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Case Report Open Access

Invasion of the Left Atrium by a Squamous Lung Cancer

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Case Report

We report a case of a 58 year old healthy white man, asymptomatic with no medical history ever reported and non-smoker. No medication was taken daily.

Previously, he had no respiratory symptoms, until the day he came to the emergency room with sudden chest pain, with no irradiation and progressive intensity. A CT thoracic angiogram was performed and showed invasion of the left atrium (Figures 1-4). Bronchoscopy revealed in the right bronchi tree an exophytic mass with superficial vascularization and indirect signs of tumor. Biopsy confirmed a squamous lung cancer with moderate differentiation. Immunohistochemistry testing was performed and was positive for cytokeratin 5 and negative for cytokeratin 7, TTF1 and synaptophysin. This diagnosis wasn't verified by immune staining for p63. The echocardiogram showed an enlarged left ventricle, diffuse hypo kinesia, left ventricle ejection fraction of 36%, dilatation of the left atrium (32 cm2) and right atrium (27 cm2); a hypo echoic mass was in contact with the left atrium, but without direct invasion.

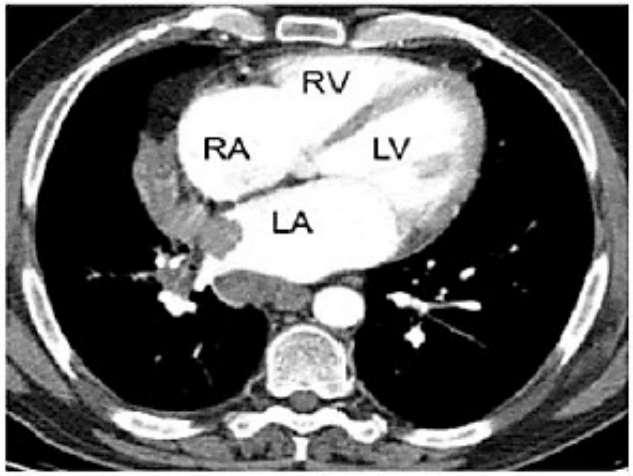

Figure 1: Inferior hilar right mass 45.5 × 44.9 mm.

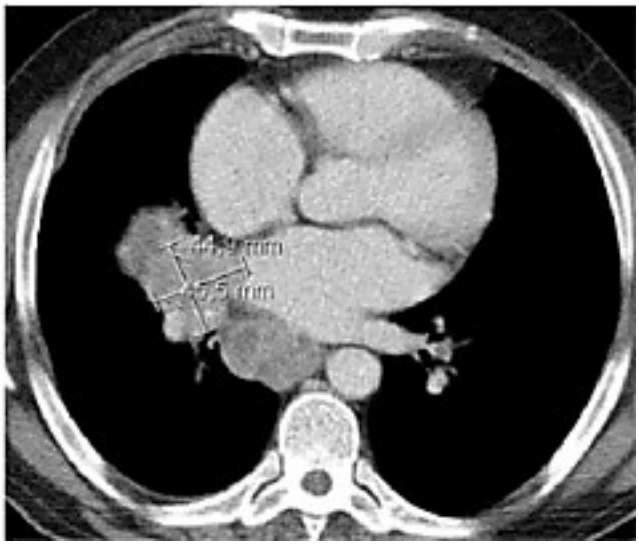

Figure 2: Inferior hilar right mass which invades the left atrium.

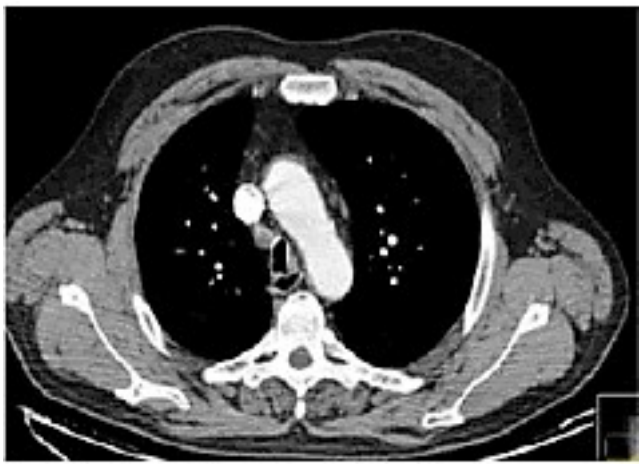

Figure 3: Right paratracheal node 14 × 18 mm.

