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CASE REPORT

Nonoperative Management of Traumatic Chylothorax

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Abstract Chylothorax is known for its rarity, and its diagnosis following blunt chest trauma is exceptional. Only a small number of cases have been reported in the literature. Severe consequences, such as cardiopulmonary abnormalities and metabolic, nutritional, and immunologic disorders, can result from chylothorax. Management of chylothorax is challenging. It can either be managed nonoperatively or surgically. Surgical treatment is required in cases of persistent or high output fistulae. We report here in three cases of blunt trauma chest following road traffic crash associated with chylothorax. All of them were successfully managed nonoperatively with inter costal tube drainage and supportive treatment sans need of any operative intervention.

Keywords Chylothorax · Traumatic chylothorax · Blunt trauma chest

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Introduction

Blunt trauma chest is a common injury which may range from insignificant rib fractures to life-threatening hemorrhage and pneumothorax. Blunt trauma chest is thus an important cause of death with a reported mortality rate of 15.5 % [1]. Pleural effusion is a common sequel to blunt trauma chest. Chylothorax is the rarest cause of such pleural effusion.

Chylothorax, according to Rotmann, was first described by Bartolet in 1633 [2] as a "rare condition with grave prognosis." Few cases of traumatic chylothorax have been reported in the literature. Whereas chylothorax is rather uncommon following blunt trauma chest, it should be suspected especially in cases of delayed development of pleural effusion. We report here three cases of traumatic chylothorax on the right side secondary to blunt trauma chest.

Case 1

A 32-year-old man presented to the emergency department (ED) of Jai Prakash Narain Apex Trauma Center (JPNATC), All India Institute of Medical sciences (AIIMS) on 9 April 2010 with alleged history of a road traffic crash (RTC) on 20 March 2010. Patient received initial treatment at a nearby private hospital where he was diagnosed to have fracture shaft of the right femur, grade 2 laceration of right lobe of the liver, a dot contusion in the right temporal lobe, and the right pleural effusion. An intercostal tube was placed in the right hemithorax, which initially drained about 500 mL of blood followed by milky white fluid from second day onwards. The volume of this effluent was increasing gradually. The patient was referred to us for further management. At the time of presentation, his airway was patent and he was breathing spontaneously with decreased air entry in the right

hemithorax. He was hemodynamically stable with blood pressure (BP) of 130/70 mmHg and pulse rate of 80 beats/min. He was fully conscious and oriented. He had a Thomas splint attached to his right lower limb and had an inter costal drainage (ICD) tube in situ on the right side draining milky white fluid approximately 2,000 mL in 24 h. We suspected chylothorax, and for bed side confirmation of it to be chyle and not pus, we mixed the drained fluid in ether and noted that fluid completely got dissolved in ether. A contrast-enhanced computed tomography (CECT) scan of chest and abdomen was performed, which revealed bilateral pleural effusion (right >left) with right ICD in situ with multiple rib fractures bilaterally. An ICD was also inserted on the left thorax, which drained about 100 mL of serous fluid. Pleural fluids from both the sides were sent for biochemical analysis that revealed high levels of triglycerides and chylomicrons in the fluid collected from the right side. Thus, a diagnosis of right-sided traumatic chylothorax was made. Patient was managed with nonoperative management (NOM) regimen with nil per oral, total parenteral nutrition, and injection octreotide 100 μg thrice daily along with incentive spirometry and intensive chest physiotherapy. His hematological and biochemical parameters revealed hemoglobin 15.2 g%, total leucocyte count 11,000 mm^3, urea 18 mg%, creatinine 0.8 mg%, serum electrolytes-Na 140 mEq, K 4.2 mEq, total serum protein 6 g%, with albumin 1.8 g%. The ICD output was monitored daily. It gradually reduced in amount and stopped by day 7 of NOM. Left ICD was removed on day 3 of insertion. His albumin level also improved to 3.3 mg/dL. He was subsequently allowed to take oral fluids and gradually normal diet was resumed. Injection octreotide was stopped on day 7 of NOM. Right ICD was then removed on day 8 of NOM. The patient then underwent a successful open reduction and internal fixation of the right femur fracture and was discharged in a stable condition. His total hospital stay was 30 days.

