Chylothorax secondary to gunshot wound

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Abstract

Background: Chylothorax is the occurrence of chyle in the pleural space due to damage or obstruction of the thoracic duct. Its etiology can be traumatic or nontraumatic. Traumatic lesion of the thoracic duct occurs after accidents or cardiothoracic surgery. Non-Hodgkin’s lymphoma is the most frequent nontraumatic etiology. Milky aspect and high content of triglycerides in pleural fluid confirm the diagnosis. The objective of this paper is to present a case of chylothorax secondary to shotgun lesion as well as to review current concepts about chylothorax and its treatment.

Clinical case: We present the case of a 33-year-old male patient with chylothorax secondary to gunshot lesion. Management was based on fasting, parenteral nutrition and pleural drainage. Patient’s evolution was satisfactory.

Conclusions: Chylothorax is an entity associated with high morbidity and mortality. Early diagnosis and appropriate treatment are essential.

Key words: chylothorax, triglycerides, thoracic duct.

Introduction

Chylothorax (CHT) is produced by the accumulation of chyle in the pleural cavity as a result of an injury or thoracic duct obstruction. Chyle is lymphatic fluid produced in the gastrointestinal tract, particularly the small intestine.

The mechanisms described in the formation of CHT include the escape of chyle from the thoracic duct or its secondary ducts, leakage through the lymphatics of the pleura and transdiaphragmatic flow of chylous ascites.1

The thoracic duct begins at the level of the second lumbar vertebra in Pecquet’s cistern. It has a length of 38-45 cm and a diameter of 5 mm, with anatomic variations in up to 50% of cases. It ascends from the front and right side of the spine at the level of the fifth dorsal vertebrae and crosses in the front.

It is situated on the left side between the esophagus and the spine, leading to the junction of the jugular and subclavian vein at the base of the neck.

The anatomic characteristics of the thoracic duct determine the location of the CHT. CHT will be on thoracic vertebra.2

CHT, according to its etiology, is classified as congenital, traumatic and nontraumatic. Nontraumatic CHT are generally caused by neoplasm, non-Hodgkin’s lymphoma being responsible for 75% of CHT associated with malignancy (due to extrinsic obstruction of the thoracic duct with large adenopathy). Other causes include amyloidosis, sarcoïdosis, subclavian vein thrombosis, superior vena cava obstruction, lymphangioleiomyomatosis, and infections (such as tuberculosis, filariasis, histoplasmosis, among others). Among traumatic causes, iatrogenic are the most frequent with 25% of the cases, as consequent to surgical procedures for the lungs, esophagus, aorta, mediastinal tumors and radical neck dissection. Between 0.2 and 3% are due to penetrating or closed trauma to the thorax after an accident.3 On some occasions it may be idiopathic. It may be complicated by respiratory failure, malnutrition due to protein loss and infections due to loss of immunoglobulins and lymphocytes.4

The object of this report is to present a case of CHT secondary to gunshot wound and to review the current concepts related to chylothorax based on scientific evidence.

Clinical Case

We present the case of a 33-year-old male. He was admitted to Hospital Médica Sur in Mexico City with hypovolemic shock...
after suffering a firearm injury with opening at the fork of the suprasternal notch with no exit wound. There were clinical and radiographic data of left hemopneumothorax requiring endopleural catheterization with a loss of 3 liters of hematic fluid as well as data of dilutional coagulopathy. For this reason we transfused erythrocyte concentrates, fresh frozen plasma, apheresis platelets, vitamin K and antifibrinolytics. On plain chest x-ray the bullet and multiple shrapnel were observed in the left infraclavicular region (Figure 1). CAT angiography was performed in which a lesion was demonstrated in the left carotid artery with leakage of contrast, perilesional hematoma and self-limited mediastinal involvement. We proceeded with conservative management and sedation to protect the airway (Figure 2A). During the first 24 h of the patient’s evolution, we performed a neurological fenestra presenting right thoracic limb paresis. Cranial MRI demonstrated the presence of left cerebral ischemia and cerebral edema (Figure 2B). We initiated anti-edema cerebral management and multimodal monitoring with measurement of intracranial pressure, bispectral index and ultrasound transcranial Doppler. On the fourth day of the patient’s stay in the ICU he presented an increase in loss from the endopleural catheterization of up to 1 liter in 24 h, changing the macroscopic characteristics from serohematic to chylous (cloudy yellow liquid) (Figure 3). With the clinical diagnosis of CHT we were able to determine triglyceride levels in pleural fluid, which were reported as 997 mg/dl, thus confirming diagnosis.

