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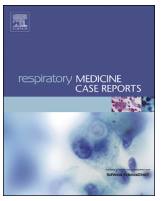

Case Report

A lepidic adenocarcinoma mimicking an eosinophilic lung

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ABSTRACT

Lepidic adenocarcinoma is a cancer with atypical radiological presentation making its diagnosis difficult and late. Here,we report the case of a 64-year-old man, who presented with respiratory distress his thoracic CT showed ground glass areas and diffuse condensations with blood hypereosinophilia. He was diagnosed to have eosinophilic lung and was placed on corticosteroid therapy but he did not show any improvement. A CT-guided biopsy showed lepidic adenocarcinoma.

1. Introduction

Eosinophilic lung is a set of heterogeneous pathologies whose common point is the infiltration of lung tissue by eosinophils. Its radiological presentation most often involves ground glass patches and bilateral condensations. It is diagnosed by the presence of eosinophils in the bronchoalveolar lavage (BAL) or in the induced sputum, and its treatment is based on a systemic corticosteroid therapy which rapidly improves the respiratory symptoms. Otherwise, another diagnosis should be considered [1,2].

Lepidic adenocarcinoma is a recent entity described in the new 2015 WHO classification of primary lung cancers [3,4]. It presents radiologically with diffuse alveolar opacities, pulmonary nodules or areas of ground glass, which is common to several pathological entities, thus making its diagnosis difficult and most often late [5].

Here, we describe the case of a patient presenting a lepidic adenocarcinoma mimicking an eosinophilic lung.

2. Case presentation

A 64-year-old patient, chronic smoker for 40 years, had a dry cough since 6 months along with mMRC stage 2 dyspnea which became complicated 1 month ago by the worsening of dyspnea resulting in mMRC stage 3, all evolving in a context of preservation of general condition. On clinical examination, his desaturation level was at 78% in ambient air with polypnea at 25 cycles per minute, cyanosis of the extremities and signs of respiratory struggle. The pleuropulmonary examination revealed crackles on the left.

The chest X-ray showed bilateral alveolar opacities.

At the biological level: hypereosinophilia was noted on several occasions (going up to 1170), a hemoglobin level at 15.9g/l, white blood cells at 13,700/mm^3 and platelets at 320,000/mm^3, a correct renal function, a negative covid 19 PCR test, a negative genexpert for tuberculosis in the sputum and a negative cytobacteriological examination of the sputum. The total IgE level was not done.

Faced with hypereosinophilia, a stool parasitology was requested which returned negative.

The thoracic CT scan (Fig. 1) showed diffuse foci of pulmonary parenchymal condensation, involving the various pulmonary lobes with areas of ground glass.

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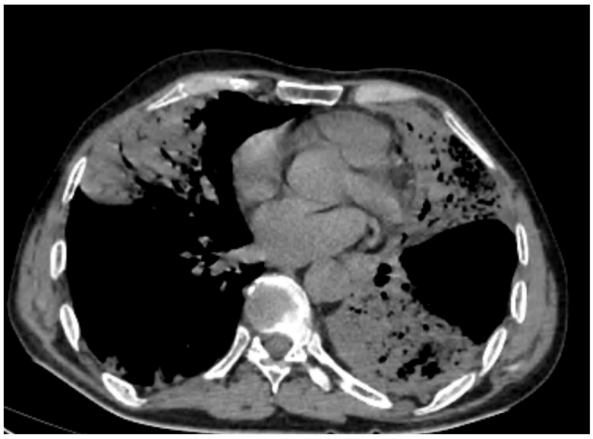
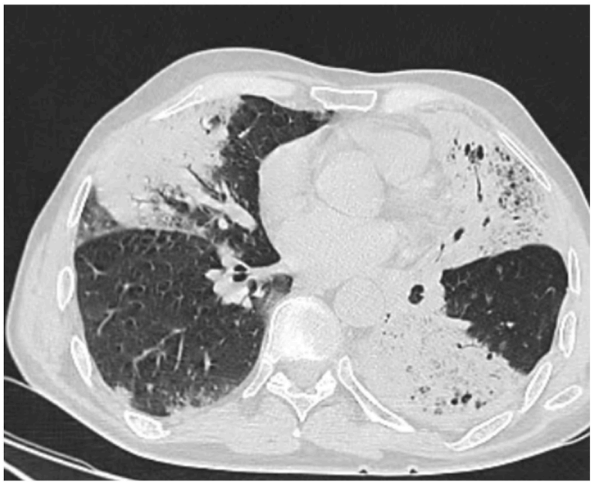


