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Case Report

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An Unusual Case of Eosinophilic Pneumonia

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Abstract

Chronic eosinophilic pneumonia (CEP) or Carrington disease is an eosinophilic pneumonitis of unknown etiology. There are no strict diagnostic criteria for CEP, it's based on well-defined clinical and radiological characteristics associated with blood and/or alveolar eosinophilia. Symptoms are non-specific and usually include respiratory and general symptoms, which are either subacute or chronic. In this article, we report a particular case of CEP for a middle-aged woman without asthma in order to present the elements of diagnosis especially those related to clinical and radiological findings.

Keywords: *Carrington disease, eosinophilic pneumonitis, hyper eosinophilia*

Introduction

Chronic eosinophilic pneumonia (CEP) was first described by Carrington in 1969. It's a rare disorder that likely affects less than 1 person per 100,000 per year, and no known trigger exists in the majority of cases [1] characterized by subacute or chronic respiratory and general symptoms, alveolar and/or blood eosinophilia, and peripheral pulmonary infiltrates on chest imaging. In this article, we aim to discuss and report unusual case of chronic eosinophilic pneumonia, in order to present the elements of diagnosis, especially clinical and radiological findings.

Observation

A 53-year-old patient, nonsmoker, with exclusive seasonal allergic rhinitis, has been presenting incessant dry cough for six months with exertional dyspnea associated with diffuse inflammatory polyarthralgia. These symptoms had worsened 10 weeks before admission to hospital, by the installation of untypical chest pain and dyspnea ranked grade 4 according to the mMRC scale (modified medical research council), evolving in a context of apyrexial and deterioration of the general health state.

The clinical examination has revealed a blood pressure of 110/70mmHg, a pulse rate of 96 beats per minute, a respiratory rate of 25 breaths per minute, and oxygen saturation of 92 %. Auscultation of her chest revealed inspiratory crackle and expiratory wheezes in both lungs. Chest X ray noted pulmonary infiltrates labile peripherals (figure 1).

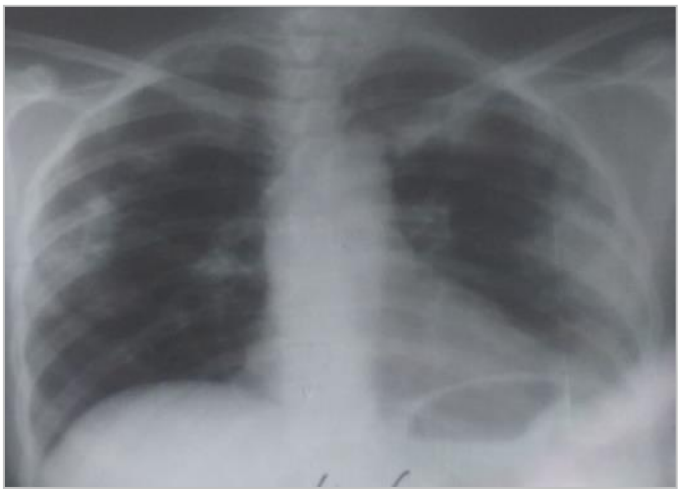

Figure 1: Chest X ray noted alveolar type infiltrative opacities with peripheral topography, bilateral: Aspect of "reverse pulmonary edema".

Indeed, urgent laboratory testing was required to eliminate a cardiovascular emergency, so the cardiac markers were normal, with high D-dimer concentration at 1500ng/ml. Therefore, thoracic angio-scan eliminates proximal pulmonary embolism. High-resolution computed tomography (HRCT) scan of the chest reveals peripheral bilateral alveolar condensation outbreaks, at the upper and middle segments, associated to bilateral multifocal frosted glass pattern (figure2).