Treatment was conservative based on the suspension of external nourishment and the initiation of parenteral nutrition. The flow rate through the endopleural tube decreased progressively and we monitored the chyle leakage. Pleural fluid characteristics were changed to serous and determination of triglycerides decreased to 80 mg/dl.

**Discussion**

CHT is a rare condition seen in the intensive care unit. CHT of traumatic origin may be an early or late complication after an accident. It usually presents itself 7-10 days after the trauma, as occurred in the clinical case reported because the chyle accumulates first in the posterior mediastinum before reaching the pleural space. Rupture of the thoracic duct in closed chest trauma occurs primarily from a sudden hyperextension of the spinal column. In 20% of cases it is associated with vertebral fractures. The most common symptoms are chest pain and dyspnea.

The diagnosis, as in the case described, should be suspected when identifying the milky pleural fluid in the thoracentesis and/or in the pleural drainage. In a majority of the cases, the fluid is milky fluid characteristic of chylothorax.
mixed with blood. Pleural fluid analysis is characterized by the presence of lymphocytes and a triglyceride content of >110 mg/dl. Levels <50 mg/dl were excluded from the diagnosis because for levels between 50 and 110 mg/dl an analysis of lipoproteins in the pleural liquid is required. In these cases the detection of chylomicrons confirms CHT.9

Complications with appropriate treatment are rare but may include malnutrition, metabolic and immunological alterations due to T-lymphocyte depletion, loss of proteins and fat-soluble vitamins, as well as residual pachypleuritis. They usually do not cause infections in the pleural fluid due to the bacteriostatic power of chyle.10,11

Treatment for the underlying disease is essential in nontraumatic CHT, producing in most cases its disappearance. In traumatic CHT the initial treatment of choice is conservative because in as many as 50% of cases the thoracic duct closes spontaneously during the first 2 weeks. Conservative treatment is aimed at improving respiratory symptoms by placing a chest tube (allowing pulmonary re-expansion), volume replacement and nutrition. Production of chyle should be minimized by resting the intestines with parenteral nutrition or with a diet modification providing medium-chain triglycerides, which have the property of being directly absorbed from the intestinal lumen to the portal venous system. Treatment with somatostatin, octreotide and, recently, with amino-caproic acid has also proven to be effective in reducing chyle production. The mechanism by which octreotide, a somatostatin analog, decreases the chyle production includes reduction of the intestinal absorption of fats, principally triglycerides and increased fecal excretion.12-17

In the event that daily loss of chyle is >1500 ml for >5 days through the chest tube, or the flow does not decrease after 15 days of conservative treatment or nutritional complications appear, then surgical treatment is indicated. Among the surgical options is ligation of the thoracic duct through thoracoscopy or thoracotomy. Other options include pleurectomy, chemical pleurodesis, pleuroperitoneal window and percutaneous thoracic duct embolization.18,19

In conclusion, this report describes a patient who developed CHT secondary to a gunshot wound projectile in the neck. CHT requires a high index of clinical suspicion for diagnosis and is confirmed by the presence of chyloous pleural fluid with elevated concentration of triglycerides. Timely and appropriate treatment is essential to prevent associated complications, as demonstrated in this case report. The patient responded satisfactorily to conservative management based on parenteral nutrition and pleural drainage.

References