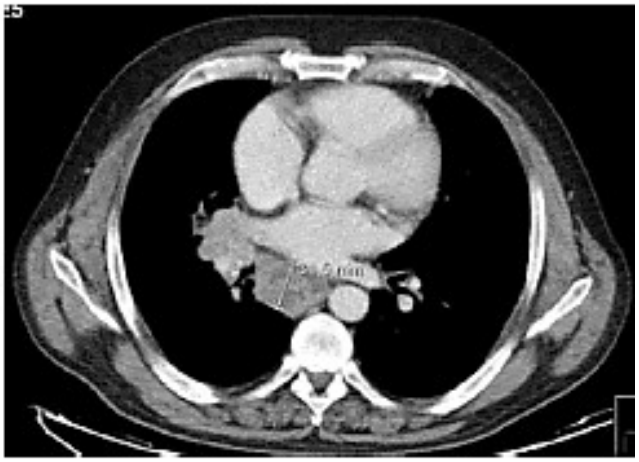

Figure 4: Node in the azygos-esophageal recess 36 × 48 mm.

A Positron Emission Tomography-CT- FDG showed a hilar mass on the right lung with a maximum SUV of 8.4, right hilar nodes (SUVmax 1.3), subcarinal nodes. Right adrenal nodule was identified (SUVmax 2.3) (Figure 5).

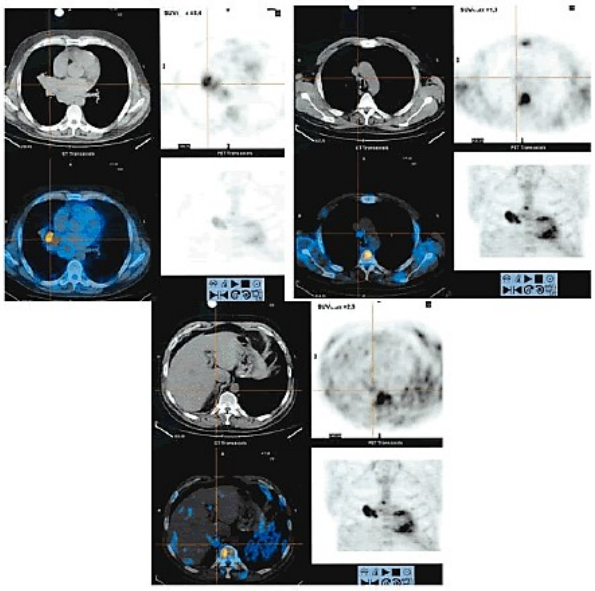

Figure 5: Right hilar mass (SUVmax 8.4), right hilar nodes (SUVmax 1.3), subcarinal nodes. Right adrenal nodule (SUVmax 2.3).

The patient was staged as a clinical T2aN2M1b, Stage IV, and was proposed in a Multidisciplinary Team (MDT) for palliative chemotherapy [1,2]. Being the patient´s performance status aggravating (PS 1), weighing at this time 98 kg, with symptomatic, namely sporadic haemoptysis, chemotherapy was started with carboplatin AUC 5, day 1 and gemcitabine 1000 mg/m2, day 1 and 8, every 21 days. A consult of cardiology was requested. Genetic tests were also asked for (EGFR mutational status and translocation of ALK). After three cycles of chemotherapy, the patient improved his PS, no dyspnoea, no haemoptysis. Thoracic CT scan showed stable disease. He continued until six cycles of chemotherapy were performed. The following CT scan performed, showed progressive disease in the hilar right mass, right paratracheal node, node in the azygos-esophageal recess and a new hypervascular image in the V hepatic segment suspicious of a metastatic lesion, but no right adrenal node (Figures 6-8).

