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Chylothorax Revealing Non-Hodgkin's Lymphoma: A Case Report

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Abstract:
Chylothorax is a rare condition resulting from the extravasation of chyle into the pleural space due to obstruction or injury of the thoracic duct or its tributaries. It has diverse etiologies, both traumatic and non-traumatic, and requires multidisciplinary management. We report the case of a 48-year-old patient, a chronic smoker with a 5-pack-year history, who presented with left-sided laterothoracic pain and stage II mMRC dyspnea of progressive onset for 2 months prior to admission. This revealed left-sided pleurisy with significant fluid accumulation and mediastinal adenopathy, with a reduced caliber of the left main bronchus. Flexible bronchoscopy revealed tumor-like infiltration of the left lingula and lower lobe, and bronchial biopsies were consistent with type B non-Hodgkin's lymphoma. The patient was started on chemotherapy according to the R-ACVBP protocol, with good radiological and clinical progress.

I. Introduction

Chylothorax refers to the presence of chyle in the pleural space, which often occurs due to interruption of the thoracic duct or reduced lymphatic drainage. A white or milky pleural fluid with a triglyceride concentration above 110 mg/dL strongly supports the diagnosis of chylothorax, which is almost always exudative. Lymphoma is the primary non-traumatic cause of chylothorax, which can lead to numerous complications, including infectious, hemorrhagic, and nutritional issues. In 50% of cases, these complications can result in death if left untreated or inadequately managed, highlighting the importance of appropriate multidisciplinary care (2)

II. Patient and observation

A 48-year-old patient, a chronic smoker with a 5-pack-year history, presented with left-sided laterothoracic pain of the side-stitch type, progressively worsening over the 2 months prior to admission. This was associated with stage II mMRC dyspnea, occurring in the context of apyrexia and slight general condition decline (asthenia and anorexia).

Clinical examination revealed a patient in good general condition (PS 0), hemodynamically and respiratorily stable, with a BMI of 28 (overweight). Pleuropulmonary examination revealed a liquid effusion syndrome covering the entire left hemithorax, with no repression of heart sounds. The rest of the somatic examination was unremarkable.

Chest X-ray showed an opaque left hemithorax with deviation of mediastinal structures toward the contralateral side **(Figure 1).**

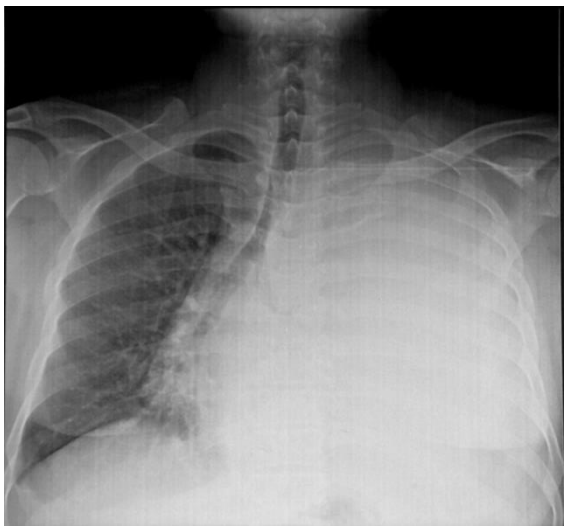

Figure 1: Chest X-ray, showing an opaque left hemithorax with deviation of the mediastinal structures toward the contralateral side.

Thoracic ultrasonography confirmed a large-volume pleural fluid effusion on the left and a small-volume effusion on the right that could not be punctured. Biochemical analysis of the pleural fluid collected from the left side suggested exudative chylous pleurisy, with protein at 62 g/L, triglycerides at 12.64 g/L (>1.1 g/L), total

cholesterol at 0.9 g/L (<2 g/L), and the presence of chylomicrons **(Figure 2).**

Figure 2: Milky appearance of the pleural fluid suggestive of chylothorax

The patient initially underwent an evacuating pleural puncture, yielding 1200 mL of chylous fluid. A low-fat (<10 g/d), high-protein, long-chain triglyceride-free, medium-chain triglyceride-rich diet was instituted. The patient was given a list of permitted and prohibited foodstuffs.

The blood count, fluid and electrolyte balance, and lipid profile showed no abnormalities, except for an elevated LDH level of 290 IU/L.

