Aetiology and management of chylothorax in adults
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Summary
Though rare in incidence, chylothorax can lead to significant morbidity and mortality. Its occurrence corresponds to increased mortality following esophagectomy. Leakage of chyle and lymph leads to significant loss of essential proteins, immunoglobulins, fat, vitamins, electrolytes and water. The presence of chylomicrons and a triglyceride level >110 mg/dl in the aspirated pleural fluid confirms the diagnosis of chylothorax. Identifying the aetiology using different diagnostic tests is important in planning treatment. While therapeutic thoracentesis provides relief from respiratory symptoms, the nutritional deficiency will continue to persist or deteriorate unless definitive therapeutic measures are instituted to stop leakage of chyle into the pleural space. Definitive therapy consists of obliteration and prevention of recurrence of chylothorax. Aggressive surgical therapy is recommended for post-traumatic or post-surgical chylothorax.

Keywords: Lymph; Thoracic duct; Chylothorax

1. Introduction
Chylothorax represents chyle in the pleural cavity. Chyle represents lymphatic fluid enriched with fat and its digestive products absorbed by the intestinal epithelium. The abundant presence of chylomicron in the pleural fluid aspirate is diagnostic of chylothorax. Chyle is collected and transported by the thoracic duct into the circulation. Damage to the duct leads to leakage of chyle into the pleural space. Obstruction to the centripetal flow of chyle can lead to rupture of the lymphatics and leakage of chyle into the pleural spaces. We discuss the aetiology, clinical features, investigations and management of this rare condition.

2. Anatomy of the thoracic lymphatic system
The lymphatic system includes cisterna chyli, thoracic duct, lymph glands, and lymphatic vessels. The thoracic duct most commonly originates from the cisterna chyli. Cisterna chyli is a lymphatic sac located anterior to the second lumbar vertebra and posterolateral to the abdominal aorta. Arising at the upper end of cisterna chyli, the thoracic duct passes posterior to the median arcuate ligament of the diaphragm between the azygos vein and the aorta in the right hemithorax, at the level of the diaphragm. The thoracic duct then ascends in the posterior mediastinum immediately anterior to the vertebral column on the right side of the descending thoracic aorta, in the aorto-oesophageal recess. In the lower thorax, it is on the right side of the distal oesophagus, but crosses to the left at the level of the fifth or sixth thoracic vertebra. It then ascends posterior to the aortic arch and anterior to the subclavian artery. After exiting the thorax via the superior thoracic aperture in the neck, it forms an arch anterior to the scalenus anterior muscle at the level of the seventh cervical vertebra by turning laterally and downwards to terminate at the junction of the left subclavian and internal jugular veins.

The thoracic duct has an approximate length of 36—45 cm and a diameter of 2—3 mm. It drains intestinal chyle to the bloodstream and the lymphatics of the body, except for the right side of the head and neck, right upper limb, right lung, right side of the heart and the convex surface of liver which in turn drain by the right lymphatic duct.

Significant variations to the above-described pattern can occur. Embryologically, the thoracic duct is a bilateral structure and hence many anatomical variations are possible. The pattern described above is true only in about 65% of the population [1]. The thoracic duct duplicates or triplicates itself in more than 40% of the population [1—4]. These branches may coalesce to form a plexus in the mid portion of the duct and end independently or as one duct [5]. Infrequently, the upper portion of the thoracic duct divides into two branches that drain separately, one in the usual...
manner and the other reaching the right subclavian vein [5]. This variation from the normal anatomical pattern explains the incidence of chyle leak despite care and attention the surgeon might have practised in identifying and protecting the main thoracic duct during an operation such as oesophagectomy.