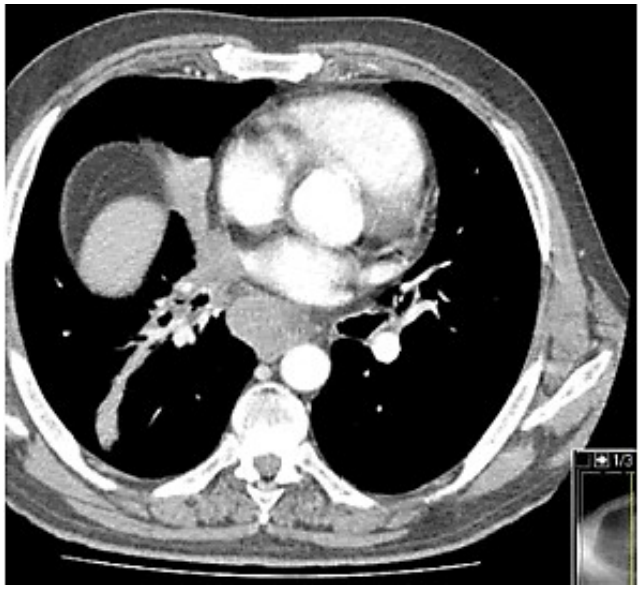

Figure 6: Right hilar mass 49 × 32 mm.

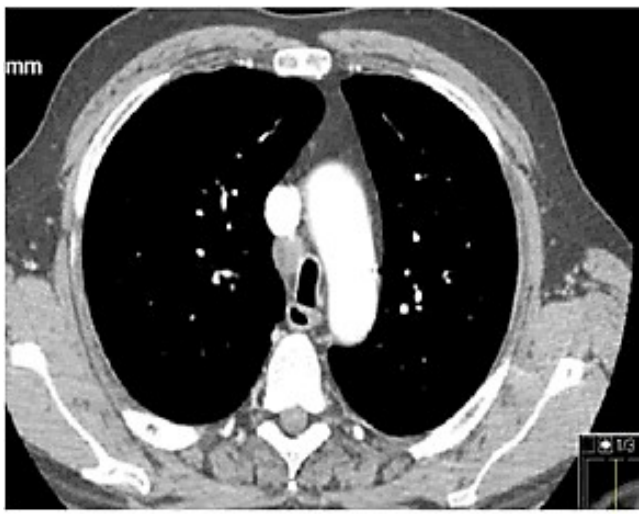

Figure 7: Right paratracheal node 20 × 15 mm.

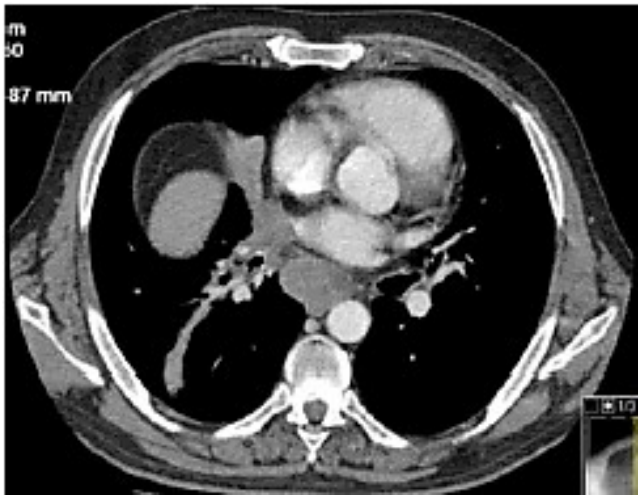

Figure 8: Node in the azygos-esophageal recess 45 × 28 mm.

Clinically the patient lost 8 Kg (90 Kg) in two months, PS was stable. Echocardiogram showed improvement in the left ventricle ejection fraction with 40%, but maintained global hypokinesia. Genetic results showed a non-mutated EGFR and no ALK translocation was identified. At this stage we have a 58 year old man with PS1, with progressive clinical and documented imaging disease. No authorisation was obtained from our hospital to performed Nivolumab in a second line of treatment 3, and so docetaxel 75 mg/m2, every 21 days was started. Hepatic image was clarified by an abdominal ultrasound and confirmed to be a haemangioma. Thoracic CT scan after 3 cycles of docetaxel showed stable disease. After six cycles, the patient maintained also stable disease (Figure 9), and recovered weight (95 kg). The patient stayed in surveillance.

