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Case Report Open Access

Middle Aged Female with Shortness of Breath, Congestive Heart Failure and Left Pleural Effusion-What is the Diagnosis?

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Abstract

A chylothorax is formed when the thoracic duct is disrupted and the chyle enters the thoracic space. The causes are traumatic-surgery or trauma to the chest and non-traumatic with malignancy and specifically lymphoma playing major role, idiopathic and miscellaneous-sarcoidosis, cirrhosis of the liver, heart disease, childbirth, Castleman's disease. Retrosternal goiter is an extremely rare cause of chylothorax and the first case described was in 1996.

We are describing a 44 year old African-American female with left pleural effusion and multiple admissions due to it to the hospital. The left pleural effusion was attributed to be a result of heart failure with preserved ejection fraction which the patient had. Despite aggressive dieresis the patients left pleural effusion and shortness of breath persisted which let to 3 admissions to the hospital. After investigating further the effusion turned out to be chylothorax due to compression and disruption of the thoracic duct by enlarged retrosternaly located multinodular goiter.

After left lobectomy and removal of the isthmus of the thyroid the left chylothorax resolved and never recurred in the following 4-years.

Keywords: Chylothorax; Multinodular goiter; Heart failure with preserved ejection fraction; Shortness of breath

Introduction

Chylothorax is characterized by pleural fluid with a turbid or milky appearance due to high triglyceride content in the form of chylomicrons that enters the pleural space as a chyle most commonly from the disruption of the thoracic duct [1]. The chyle is rich in triglycerides, which produces the milky, opalescent appearance of the pleural fluid. Chyle also contains lymphocytes as the major cellular content. The electrolyte content of the chyle is similar to plasma and the protein content is usually greater than 3 g/dl. The diagnosis of chylothorax is made by the milky appearance of the pleural fluid and by measuring the pleural fluid triglyceride concentration which is typically more than 110 mg/dl or in cases in which the triglycerides are between 50-110 mg/dl by lipoprotein analysis of the pleural fluid which shows the presence of chylomicrons [2].

Malignancy is a leading cause of non-traumatic chylothorax responsible for 63% of non-traumatic causes. Lymphoma is the single most common cause responsible for 50% of the non-traumatic causes. Other common causes of chylothorax are Castleman's disease, sarcoidosis, histoplasmosis, lymphangioleiomyomatosis, Noonan syndrome, filariasis, heart failure, lymphangitis of the thoracic duct, aneurysm of the thoracic aorta that erodes the duct, Cirrhosis of the liver, childbirth and idiopathic [3,4].

Surgical procedures and chest trauma are the most common causes of the traumatic chylothorax [5].

We describe a case of chylothorax in a female patient with large retrosternal goiter who was admitted to the hospital with diagnosis of heart failure exacerbation and was given diuretics on admission.

Case Presentation

Our patient was 44 year old African-American female who was admitted to our hospital twice in the last one month with recurrent shortness of breath and left sided thoracic pain. She was found to have hypertension, morbid obesity, heart failure with preserved ejection fraction impaired relaxation pattern due to hypertension and osteoarthritis of the knees. The patient was treated each time with diuretics and blood pressure control with ACE-inhibitors and the left pleural effusion was attributed to be a sign of decompensated heart failure. The patient had minimal improvement of symptoms upon those 2 discharges and return for the third time in the ER with worsening shortness of breath and persistent left pleural effusion. The patient's medications were Lasix 40 mg twice a day and Lisinopril 10 mg a day. There was no history of trauma or surgery of the chest.

On the physical examination the patient was morbidly obese-Body Mass Index 40, tachypnic at 25 breaths per minute. Dullness on percussion was found on the left side of the chest with diminished breath sounds there. There were no crackles in the lungs, the neck was flat without jugular venous distention and no edema of the lower extremities was found. The patient had regular heart rate of 80 beats per minute with positive S 4. The BNP was normal and the d-dimmer test was normal. We also found that the patient has a large multinodular goiter with retrosternal extension.