3. Biochemistry of chyle

Thoracic duct transports chyle and lymph from the intestines, liver, abdominal wall and lower extremities into the systemic venous system. The primary function of the thoracic duct is the transport of digestive fat into the venous system. Normally, in an adult the volume varies between 10 and >100 ml/kg body weight, depending on diet, intestinal absorption, and degree of physical activity [6]. Almost all the chyle is derived from the intestinal lacteal system, which gives it a characteristic milky appearance [6]. Up to 70% of absorbed dietary fat passes through the intestinal lacteal system. Chyle contains significant quantities of chylomicron, triglyceride, cholesterol and fat-soluble vitamins. The other constituent, namely lymph, is derived mostly from the gastro-intestinal tract and liver. It contains a combination of lymphatic and gut-derived substances, including lymphocytes, immunoglobulins, enzymes, and products of digestion [7]. The electrolyte content of chyle is similar to that of serum. The thoracic duct transports 60—70% of ingested fat to the bloodstream usually at a concentration of about 0.4—6 g/dl. The concentration of protein in chyle is 2.2—6 g/dl [7]. Chyle also contains from 400 to 6800 white blood cells/ml. The majority of these are lymphocytes.

Table 1 shows the general composition of chyle. Those fatty acids with fewer than 10 carbon atoms in the chain are absorbed directly by the portal venous system. This particular fact forms the basis for the use of medium-chain triglycerides as an oral diet in the conservative management of chylothorax.

4. Mechanism of chylothorax

The fundamental mechanism behind chylothorax is leakage of chyle into the pleural space. This generally implies disruption of the thoracic duct in the majority of instances. Trauma to the thoracic duct is the commonest mechanism of chylothorax. The commonest cause is thoracic surgery, particularly involving dissection of the mediastinum. Central venous catheterisation and similar procedures can lead to extensive venous thrombosis in the neck. This impedes drainage of lymph and chyle into the subclavian veins, followed by thoracic duct drainage, with resultant chylothorax. Often, 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.

Mediastinal lymphadenopathy can in turn lead to chylothorax. The enlarged lymph nodes compress the lymphatic channels and thoracic duct and impede centripetal drainage of lymphatic flow from the periphery of lung parenchyma and pleural surfaces. This leads to diffuse extravasation or oozing of chyle and lymph into the pleural space.

4.1. Rare mechanisms leading to chylothorax

In hepatic chylothorax, chylous ascites crosses the diaphragm and accumulates in the pleural space. The abdominal source of chylothorax can be demonstrated by intraperitoneal injection of a radioisotope (99mTc-sulphur colloid) [8]. Chyloperitoneum results from leakage of chyle into the peritoneal cavity. Chylothorax as a complication of chyloperitoneum has been observed in various clinical conditions. As in hepatic chylothorax, chylous fluid from chyloperitoneum can cross over to the pleural space to cause chylothorax.

The mechanisms of chylothorax in idiopathic causes of chylothorax, such as Down’s syndrome and Noonan’s syndrome, are unclear. The mechanisms involved behind chylothorax in Noonan syndrome may be multifactorial such as congenital heart disease, coagulation-factor deficiency, pterygium colli, and lymphangiomatosis of the pleura, lungs, and chest wall.

Yellow-nail syndrome, defined as a triad of slow-growing yellow nails, lymphoedema, and pleural effusion, is due to hypoplastic or dilated lymphatics [9,10]. Bilateral chylothoraces are more common in yellow-nail syndrome.

5. Effect of chylothorax

Loss of chyle and lymph into pleural space can lead to drastic consequences because of the loss of essential proteins, immunoglobulins, fat, vitamins, electrolytes, and water. Large chylothoraces commonly lead to hypovolaemia due to the large volume loss. 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. Advanced cases exhibit clinical features of severe malnutrition. Hypoproteinaemia, acidosis, and hypocalcaemia are the most commonly recognised phenomena [11]. Mortality is high in patients with recalcitrant or untreated chylothorax. While repeated therapeutic thoracentesis provides relief from
respiratory symptoms, the nutritional deficiency will continue to persist or deteriorate unless definitive therapeutic measures are instituted to stop leakage of chyle into the pleural space. It is equally important to provide adequate nutritional support replenishing fluid loss, vitamin deficiencies and electrolyte loss while specific therapeutic measures are planned.