A thoraco-abdominal-pelvic CT scan revealed bilateral pleurisy, with a large volume on the left and a small volume on the right. It also showed a mass of subcarinal and left hilar adenopathies, with reduced caliber of the left bronchus, as well as retroperitoneal adenopathies, the largest of which measured 3 cm in diameter **(Figures 3 and 4)**

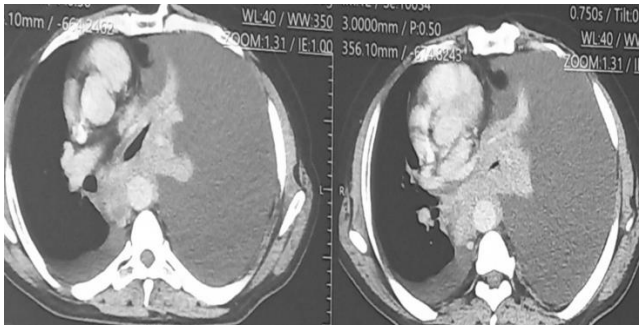

Figure 3: Chest CT scan, axial section (mediastinal window): Bilateral pleurisy of great abundance on the left and low abundance on the right with Magma of subcarinal and left hilar adenopathies with reduced caliber of the left bronchus.

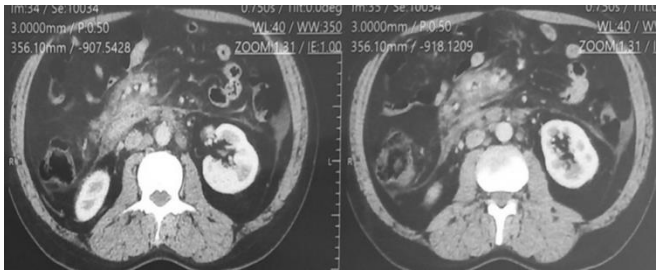

Figure 4: Abdomino-pelvic CT scan (axial section) showing retroperitoneal adenopathies, the largest of which is 3 cm in diameter.

Flexible bronchoscopy revealed infiltrative stenosis of the lingula orifice and the left Fowler's area **(Figure 5).**

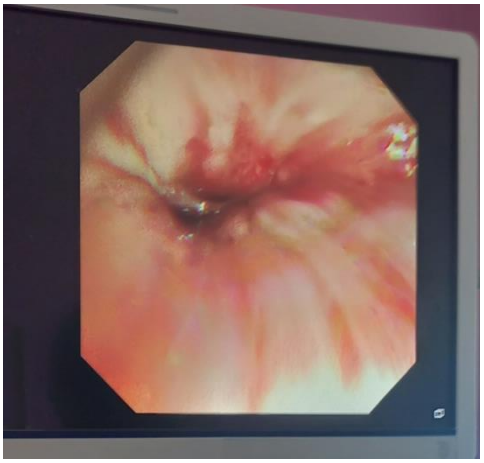

Figure 5: Flexible bronchoscopy showing infiltrative stenosis of the lingual

Anatomopathological and immunohistochemical analysis of the bronchial biopsies was consistent with diffuse large B-cell non-Hodgkin's lymphoma.

PET-CT revealed a locally advanced, polylobed mediastinal tumor process beneath the left hilar carina, with involvement of laterocervical, bilateral axillary, and subdiaphragmatic lymph nodes. It also showed bilateral pleural effusion with hypermetabolic nodular thickening of the left pleura and multiple secondary bone lesions **(Figure 6).**

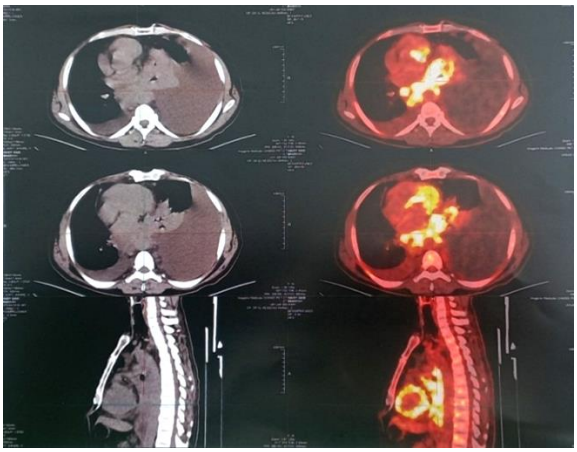

Figure 6: PET-Scan: Locally advanced mediastinal polylobed tumor process under the left hilar carina with laterocervical, bilateral axillary and subdiaphragmatic lymph node involvement, bilateral effusion with hypermetabolic nodular thickening of the left pleura, multiple secondary bone foci.

According to the Ann Arbor classification, the large-cell B lymphoma is classified as stage IV-A, and the IPI prognostic score indicates a poor prognosis.