a



b

Fig. 1. Chest CT (a: mediastinal section, b: parenchymal section) showing bilateral consolidations with ground glass.

A bronchial fibroscopy was performed showing a normal endoscopic appearance. Staged bronchial biopsies returned to normal, fibroaspiration in search of neoplastic cells was done but did not detect tumor cells and the patient did not tolerate BAL.

A spirometry objectified a ventilatory disorder of restrictive pace with a forced vital capacity at 3.02l or 43% and an arterial blood gas: hypoxia at 40 mmHg.

The diagnosis of the eosinophilic lung was retained on the radiological aspect and hypereosinophilia and the patient was put on a bolus of corticosteroids followed by an oral corticosteroid therapy at a dose of 1mg/kg/day. The initial evolution after 15 days was marked by an improvement in the patient's dyspnea with an increase in saturation to 83% in ambient air, a slight decrease in the eosiniphil level to 1000/mm^3 and radiological stability. The patient was discharged on oral corticosteroid therapy and oxygen therapy 3l per minute.

The follow-up at 2 months showed reaggravation of symptoms with stage 3 dyspnea, 78% desaturation on AA and the appearance of recurrent episodes of low abundance hemoptysis. Eosinophil count increased to 3630/mm^3. A thoraco-abdomino-pelvic CT scan was redone showing the same radiological appearance with no lesions outside the lung. The diagnosis of eosinophilic lung was questioned, therefore a CT-guided biopsy was performed, and the result showed tumoral proliferation consisting of atypical cells cohesively colonizing the alveolar structures without signs of stromal invasion which is histological appearance of a non mucinous lepidic adenocarcinoma (Fig. 2).

The case of our patient was presented in a multidisciplinary consultation meeting (RCP) and the diagnosis of invasive lepidic adenocarcinoma was retained because of the radiological presentation. It was decided to put the patient on chemotherapy and to search for the EGFR mutation.

The patient received his first course of carboplatin – pemetrexed.

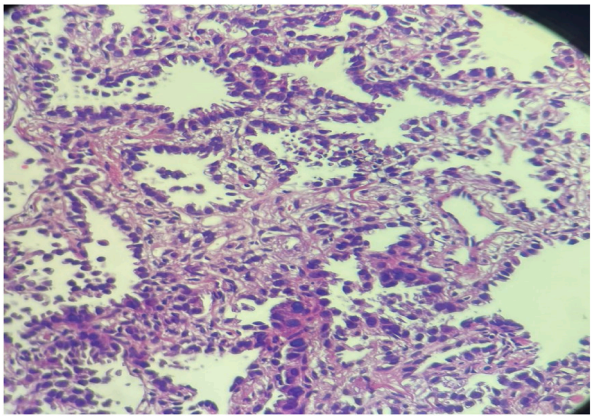


Fig. 2. Adenocarcinoma with lepidic architecture: tumoral proliferation made of atypical cells, cohesively colonizing the alveolar structures (x 40).