Continued loss of proteins, immunoglobulins, and T-lymphocytes into the pleural space leads to immunosuppression [12,13]. Furthermore, B-lymphocyte-mediated immune functions are impaired by prolonged chyle drainage [12,13]. These factors predispose the patient to opportunistic infections [14]. However, infection of a chylothorax itself is very uncommon because chyle is inherently bacteriostatic.

The bioavailability of certain drugs could be severely impaired in the presence of significant chyle leak. Sequestration of drugs in chyle should be recognised early, to prevent subtherapeutic plasma levels in patients undergoing drainage of chylothorax. There are reports of this phenomenon causing subtherapeutic digoxin [15], amiodarone [16] and cyclosporine [17] levels in the serum of patients.

6. Aetiological classification of chylothorax

There are a number of classifications for chylothorax in the literature [18,19]. Table 2 shows a modified DeMeester classification of chylothorax into congenital, traumatic, neoplastic and miscellaneous [18].

Among traumatic chylothoraces, iatrogenic causes constitute the majority [20]. Thoracic surgery constitutes the majority of iatrogenic causes. Oesophageal resection is the most common iatrogenic cause of chylothorax, and an incidence of 4% has been noted in the literature [20]. The proximity of the thoracic duct to the oesophagus, presence of collateral channels, and highly variable course leads to its injury during oesophageal and pulmonary resection. Chylorax is more likely in patients who undergo oesophageal resection by the transhiatal approach than the thoracic approach [21].

Other surgical procedures in the vicinity of the thoracic duct can inadvertently damage the thoracic duct. Laceration of the thoracic duct during catheterisation of the subclavian vein can lead to a chyle leak. Extensive venous thrombosis complicating central venous catheterisation has resulted in bilateral chylothorax and chylopericardium [22]. Thoric duct injury can occur following hyperextension of the cervical vertebral column and fracture-dislocation of the spine [23]. Isolated thoracic duct injuries as a result of penetrating chest trauma have resulted in chylothorax [24].

Non-iatrogenic causes are responsible for approximately 20% of cases of traumatic chylorax. Malignant obstruction of the thoracic duct is the commonest cause of nontraumatic chylothorax. Among the neoplastic aetiologies for chylothorax, lymphoma accounts for 70% of cases [20]. One of the less-recognised aetiologies of the chylothorax is hepatic cirrhosis [20]. As mentioned before, chylous pleural effusion results from the transdiaphragmatic passage of chylous ascitic fluid. Majority of hepatic chylothoraces are rightsided in occurrence.

Primary lymphatic disease is an uncommon cause of chylothorax. Mediastinal megalymphatics, congenital atresia of the thoracic duct [25] and localised chylous leak from the hilum of the lung can all lead to a chylothorax. Chylothorax can also occur as both an early or late complication of mediastinal radiotherapy when administered for a multitude of indications [26].

Lymphangioleiomyomatosis is an uncommon progressive thoracic pathology in females of childbearing age. It results in nodular and diffuse interstitial proliferation of the smooth muscle in the lungs, lymph nodes, and thoracic duct. Two-thirds of the patients have chylous pleural effusion [27–29]. Gorham’s disease, also known as massive osteolysis, is a rare disorder in which vascular channels dilate within medullary bone and destroy the bone [30]. Chylorax, as a result of leakage of chyle from lymphatic networks, is a life-threatening complication accompanying Gorham’s disease of the thoracic skeleton [31]. Incidence of chylothorax complicating Gorham’s disease is about 17% [31].