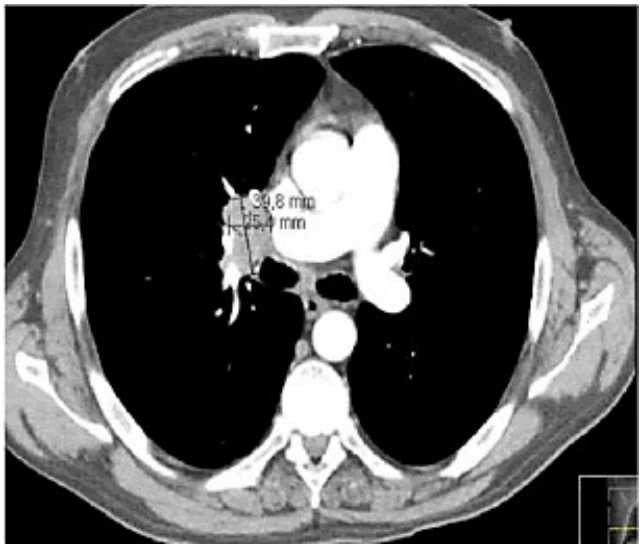

Figure 9: Right hilar mass 39.8 × 25.4 mm, subcarinal nodes with 13 mm.

Nine weeks after having stopped chemotherapy, the CT scan showed progressive disease of the hilar right mass (Figures 10-12). Echocardiography re-evaluation showed improvement of the left ventricle ejection fraction of 64% and no signs of invasion of the left atrium. Clinically, he presented with a PS of 0 and his weight increased to 95 kg.

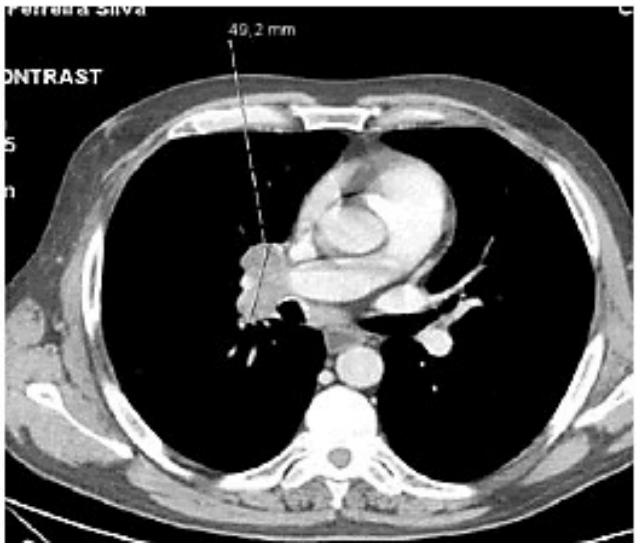

Figure 10: Right hilar mass 49.2 mm.

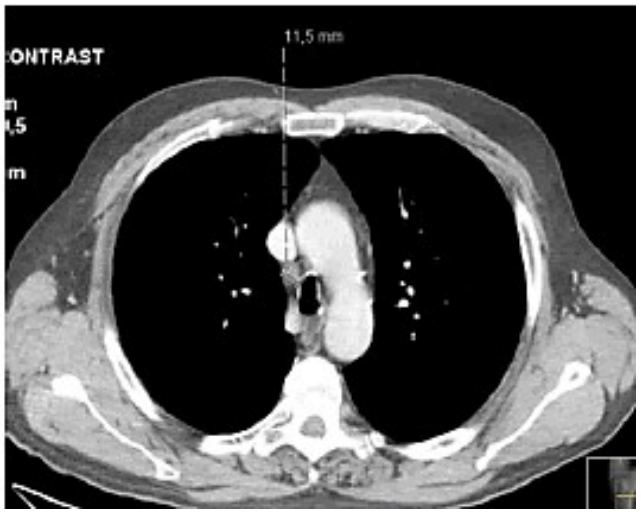

Figure 11: Right paratracheal node 11.5 mm.

