. It is appreciated that patients draining more than 1,000 millilitres per day may benefit from earlier (5-7 days) surgical intervention\(^7\) because of a higher mortality with increased output.

Failure of conservative treatment in cases of surgical chylothorax requires a surgical intervention for definitive management. Lampson\(^2\) first demonstrated that chylothorax could be controlled by ligation of the thoracic duct. Pre-operative administration of lipophilic dye (e.g., Evans blue or cream) helps to locate the site of lymphatic leakage during the procedure. The thoracic duct is then identified, isolated, and ligated just above the aortic hiatus between T-8 and T-12.\(^7\) After ligation, there is usually some obstruction to lymph flow distal to the ligated site until new collateral channels are formed (2 to 3 weeks).\(^7\) An abdominal approach to ligate the thoracic duct is an alternative in patients where thoracic approach is not feasible.\(^7\) Others have described a posterior extra-pleural approach to ligation of the thoracic duct.\(^7\) Most recently, video-assisted thoracic surgery has provided an effective and potentially less invasive approach to chylothorax.

\[\text{Figure 2. Surgical treatment and management of chylothorax}\]
Successful ligation of the thoracic duct by thoracoscopy using fibrin glue or endoscopic clips at the site of leak has been attempted, and has the advantages of less post-operative pain and a shorter hospital stay. Graham and colleagues have described successful use of video-assisted thoracic surgery in 10 patients of chylothorax and recommend early operative intervention with this procedure. Early thoracoscopic repair has two major advantages: (i) the risk of malnutrition is minimised; and (ii) post-thoracotomy pain and discomfort are avoided. In situations where it is impossible to identify the duct surgically, especially in malignancy and radiation fibrosis, pleurodesis or pleurectomy may be used. These procedures have not only been used alone but also in combination with thoracotomy and ligation of the thoracic duct. Talc pleurodesis is most often used in chylothorax due to malignant disease that is resistant to conservative treatment. Other approaches include intrapleural infusion of tetracycline, fibrin glue, and OK-432, a Substrain of streptococcus pyogenes. Intra-pleural bleomycin has been found to be more effective in patients with chylothorax secondary to lymphoma.

The surgical approach to nontraumatic chylous effusions is more variable. Milsom and co-workers recommend pleuroperitoneal shunting after failure of conservative therapy and before thoracotomy. Murphy and associates have recommended placing the shunt if the drainage persists beyond five days. A pleuro-peritoneal shunt consists of pleural and peritoneal catheters connected to a manual pumping

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Occlusion of the shunt with fibrinous debris occurs in liver, which in turn drain into the right thoracic duct. Those of the diaphragm on the right side overlying the cavity is absorbed by the lymphatic vessels, mainly compared to surgery even in debilitated patients. 92-94 and can be safely preformed at an earlier time minimally invasive procedure with low morbidity 5. Sassoon CS, Light RW. Chylothorax and pseudochylothorax. 

CONCLUSIONS

There are numerous causes for chylothorax. The aetiology should be ascertained because treatment will be tailored to the underlying pathogenesis. Other considerations include nutritional status, severity of the underlying disease, and, proper delineation of the thoracic duct anatomy in planning treatment. When conservative management fails, appropriate surgical intervention or percutaneous embolisation must be considered.

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