Table 2

Aetiological classification of chylothorax

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<th>Congenital</th>
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<tr>
<td>Atresia of thoracic duct</td>
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<td>Birth trauma</td>
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<td>Pleural thoracic duct fistula</td>
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<tr>
<td>Traumatic</td>
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<td>Penetrating</td>
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<td>Surgical</td>
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<td>Cervical</td>
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<td>Excision of lymph nodes</td>
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<tr>
<td>Radical neck dissection</td>
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<tr>
<td>Thoracic</td>
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<tr>
<td>Ligation of patent ductus arteriosus</td>
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<td>Surgery for coarctation of aorta/aortic aneurysm</td>
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<td>Post-esophagectomy</td>
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<td>Surgery for mediastinal tumours</td>
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<tr>
<td>Post-pneumonectomy</td>
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<td>Abdominal</td>
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<tr>
<td>Post-sympathectomy</td>
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<td>Radical lymph node dissection</td>
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<td>Diagnostic procedures</td>
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<td>Lumbar arteriography</td>
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<td>Subclavian vein catheterisation</td>
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<td>Neoplasms</td>
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<td>Miscellaneous</td>
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chylothorax [19]. Half of all chylothoraces are right-sided, one-third is left-sided, and the remainder are bilateral [32].

Chylothorax may be the initial manifestation in many patients. This mode of presentation is more likely in patients with occult lymphoma or malignancy involving thoracic lymph nodes. Insidious onset of chyous pleural effusion is a common feature in patients with lymphangioleiomyomatosis. Such patients usually present with chest discomfort and dyspnoea, or pleural effusion as an incidental finding on chest radiograph.

8. Investigations

In patients with postoperative and post-traumatic pleural effusions, attention should be directed to the anatomical location of surgery or trauma. Persistent drainage of pleural fluid via the thoracostomy tube should raise suspicion. The pleural fluid may not appear chyous if the pleural fluid is mixed with blood or if the patient is fasting.

Chest roentgenography with lateral views as well as decubitus views may be helpful in determining the size and location of the chylothorax. In complicated cases, other imaging techniques such as computed tomography (CT) of thorax may be required. CT is useful when chylothorax is associated with trauma, or where an underlying tumour is suspected. Patients with chylothorax and no obvious trauma should undergo CT of the chest to assess the mediastinum and hilum for enlarged lymph nodes. Bipedal lymphangiography has been recommended to identify the cause and detect the site and size of the leak [33]. Lymphangiography can help detect anastomotic leaks in the postoperative patients and complete transection or partial laceration of the duct [34,35].

Oral or nasogastric tube feeding of food with high fat content in patients with suspected chylothorax results in a dramatic change in the colour and biochemical constituents of pleural fluid [36]. Though in human subjects there is no compelling evidence to favour, a fat meal mixed with methylene blue leads to bluish-green discolouration of the pleural fluid in chylothorax, thereby helping at times to localise the leak. Some have recommended this technique as a diagnostic test for chylothorax. However, methylene blue staining of the fat meal may not be helpful in post-traumatic chylothorax since discolouration of pleural fluid can also occur in patients with oesophageal perforation.

The most important diagnostic step is thoracentesis to obtain a sample of pleural fluid for biochemical and other analyses. The appearance of the pleural fluid can be misleading because not all chyous pleural effusions appear milky white or whitish. Approximately 50% of patients may demonstrate bloody, yellow or green turbid, serous, or serosanguineous effusions [37]. The usual milky appearance of the effusion may also be seen in pseudochylothorax or in empyema where the purulent fluid contributes to the whitish colour. Even though typical chyous fluid appears creamy or milky white, the definitive diagnosis of a chyous pleural effusion is based on presence of chylomicrons in the fluid. Chylomicrons stain with Sudan III stain of cytological preparations. Normally, the diagnosis of chylothorax is based on the biochemical analysis of pleural fluid. Lipoprotein analysis of pleural fluid will confirm the presence of chylomicrons. Even though the presence of chylomicrons in the pleural fluid is synonymous with chylothorax, lipoprotein analysis is not available in all medical centres. In lieu of lipoprotein analysis, quantitation of triglyceride in pleural fluid can be used to diagnose chylothorax.