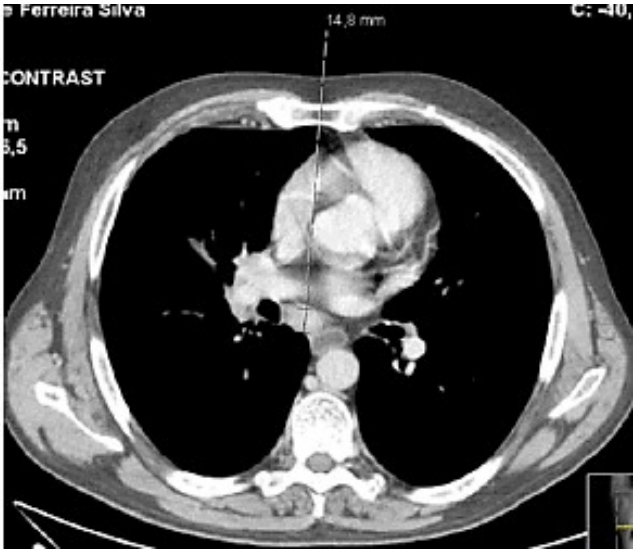

Figure 12: Node in the azygos-esophageal recess 14.8 mm.

At this moment the options were: considering that the adrenal node was benign and no liver metastases, radiotherapy was considered in MDT discussion, but rejected due to cardiotoxicity risk and eventual invasion of the left atrium in the first CT thoracic angiogram [4,5]. If progression of the principal mass was assumed, a third line of treatment should be considered (Figure 13).

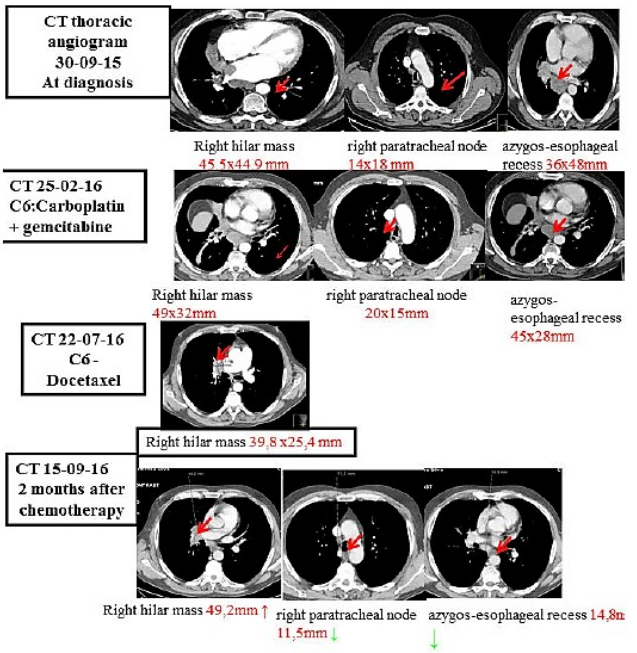

Figure 13: Evolution of disease.

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Discussion

The author presents a clinical case from a Portuguese public hospital, in a country with economic instability and a short budget for health care [6]. Due to bureaucratic strategies the patient finally managed to perform immunotherapy in third line with an apparently good clinical response.

The main point is that patients with a good health care insurance can have access to better therapeutics in a faster way, be submitted to less toxic therapies and improved quality of live [7]. This patient in particular was lucky to have a good PS since the beginning, aged 58 years at diagnosis, submitted to two lines of chemotherapy with a reasonable response 1 year after diagnosis. Could the life expectancy and quality of life have been changed in the long run, if nivolumab could have been started earlier? Studies show that the overall survival, response rate and progression free survival with nivolumab versus docetaxel in second line in squamous histology is superior with nivolumab and also, with improved duration of response after having stopped treatment and with a better quality of life [3]. However each patient has its' own characteristics, as well as each tumour, not neglecting that cancer is a heterogeneous disease in constant change, as we treat it with different drugs. The second point is shouldn't everyone have the same opportunities of treatment within each country [8,9] where all of us pay taxes for the national health care system. Obviously, those that can afford a private insurance might be more fortunate.

Cancer is not only a heterogeneous disease in constant change, which lives in our country in an economic survival crisis, with different opportunities within public hospitals. It´s not only a fight anti-cancer but unfortunately, also a fight against each public hospital budget survival [10].

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