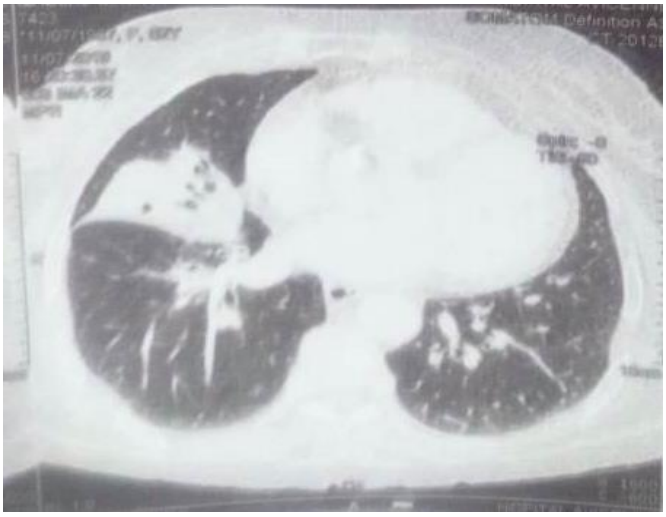

Figure 2: HR-CT of the chest reveal peripheral bilateral alveolar condensation outbreaks, at the upper and middle segments, associated to bilateral multifocal frosted glass pattern.

Laboratory data revealed inflammatory syndrome, a peripheral white blood cell count of 10.46/mm3 with hyper eosinophilia at 1100/ul. Other blood biochemical examinations showed no specific abnormalities. Against this backdrop, bronchoalveolar lavage (BAL) was performed, the percentage of eosinophils was 12%, tuberculosis test results were negative, and the cultures for aerobic and anaerobic bacteria remained sterile. Pulmonary function tests or PFTs showed mixed obstructive and restrictive ventilator defect with VEMS/CVF at 69%, CPT at 70 %, with decrease in carbon monoxide transfer at 64%. Blood gas analysis showed hypoxemia at 72 mmHg with normocapnia.

After removing all other causes of the eosinophilic lung, the final diagnosis consisted in Carrington's disease, since systemic steroid therapy was given for 6 months (0.5 mg/[kg d]) ,which led to spectacular clinical and radiological improvement after 2 months of treatment (figure3).

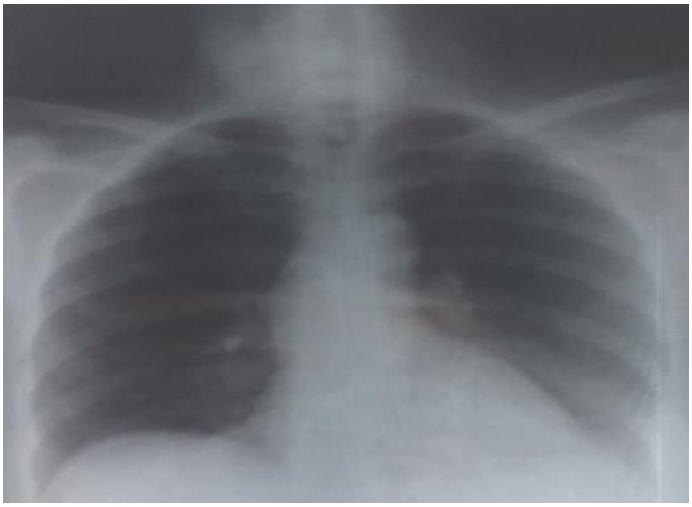

Figure 3: Chest X ray shows radiological cleansing after 2 months of treatment.

Discussion

ICEP is a rare disorder due to unknown cause occurring in 0 to 2.5% of patients with diffuse infiltrative pulmonary disease. It was first described by Carrington et al in 1969 (Carrington disease) [2], it is twice as frequent for women as for men [2]. ICEP may affect every age group but it is rare in childhood. The survey [3] showed a low prevalence of acute eosinophilic pneumonia and CEP in the pediatric population. In all published cases [4,5], we often find a positive family history of asthma or other atopic diseases, in two thirds of patients, which can precede the disease of over 20 years. Recently, cases of eosinophilic pneumonia have been reported after radiotherapy for breast carcinoma [6]. Otherwise, tobacco appears to have a protective role [4,5] because of its low prevalence (6.5%) among active smokers.