Pleural fluid triglyceride levels have been used in diagnosing chylothorax[8]. Pleural fluid triglyceride levels >110 mg/dl, presence of chylomicrons, low cholesterol level, and elevated lymphocyte count are diagnostic of a chylothorax. When the pleural fluid triglyceride level is >110 mg/dl, there is <1% chance of it not being chylous, and pleural fluid with a triglyceride value of <50 mg/dl has no more than a 5% chance of being chylous. When the triglyceride level is between 55 and 110 mg/dl, a lipoprotein analysis is indicated to detect chylomicrons. Other criteria for chylothorax include a pleural fluid to serum triglyceride ratio >1, and a pleural fluid to serum cholesterol ratio <1.

In hepatic chylothorax, pleural effusion is usually a transudate, and has lower cholesterol (22–64 mg/dl) levels than chylous effusions resulting from other causes [8]. In a given patient, the biochemical characteristics of hepatic chylothorax are identical to those of ascitic fluid. In hepatic chylothorax, the abdominal source of chylothorax can be demonstrated by scintigraphy after the intraperitoneal injection of a radioisotope (99mTc-sulphur colloid), with the appearance of radioisotope in the pleural cavity within 90 min [8].

9. Pseudochylothorax versus chylothorax

Pseudochylothorax, also known as chyliform pleural effusion or cholesterol pleural effusion, is the term applied to pleural fluid that mimics true chylous pleural effusion in appearance but lacks the biochemical criteria for chylothorax. In contrast to true chylothorax, it is chronic and may be present for months to years. Pseudochylothorax is more likely to result from long-standing pleural effusion. The fluid is yellow or opalescent green and may initially be mistaken for a chylous effusion, an empyema, or both [38].

High cholesterol levels are typical of a pseudochyloous pleural effusion. Cholesterol levels are generally >200 mg/dl and may even exceed 1000 mg/dl [39]. The fluid may demonstrate rhomboid-shaped cholesterol crystals on microscopy, which do not stain with Sudan III stain [39]. The precise origin of cholesterol in pseudochylothorax is unknown, but it is attributed to the continued breakdown of chronic inflammatory cells in a long-standing effusion. Tuberculosis pleural effusions account for approximately 54% of all cases [40]. Rheumatoid arthritis and trapped lung syndrome are rare causes of pseudochylothorax [41].

Pseudochylous pleural effusions are usually sterile and require no treatment, unless they are large and cause progressive respiratory symptoms. In symptomatic patients, management with pleural drainage, alone or in combination with specific therapy is indicated. Complicated cases or
tuberculous pseudochylothorax that leads to progressive respiratory distress may require pleural decortication.

10. Management of chylothorax

10.1. Medical management

There is universal agreement to the fact that the nutritional state of the patient worsens early after development of a thoracic duct leak and that this complication should be aggressively attended to. Patients almost always require aggressive nutritional support to reverse hypovolaemia, immunosuppression, and protein and electrolyte deficiencies. Once the haemodynamic status and nutritional status are stabilised, attention should be diverted to specific therapy. The general approach to the problem varies in that some clinicians adopt early surgical intervention while others adopt a conservative approach to the problem. Table 3 summarises various treatment modalities practised in chylothoraces.

Though conservative approach may have a role to play in small chylothoraces, therapeutic thoracentesis is the initial step in large chylothoraces that cause respiratory distress. Intercostal tube drainage is the preferred method of thoracentesis in most centres. During the period of excessive chyle leak, patients are generally advised nil by mouth or a diet rich in low-fat, medium-chain triglycerides. Medium-chain triglycerides are absorbed directly into the portal circulation. The latter approach resolves approximately 50% of congenital and traumatic chylothoraces [42]. In chronic chylothorax and in patients with rapid loss of nutrients into the pleural space, total parenteral nutrition is indicated [42].

