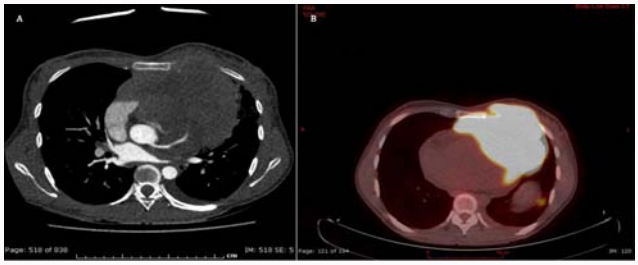

Figure 1: Initial imaging performed before any treatment attempted. A) Aortic CECT (Contrast-Enhanced CT-scan) shows a large heterogeneous invasive anterior mediastinal mass with pleural, anterior chest wall and pericardial invasion. Moreover, there is soft tissue infiltration surrounding the left coronary artery with wall irregularities; B) FDG-PET (Fluoro Desoxy Glucose-Positron Emission Tomography) scan shows the large hypermetabolic (SUV: 17) anterior mediastinal mass with evidence of chest wall & pericardial invasion.

Case Report

A 28-year-old non-smoker Caucasian woman, mother of three kids, who had no history of any chronic illness and denied excessive alcohol consumption, presented to the emergency department complaining of a progressive left shoulder and back pain, palpitations, fatigability, dyspnea on exertion, orthopnea and paroxysmal nocturnal dyspnea. All those symptoms developed over the last few months. She also described a swollen mass on her chest and another in her back that made it uncomfortable for her to lie on her back. She had no history of weight loss, but described occasional night sweats.

On clinical examination, she was fully conscious, alert and oriented. Her blood pressure was 138/90 millimeter of mercury (mmHg), heart rate 118 beats per minute, body temperature 36.4°C, respiratory rate 20 per minute, and oxygen saturation 100% on room air. An electrocardiogram was immediately done and revealed an ST-segment elevation in leads V2 through V4 with reciprocal ST depression in leads III and a VF. Aspirin, Ticagrelor and intravenous unfractionned heparin were started immediately. Laboratory results revealed a white blood cell count of 9.1 g/L, hemoglobin of 125 g/L and platelets of 414 g/L. Troponin I were equivocal (0.090 ng/mL, normal: <0.034), lactate deshydrogenase was 418 (elevated). The rest of the laboratory results were normal. The patient was transferred to the cardiac catheterization lab for emergency primary coronary angiography with possible angioplasty.

The coronary angiogram showed no atheromatous lesions of the coronaries, but it revealed a stiff left anterior descending artery that did not move in the images because of an anterolateral akinesia.

Echocardiography was performed, in an attempt to understand the cause of her symptoms and her unusual coronary angiogram findings. This exam revealed a massive anterior mediastinal mass that compressed most of the right ventricle and conusarteriosus as well as a part of the left ventricle. As there was no visible separation between the pericardium and myocardium in certain views, it was suspected that the tumor infiltrated the myocardium. Moreover, the interior of the left ventricle showed a small mass at the apex, but it was hard to know if it was a trabeculae, or the infiltration by the tumor. The compression of the heart by the tumor led to an anterolateral, apical and right ventricular hypokinesia and an acceleration of the pulmonary artery flow: the maximum and average gradients of the valve were respectively 42 mmHg and 27 mmHg, with the pulmonary artery systolic pressure estimated at 42 mmHg+15 mmHg (however

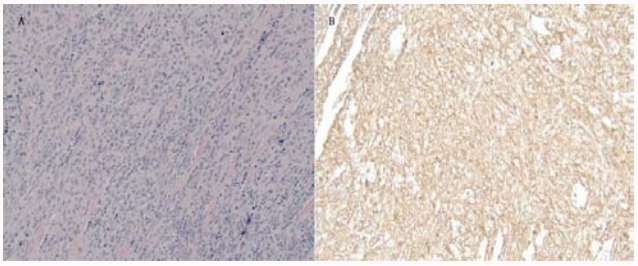

Figure 2: Pathology of the biopsied mass lesion. A) Hematoxylin and eosin stain (X20) of the left anterior hemithorax specimen showing a diffuse large cells lymphoma proliferation, dissociating the striated muscular fibers; B) Immunohistochemistry of the left anterior hemithorax specimen showing a CD20 positive proliferation.

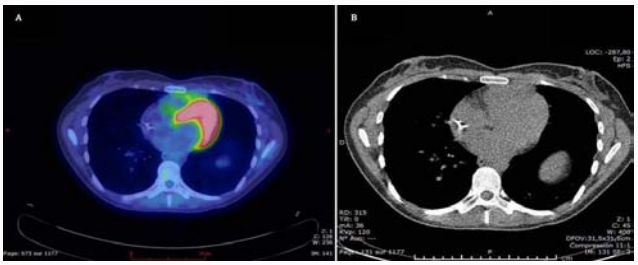

Figure 3: Imagining performed after chemotherapy. A) FDG-PET scan (obtained after five chemotherapy cycles) shows a complete remission with only a metabolically inactive anterior mediastinal lesion left; B) Low-Dose CT-scan (obtained after five chemotherapy cycles) shows the regression of the anterior mediastinal mass.

difficult to evaluate due to the presence of a very mild tricuspid regurgitation). Left ventricular ejection fraction was moderately reduced to 40% to 45%. The inferior vena cava was dilated and not compliant to respiratory variations. There was a 15 millimeters posterior pericardial effusion.

Cardiac ventriculogram, CT (Computed Tomography) scan, MRI (Magnetic Resonance Imagery) (Figure 1A) and PET (Positron Emission Tomography) scan were performed. All those imaging modalities confirmed the extension of the tumor transmurally, infiltrating the heart muscle (Figure 1B). The mass was extending to the anterior left hemithorax. A left pleural effusion was also present, as well as right hilar and paratracheal lymphadenopathies, a left lung nodule, abdominal lesions and a subcutaneous right hemithorax nodule. The Ann Arbor staging was determined as IV-X.

The mass measured 14.7 cm (transverse) by 8.7 cm (anteroposterior) by 17.2 cm (craniocaudal), infiltrating and compressing the right ventricle and especially the conus arteriosus. The aorta and pulmonary trunk were displaced by the tumor, but were still patent.

The general surgery team performed an excisional biopsy. They felt that the most accessible part of the tumor was located at the anterior left hemithorax. The biopsy was performed under local anesthesia in the OR, with a team of anesthesiologist and ENT surgeon available if there were any complications regarding her airway. The specimen was sent to our pathology department. It revealed that the mass was a non-germinal center DLBCL that originated from the mediastinum (Figure 2). The oncology team installed telemetry as a way to be warned of any complications and presented the patient to the cardiac surgery team in case of a cardiac rupture.