There are no strict diagnostic criteria for ICEP. Diagnosis is usually based on the association of [7,13]:

- Respiratory symptoms of usually more than 2 weeks duration.
- Alveolar and/or blood eosinophilia (alveolar eosinophilia ≥ 40% at bronchoalveolar lavage (bal) differential cell count; blood eosinophilia ≥ 1000/mm3).
- Pulmonary infiltrates with usually a peripheral predominance on chest imaging.
- Exclusion of any known cause of eosinophilic lung disease.

Symptoms are non-specific and usually include both respiratory and general symptoms, which are either subacute or chronic [2]. They combine respiratory signs with cough, dyspnea, chest pain and constantly, alteration of the general health state with fever and night sweats, sometimes leading to tuberculosis disease. Pulmonary auscultation is found in one-third of patients with crickets or sibilants [3,2]. Extra thoracic features are often missing in CEP, when the symptoms and signs of extra-pulmonary involvement are present, a diagnosis of Churg-Strauss syndrome (CSS) or idiopathic hyper eosinophilic syndrome (IHS) should be considered [7]. In our case study, the patient presented mainly respiratory symptoms with general signs..Our observation responds to the typical radiographic finding. According to published studies, CEP is characterized by peripherally located, with ill-defined dense opacities with non-segmental distribution [9]. Infiltrates can be either unilateral or most often bilateral. The most distinctive chest X-ray images mimic the photographic negative of acute pulmonary edema this typical pattern, however, is absent in the majority of cases of CEP and is not specific for the disorder [8]. CT-scan may show opacities that are not discernible on [7], it is often characterized by Peripheral and patchy consolidation with upper lobe dominance [7,9], as mediastinal lymph node enlargement has been described [7].

Circulating eosinophilia is present in most cases of literature, usually in excess of 1000/mm3 [7,3]. However, in the absence of significant blood eosinophilia, a diagnosis of CEP is supported by the demonstration of bronchoalveolar lavage eosinophilia (≥ 40%) [7]. In our

case, the patient has been a hypereosinophilia of the blood at 1100/ul.

While this diagnosis is entertained, a BAL should be performed as early as possible, as it is the diagnostic study of choice. Bronchoalveolar lavage in CEP always reveals abnormally high eosinophil levels [7] Lung biopsies are rarely needed to make the diagnosis, when performed, lung biopsy shows an interstitial and alveolar inflammation with a clear-cut predominance of eosinophils [4,11]. Infiltration of pulmonary vessels with eosinophils may be observed but necrotizing or granulomatous vasculitis are not present in ICEP [12]. The percentage of eosinophiles in our case was 12%.

Pulmonary function tests are important for disease monitoring, particularly in the context of frequent association with asthmatic disease. In acute times, they often show restrictive ventilatory disorder and a decrease in carbon monoxide transfer [4]. CEP can be associated with either a restrictive or an obstructive pattern on pulmonary function tests. The latter pattern is more often present in patients with a history of asthma [4,10]. Regarding to our patient, she presented a mixed ventilator defect with reduce in carbon monoxide transfer a 64%.

The therapeutic approach is not standardized, but there is general agreement that treatment of CEP is based on systemic steroid therapy [3,7]. After initiation of treatment, symptoms as well as blood eosinophilia regress within a few hours and chest imaging results normalize within a few days [7]. This dramatic response to therapy has led to the consideration of corticosteroid challenge as a diagnostic test for CEP [9]. So, our patient was put under systemic steroid therapy and the evolution was remarkably favorable a few days of treatment.

The short-term prognosis for patients with CEP is generally favorable, given the remarkable and timely clinical improvement with corticosteroids. Around half of patients initially diagnosed with CEP showed clinical improvement, without relapse or need for repeat treatment [5].

Conclusion

Carrington's disease is a rare pathology with non-specific criteria, which leads to a diagnostic delay. The combination of chronic respiratory symptomatology, alveolar infiltrate, eosinophilic alveolitis at the LBA with a spectacular response to corticosteroid therapy makes it possible to perform the diagnosis without recourse to pulmonary biopsy.

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