Malignant chylothorax has been treated with radiotherapy and/or chemotherapy, with results that have not been consistently rewarding [43, 44]. Generally, radiation therapy to a total dose of 2000 rads controls most cases of malignant chylothorax [44, 45]. When surgical closure of the site of chyle leak is not practical, talc pleurodesis is an option in malignant chylothoraces [46]. One report of 19 patients with 24 chylothoraces secondary to lymphoma and refractory to chemotherapy or radiation therapy documented that talc pleurodesis via thoracoscopy under topical anaesthesia and conscious sedation resulted in a 100% success rate in the prevention of recurrence of chylothorax at 30, 60 and 90 days following the procedure [47]. Other methods to control chylothorax include phrenic nerve crush and reimplantation of the thoracic duct into a vein or re-anastomosis of a torn thoracic duct [48]. Somatostatin or octreotide infusions have been used successfully to reduce intestinal chyle production and secondarily reduce chyle leak [49–51]. Somatostatin treatment is an important adjuvant in the conservative management of chyle leak [52, 53].

10.2. Surgical management

Aggressive surgical therapy is recommended for post-traumatic or post-surgical chylothorax. Mortality with conservative management of chylothoraces after oesophagectomy approaches 50% whereas with active surgical intervention, it drops to about 10% [21, 54]. Many advocate conservative management for a maximum of 2 weeks in the absence of a strong indication for surgery [55, 56]. Children who develop chylothorax after oesophageal surgery are more likely to develop overwhelming bacterial and fungal sepsis if a conservative approach alone is instituted [57].

The general consensus is that surgical management should be adopted if medical management fails. The clinical parameters that prompt surgical intervention and the type of intervention vary between centers. In general terms, surgical intervention offers better results than conservative management when the daily chyle leak exceeds 1.5 l in an adult or >100 ml/kg body weight per day in a child [55]. Surgical intervention is usually indicated when the chyle drainage rate is more than 1 l/day for a period more than 5 days [58].

Lymphangiography will help to delineate the anatomy of the lymphatic channels and thoracic duct as well as the site of leak, though this is laborious. Other methods which are helpful to locate the leak includes preoperative subcutaneous injection of 1% Evans blue dye in the thigh or enteral administration of a fat source like olive oil or cream. Methylene blue may be added to the fat source to highlight the site of leak.

10.3. Techniques of ligation of the thoracic duct

Basically, if the chyle leak can be identified, direct ligation with nonabsorbable suture should be performed on either side of the leak. If the site of leak is not identifiable easily, extensive dissection to find this is strongly discouraged to minimise trauma and further leaks. Mass ligation of all tissue connecting the aorta, spine, oesophagus and pericardium is performed above the diaphragmatic hiatus via the right pleural space. This is traditionally performed through a sixth or seventh space thoracotomy. Parietal pleurectomy helps in these cases by promoting pleural symphysis.

If the drainage volume is high or chylothorax occurs after an oesophageal operation, early re-operation should be considered. The surgical intervention of choice in thoracic duct injury is thoracic, abdominal or cervical ligation of the thoracic duct [59]. Many surgeons prefer to ligate the thoracic duct at the diaphragmatic level because this procedure has the advantage of stopping flow from any accessory ducts that may not be recognised [60, 61].