Case 2

A 23-year-old man was brought to the ED of JPNATC, AIIMS with alleged history of RTC. On primary survey, his airway was patent. He was breathing spontaneously; however, there was decreased air entry in bilateral lung fields along with bilateral subcutaneous emphysema. Bilateral ICD tubes were inserted. Each tube drained about 100 mL of blood along with gush of air. He was hemodynamically stable with a BP of 120/70 mmHg and pulse rate of 80 beats/min. He was fully conscious. There were no other injuries. Focused assessment sonography in trauma (FAST) examination was negative. A CECT scan chest revealed bilateral hemopneumothorax with multiple rib fractures. He was then shifted to surgical ward. The next day, his right ICD showed an increased output of 700 mL, which was milky white. This fluid on biochemical analysis was found to be consistent with chyle and thus a diagnosis of traumatic chylothorax was made. The patient was started on NOM regimen. The right ICD output gradually diminished. He was then allowed orally and the right ICD was removed on day 6 of NOM. Left ICD was removed on day 3 of insertion. He was then discharged on day 10 in a stable condition.

Case 3

A 40-year-old man presented to us with alleged history of RTC. The patient was driving a car when he hit another four wheeler and sustained a steering wheel injury to the anterior chest and abdomen. At presentation, his airway was patent and was breathing spontaneously. However, there was decreased air entry in bilateral basal zones. His blood pressure was 140/80 mmHg and pulse was 112 beats/min. He was conscious and oriented. A FAST examination was positive for fluid in the hepatorenal pouch. A CECT chest and abdomen revealed bilateral hemothorax along with lung contusion and traumatic dissection of descending aorta just above the aortic hiatus along with grade 2 laceration in segments 6 and 7 of the right lobe of the liver along with hemoperitoneum. The patient underwent an angiography-guided stent graft placement across the dissection. He was then transferred to the intensive care unit for closed observation. Bilateral ICD were inserted, which drained about 500 mL on either side. The patient, however, developed respiratory distress and had to be intubated. On the second day, the right-sided chest drain showed around 500 mL of milky white fluid that on analysis was found consistent with chyle. He was then started on NOM regime. There was a gradual decrease in the chest drain. However, patient developed progressive abdominal distention from day 4. A diagnostic peritoneal lavage was done, which revealed bile and an exploratory laparotomy had to be performed. On exploration, only liver laceration was found with biliary leak. The laceration was sutured. Abdomen was closed after thorough peritoneal lavage and placement of drains. The ICD output became nil by day 7. The patient was then started on nasogastric feeds, and ICDs were removed by day 9. He was shifted to the surgical ward on day 15 and discharged in a stable condition on day 20.

Discussion

Traumatic chylothorax, though still rare, is a well-known clinical entity. Bessone et al. [4] described a classification system that divides chylothoraces into four subclasses

(traumatic, congenital, iatrogenic after operation, and non-traumatic). The most common among these is iatrogenic following cardiothoracic procedures (0.2 %), neck dissections, or puncture of the left subclavian vein [5–9]. Non-traumatic causes include extrinsic compression or intrinsic obstruction of the thoracic duct by malignant lymphoma, metastatic cancer, and cirrhosis of the liver and other neoplastic disorders. On very rare occasions, a chylothorax can follow tuberculosis or amyloidosis.

Chylothorax was first described by Bartolet in 1633 [1, 2]. In 1988, Dulchavsky et al. [10] reported 19 cases of chylothorax after blunt chest trauma. Traumatic chylothorax can follow both blunt as well as penetrating chest injuries. Different mechanisms have been proposed [10] for the possible mechanism of chylothorax following blunt trauma chest which include the following:

1. Thoracospinal flexion and hyperextension mechanism, especially in young patients with an elastic thorax. The duct is ruptured by stretching it beyond its limit.
2. The right crus of diaphragm may shear the duct and open it.
3. Falls from a great height, which cause fractures of the thoracic vertebrae, or serial rib fractures in the dorsal thoracic aperture may induce rupture of the thoracic duct.

According to Milano et al. [11], the initial leak may be contained within the mediastinum and then gradually self-drain into one or both hemithoraces up to 7 or 10 days later. A gradually progressive chylothorax is better tolerated than a rapid collection as it may be accompanied by cardiopulmonary embarrassment. Forster et al. [12] presented cases of fracture to the thoracic spine and consecutive chylothorax.