The therapeutic management of the lymphoma involved chemotherapy using the R-ACVBP protocol (R: Rituximab, A: Adriamycin 75 mg/m² on D1, C: Cyclophosphamide 1200 mg/m² on D1, V: Vindesine 2 mg/m² on D1 and D5, B: Bleomycin 10 mg/m² on D1 and D5, P: Prednisone 40 mg/m² from D1 to D5). After four sessions of chemotherapy, the patient remained in good general condition, with improvement in dyspnea to stage I MMRC. On pleuropulmonary examination, there was the presence of pleural effusion in the left basithoracic region. Follow-up chest X-ray showed clear regression of the pleural effusion **(Figure 7).**

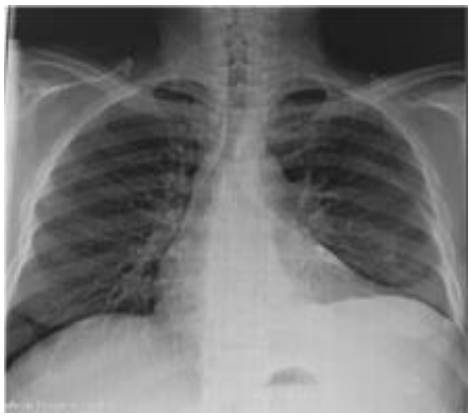

Figure 7: Front thoracic X-ray after 4 sessions of chemotherapy showing clear regression of the chyothorax

The patient is undergoing regular follow-up in both hematology and pulmonology and has benefited from iterative evacuating pleural punctures, with continued adherence to the prescribed diet.

III. Discussion

Chylothorax is characterized by a lactescent pleural effusion resulting from damage to the thoracic duct, causing chyle to leak into the pleural space. Diagnosis is based on triglyceride levels and/or the presence of chylomicrons in the pleural fluid. The most common causes of chylothorax are traumatic, mainly following surgery. Non-traumatic causes frequently include tumors, particularly lymphomas (2).

Low-volume or early-onset chylothorax is clinically silent and does not differ from other pleural effusions. Large-volume chylothorax, or early-onset chylothorax, can lead to dyspnea, cough, chest pain, and hypovolemia. As the chyle itself does not cause inflammatory irritation, pleuritic pain and fever are absent. In chronic cases, where leakage goes unchecked or unnoticed, malnutrition ensues, with weight loss and muscle wasting (3).

Pleural fluid is milky in appearance and often lymphocytic exudative, but it may be transudative in 25% of cases. The diagnosis of chylothorax is made in the presence of: a pleural fluid triglyceride level >1.24 mmol/L (110 mg/dL) with a cholesterol level <5.18 mmol/L (200 mg/dL), with chylomicrons present in the majority of cases (4).

The etiologies of chylothorax can be classified as traumatic or non-traumatic, and traumatic cases can be subdivided into two categories: iatrogenic and non-iatrogenic. Non-iatrogenic trauma accounts for 20% of traumatic causes, directly damaging the thoracic duct or causing tissue damage in the vicinity. Iatrogenic causes are mainly thoracic surgery, especially of the esophagus, which causes chylothorax in 4% of cases. Other iatrogenic traumatic causes include damage to the thoracic duct following central venous catheterization (5).

Non-traumatic etiologies include malignant tumors, notably lymphoma in 70% of cases (non-Hodgkin's > Hodgkin's), and, rarely, metastatic tumors. Non-neoplastic causes include sarcoidosis, retrosternal goiter, amyloidosis, and thrombosis of the superior vena cava. Congenital

anomalies of the ducts and diseases of the lymphatic vessels, such as yellow nail syndrome, as well as infectious causes such as tuberculous lymphadenitis, histoplasmosis, or others, may also be involved. It can also be idiopathic (5).

In certain situations, thoraco-abdominal CT scans can locate the leak and provide valuable information on the cause of the chylothorax. Magnetic resonance imaging (MRI) can also provide useful anatomical details and pinpoint the leak for possible intervention. Lymphangiography and lymphoscintigraphy, though rarely performed nowadays, are also useful for identifying the location of the leak, assessing the permeability of the thoracic duct, and differentiating between partial and complete sectioning of the duct. These imaging techniques also provide valuable insights for selecting the appropriate treatment (2, 6).

To date, no prospective study has been able to compare the various therapeutic options for chylothorax. If the clinical condition permits, conservative treatment (treating the underlying cause, pleural drainage, and appropriate dietary measures) should be the first choice, even in chylothorax of traumatic origin (6,7). When the origin is non-traumatic, specific management of the underlying pathology is essential, such as chemotherapy and radiotherapy for lymphoma, combined with conservative treatment, which may lead to resolution of the chylothorax. In chylothorax of traumatic origin, conservative treatment should also be proposed as first-line therapy. However, in nearly 50% of chylothoraxes related to surgical complications, invasive management will be required (8,9).

IV. Conclusion

Chylothorax remains a rare condition, but one that can lead to serious long-term consequences if left untreated or inadequately treated, arising from a variety of traumatic and non-traumatic etiologies. Diagnosis is clinical and biological, and initial treatment is conservative, primarily involving pleural drainage and appropriate dietary measures, which must always be combined with treatment of the underlying pathology. In the event of failure, surgical management or interventional radiology, including lymphatic duct occlusion, are effective treatment options. Prognosis depends on the underlying etiology.

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