<table>
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<tr>
<th>Table 3</th>
<th>Summary of treatment modalities in chylothorax</th>
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<td><strong>Conservative strategy</strong></td>
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<td>Nil by mouth</td>
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<td>Medium-chain triglycerides by mouth</td>
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<td>Total parenteral nutrition</td>
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<td>Drainage of chylothorax</td>
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<td>Thoracentesis</td>
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<td>Intercostal tube drainage</td>
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<td>Ensuring complete lung expansion</td>
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<td><strong>Operative strategy</strong></td>
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<td>Direct ligation of thoracic duct</td>
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<td>Mass ligation of supradiaphragmatic thoracic duct</td>
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<td>Pleuroperitoneal shunting</td>
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<td>Pleurectomy</td>
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<td>Pleurodesis – glue or talc</td>
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<td>Radiotherapy</td>
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Thoracoscopic ligation of thoracic duct has been well described [62–64]. After enteral feeding of about 50 ml of cream, thoracoscopy is performed under single lung ventilation. The first port is inserted in the right sixth or seventh intercostal space in the midaxillary line. A 30° scope is inserted through this port and the pleura inspected. A second port in the right eighth posterior intercostal space is used for dissection and division of the inferior pulmonary ligament. A third port in the anterior axillary line superiorly helps instrumentation to retract the lung. The pleural reflection is incised above the diaphragm. If the thoracic duct is easily identifiable at this stage it should be dissected carefully. A short segment of the duct is excised and the remaining ends clipped. If the duct is not identifiable, mass ligation of all tissue as described above is done using clips. The operation is finished with the placement of an intercostals drain. The drain can be removed when the drainage ceases with the patient on a normal diet.

Chyle leak following oesophagectomy occurs in about 10% of cases. If detected during the procedure, they should be closed immediately. Most postoperative chylothoraces in this setting do not heal with conservative management. Early surgical closure of the duct is the preferred option. Chylothorax noticed after pulmonary lobar resection is unusual. An initial course of conservative management is logical if the lung is fully expanded closing the thoracic cavity. If a large residual space remains early surgical closure of the duct is recommended. In patients with unavoidable pleural spaces with a chyle leak, pleuroperitoneal shunts may be appropriate.

When the thoracic duct is unidentifiable, talc pleurodesis could be tried. This traditional technique has a success rate of 95% and negligible morbidity [62,65]. When the chylothorax is complicated, loculated, or the site of chyle leak cannot be established, pleural decortication and surgical pleurodesis may be indicated. Fibrin glue has also been used to seal chyle leak by inducing pleurodesis [66–68].

Prophylactic ligation of the thoracic duct during oesophageal surgery has been recommended by some surgeons to prevent chylous fistula [21,69]. Some advocate routine ligation of the thoracic duct, especially with tumours of the midesophagus [69]. The incidence of post-oesophagectomy chylothorax reduced from 9 to 2.1% when elective ligation of the major thoracic duct was performed in a series of 255 patients [69]. The preferred site for elective ligation is in the upper abdomen or lower thorax, where there is more constant anatomy [59]. In the event that the duct cannot be identified, mass ligation of the tissue with teflon-pledgetted non-absorbable sutures between the aorta and azygous vein may be performed [60]. Though this technique was originally
described by Murphy and Piper [70], it was later popularised by Patterson and colleagues [60]. If the leak is in the upper thorax or neck, ligation of the thoracic duct is performed in the Poirier’s triangle, located between the internal carotid artery, arch of aorta, and vertebral column [59].

Pleuroperitoneal shunts have been used but these are of limited help in idiopathic cases [69,71]. Brofman et al. found that pleuroperitoneal shunts were effective in palliation of pleural effusions in yellow nail syndrome [72]. Therapy using externalised pleuroperitoneal shunting in chylothorax after surgical correction of congenital heart disease is considered safe, effective, and minimally invasive [73]. Though attempts have been successfully made by intervention radiologists to cannulate and embolise the leaking thoracic duct, success has been limited [74] and the procedure not reproducible at many centres.

10.4. Prognosis of chylothorax

In the past, the mortality due to chylothorax was in excess of 50% [75]. 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 [76]. Malignant chylothorax, chronic debilitating chylothorax and bilateral chylothoraces have worse prognosis [77]. The success of managing large chyle leaks involves aggressive nutritional support and early surgical intervention when indicated. A step-wise algorithm shown in Fig. 1 summarises the management of chylothorax in adults.

Reference