On CXR was found that the patient has cardiomegaly and left pleural effusion. Given the non-improvement of patient's symptoms on diuretics and ACE-inhibitors, multiple admissions in the last one month for shortness of breath and left pleural effusion we performed thoracentesis in the Emergency Room. We drained 1.5 liters of milky pleural fluid. Analysis of the fluid revealed 3420 WBC/ml, 760 RBC/ml, PML-6%, mononuclear cells-94%, glucose-111 mg/dl, total

protein-3.7 g/dl, LDH-78. Cytology and flow cytometry from the pleural fluid were negative for malignancy. AFB and fungal culture were negative for those infections. Triglyceride content of the pleural fluid was 990 mg/dl, cholesterol-35 mg/dl and lipoprotein analysis confirmed the presence of chylomicrons. This confirmed the presence of chylothorax. We repeated thoracentesis 3 hours after fasting and cholesterol crystals and chylomicrons were not demonstrated in the pleural fluid.

The blood total protein was 6 g/dl and the blood Lactate Dehydrogenase was 300. We also obtained CT of the chest, abdomen and pelvis which confirmed the large left pleural effusion, also tracheal deviation to the right by large left paratracheal multinodular goiter and fatty liver. No lymph node enlargement or any other focal masses to suggest malignancy were found. Thyroid function test were normal. The ultrasonography of the thyroid gland confirmed the presence of multinodular goiter, with no suspicion for malignancy nodules and no nodule which was larger than 1.5 cm. We performed FNA of the largest thyroid nodule and the cytology was benign.

The patient feeding per mouth was discontinued, Peripherally Inserted Central line was placed and the patient was started on Total Parentheral Nutrition/TPN, we continued the Lisinopril 10 mg a day and discontinued Lasix given the lack of exacerbation of the heart failure and started for better blood pressure control Hydrochlorothiazide 25 mg a day. The surgical service was consulted and they performed left thyroid lobectomy with removal of the thyroid isthmus. The surgeon saw during the procedure the thoracic duct which was severed. We did not plan before the surgery lymph angiogram, because we relied on the surgeon to see the damaged Thoracic duct during the surgery. The left thyroid lobe and the thyroid isthmus weighed 283 grams after removal. The right thyroid lobe was normal in size and without palpable nodules and was not removed. The pathology was consistent with multinodular colloid goiter. The post-operative period was uneventful. The left pleural effusion disappeared 72 hours after the removal of the goiter and the shortness of breath resolved. We gradually discontinued the TPN and enteral diet was initiated.

Now 4 years after removal of the goiter the patient remains asymptomatic, without recurrence of the chylothorax.

Discussion

The majority of patients with intrathoracic goiters are asymptomatic [6]. However clinical signs may appear when the thyroid gland enlarges. Symptoms like stridor, hoarseness and dysphagia, when present, relate to mechanical compression and displacement of the esophagus or trachea. Occasionally, a superior vena cava syndrome can be seen [7].

The common causes of chylothorax were discussed above. Very rarely the extreme thyroid enlargement with retrosternal extension can present as a chylothorax without other compressive symptoms like in our case [8].

The thoracic duct runs cephalad to the cisterna chili, through the mediastinum immediately anterior to the vertebral bodies and posterior to the esophagus. In the upper thorax, it inclines to the left passing posterior to the subclavian artery in the base of the neck. It passes behind the great vessels on the left side of the neck before emptying into the venous system at the lateral aspect of the junction between left internal jugular and subclavian veins. In our patient, it seems likely that the duct was compressed and disrupted by the thyroid either in the upper mediastinum or immediately after passing behind the esophagus in the base of the neck. The lack of edema of the head and neck and left arm indicates that the most distal aspect of the thoracic duct was not involved.

Heart failure can rarely cause chylothorax due to increased back pressure into the lymphatic vessels due to insufficient venous return, but in our case heart failure was under control and in fact our patient was over diuresed [6]. The patient did not have clinical signs of heart failure and had normal beta natriuretic peptide, increased BUN to creatinine ratio without other explanation of increased ratio of 20:1, and mild metabolic alkalosis with pH-7.48, HCO_3-33 and PCO_2- 47.

The interesting observation in our patient was that the left pleural effusion was attributed on the previous admissions to the heart failure exacerbation besides two admissions without resolution of the pleural effusion and minimal improvement of the symptoms.

This underscores the necessity of draining the pleural effusions in patients with heart failure if the effusion does not decrease or resolve after 48-72 hours of treatment with diuretics and the need of thorough evaluation of the patients for other etiologies of the pleural effusion like in our patients-the multinodular goiter with retrosternal extension which caused chylothorax. After the thyroid surgery the left sided chylothorax never recurred.

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