Anatomic Considerations

Embryologically, the lymphatic system results from a coalescence of paired primordial lymph sacs derived from the budding of adjacent developing veins. Endothelial sprouts extending caudally from the paired jugular sacs unite with sprouts extending cephalad from the cisterna chyli, resulting in a dual lymphatic drainage system. In most instances, the originally paired structures partially unite to form a singular vascular structure, the thoracic duct. A number of resultant variations have been described. The most consistent anatomic variation in the thoracic duct in the adult is found extending from the cisterna chyli, which is usually midline just anterior to the first or second lumbar vertebra. The course continues cephalad through the aortic hiatus into the posterior mediastinum to the right of midline between the aorta and the azygos vein. The thoracic duct ascends essentially always as a surgically important single structure, for elective ligation, between the 12th and eighth thoracic vertebrae. It usually crosses the midline between the sixth and fourth thoracic vertebrae behind the esophagus and enters the left posterior mediastinum. Here, it lies inconspicuously behind the aortic arch just left of the esophagus and, more superiorly, behind the left subclavian artery. In this region, it is vulnerable to operations involving mobilization of the esophagus, the aortic arch, and the left subclavian artery. It arches into the superior mediastinum anterolaterally and descends just medial to the anterior scalene muscle, emptying near the junction of the left internal jugular and subclavian veins (Pouriers triangle). An understanding of its anatomic course in the thorax explains why injury to the duct below T5–6 usually produces a right chylothorax, whereas one above this level produces a left chylothorax. Also, it should be understood that this anatomy is constant only in its variability. The thoracic duct may be a dual system or may occasionally empty as a single duct on the right.

Throughout its course, there is an elaborate collateral network of lymphatic vessels. Multiple lymphaticovenous anastomoses may communicate freely with the azygos, lumbar, and intercostal veins and the inferior vena cava.

Nonoperative Management

There are no randomized, controlled studies to direct the management of chylothorax because of its infrequent occurrences and diverse presentations. Chylothorax is almost always an incidental finding presenting most often as a pleural effusion that is indistinguishable from a hemothorax on standard chest X-rays. An exact diagnosis of chylothorax can only be made by aspirating the effusion and subject it to biochemical analysis specifically for lipids (Table 1).

Nonoperative management of traumatic unilateral or bilateral chylothorax consists of (1) ICD tube thoracostomy, (2) nil per oral, (3) total parenteral nutrition, and (4) somatostatin and its analogues. All our three patients could be managed nonoperatively with this regimen. Tube drainage is considered to be more effective because pleural surface opposition to the fistula may accelerate healing. Also, this provides a means of accurately monitoring the rate of chyle

Table 1 Biochemical parameters to diagnose pleural fluid as chyle

Color	Milky white (can be serous)
Specific gravity	≥1.012
pH	7.4–7.8
Triglyceride	>100 mg/dL
Cholesterol to triglyceride ratio	<1
Electrophoresis	Chylomicrons

leakage. In cases of high-put fistulae, large amount of fluid, electrolytes, protein, fat, fat-soluble vitamins, and lymphocytes predominantly the T cell variety may be lost, which results into severe nutritional depletion and immunodeficiency state. For adequate replacement, monitoring of patient's weight, total protein, serum albumin, absolute lymphocyte count, and electrolytes is required. Administration of medium-chain triglycerides (MCTs) orally as a source of fat is an invaluable adjunct in the NOM of chylous effusions [1]. They are absorbed directly into the portal system bypassing the intestinal lacteal vessels. With the use of MCT, not only is nutrition satisfactorily maintained, but thoracic duct flow is minimized to promote healing of the leak [1]. The MCT diets have had variable success rates, however, perhaps because any oral enteral intake may increase lymph flow. If drainage remains unchanged, oral intake should be discontinued and total parenteral nutrition should continue. Some authors believe that the optimal approach includes a regimen of no oral intake, total parenteral nutrition, and tube thoracostomy drainage at the outset. Somatostatin can also be given to reduce chyle flow [13, 14]. We followed the same regimen with octreotide (a somatostatin analogue).

However, many authors recommend operative therapy after 1–2 weeks of NOM [1, 15, 16]. Certainly, imminent nutritional deterioration during NOM or the reaccumulation of chyle following the reinstitution of dietary fat is an indication for surgical management. To simplify the proper course of management, Selle and associates [14, 15] outlined that operative intervention is indicated when the average daily loss has exceeded 1,500 mL in adults or 100 mL/year of age in children for a 5-day period or when the chyle flow has not been diminished by 2 weeks of NOM.

Operative Management

The failure of nonoperative management under most circumstances necessitates an operative intervention for definitive therapy. For most operative and nonoperative traumatic chylous effusions, usually a right-sided thoracotomy or thoracoscopy [16] is needed to locate the site of disruption and to ligate or oversew the injured duct. Also, preoperative administration of a lipophilic dye or, preferably, cream may prove useful in locating the site of lymphatic leakage.

Conclusion

Chylothorax is an incidental diagnosis. Majority can be managed successfully with nonoperative management.

Conflict of Interest No financial grant has been received for this study.

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