3. Discussion

Eosinophilic pneumonia is defined by pulmonary involvement made up of pulmonary eosinophilia (>25% in BAL) associated or not with blood eosinophilia (>500 elements/mm^3). Its causes are mainly parasitosis, drugs, idiopathic eosinophilic lung (acute or chronic) and if there are systemic signs: eosinophilic granulomatosis with polyangiitis or hypereosinophilic syndrome [1,2]. Some cancers, including lung cancer, can cause hypereosinophilia secondary to the dysregulation of cancer cells, which begin to produce eosinophil growth factors such as interleukin 5 [6]. Thus, in 0.6 in 1.5% of cases, hypereosinophilia corresponds to a paraneoplastic syndrome (biological abnormalities associated with certain cancers but not related to local tumor invasion or metastatic, related to the secretion of cytokines pro-inflammatory or hormonal peptides by the tumor, regressing or coming back with treatment of the tumour), and may be associated with an infiltration intratumoral by polymorphonuclear eosinophils. A significant number of cancers is accompanied by a blood eosinophilia which evolves in an indisputably paraneoplastic and peritumoral inflammatory infiltrate is sometimes mainly due to PE. It is thus described a blood hypereosinophilia in 0.6–1.7% of solid tumors malignancies "all comers" diagnosed in centers specialized and 0.6% of lung carcinomas [7,8]. Hypereosinophilia can be so important to diagnosis that it is considered the first manifestation of this cancer. This was the case of our patient who presented with acute respiratory failure associated with: a very high level of eosinophils, a negative etiological assessment which made it impossible to perform a BAL or a CT-guided biopsy and mistakenly led us to the diagnosis of eosinophilic lung. But the absence of a spectacular response to corticosteroids led us to look for another diagnosis, hence the CT-guided biopsy revealing lepidic adenocarcinoma.

Lepidic adenocarcinomas (formely known as bronchioloalveolar carcinomas) represent a rare entity (2–7% of non-small cell lung cancers). The latest IASLC/ATS/ERS 2011 classification categorized bronchioalveolar carcinomas into adenocarcinomas in situ, minimally invasive adenocarcinomas and invasive adenocarcinomas while the 2015 WHO classification removed the definition of mixed invasive adenocarcinoma and replaced it with the definition of invasive adenocarcinoma whose predominant architecture is described (lepidic, acinar, papillary, micropalpular, solid, mucinous, etc.) [3,5,9].

Bronchioloalveolar carcinoma can have several radiological presentations: it can show as a solitary nodule or a mass, a localized condensation with air bronchogram or multifocal and diffuse involvement (at 30% occurrence, it is the most common form with the worst prognosis) [5,10]. The latter corresponds to the radiological presentation of our patient with bilateral diffuse condensations and areas of ground glass. Most often, in studies of records of patients with bronchioloalveolar disease, such as the one conducted by Akira et al., in 1999 involving 38 records, most patients show a mixture of these lesions [10].

The pathological diagnosis of lepidic adenocarcinomas can be obtained by small biopsies showing atypical Clara-type cells and type II pneumocytes colonizing the surface of the alveolar walls step by step with the foci of invasion exceeding 5mm more or less vascular emboli or pleural infiltration [3].

Given the recent identification of several molecular abnormalities in lepidic adenocarcinomas (EGFR, KRAS mutations, ALK and ROS1 rearrangement), a search for these abnormalities is essential because it allows targeted therapies and confers a better prognosis on these patients (median survival of 13–17 months) [9,11].

The localized forms are treated using surgery. Although, the diffuse forms are known to be resistant to conventional chemotherapy, the third generation chemotherapy provides prolonged survival [9]. Some researchers have proposed lung transplantation for the diffuse forms without distant metastases or mediastinal lymph node involvement, with encouraging results [9].

The evolution of lepidic adenocarcinomas is marked by a lepidic and aerogenic progression explaining the late development of extra-pulmonary metastases [9,11]. This is the case of our patient who did not present extrapulmonary localization on thoraco-abdomino-pelvic CT.

4. Conclusions

Lepidic adenocarcinoma is a cancer most often diagnosed late due to its atypical radiological presentation. It would be wise to perform a CT-guided biopsy from the outset in patients with risk factors for lung cancer or those who do not show rapid clinical improvement under another therapy.

Declaration of competing interest

None.

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