Case Report

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Chron's Disease and Bronchial Stenosis: A Case Report

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ABSTRACT

Keywords: Bronchial Stenosis; Airways Stenosis; Chron's Disease Pulmonary Manifestations; Chron's Disease Extraintestinal Manifestations; Airways' Disease In Inflammatory Bowl Diseases

Abbreviations: CD: SCrohn's Disease; IBD: Inflammatory Bowel Disease; UC: Ulcerative Colitis; ILD: Interstitial Lung Disease; IL: Interleukin; TNF: Tumor Necrosis Factor; CT: Computer Tomography

Introduction

The pulmonary disease associated with inflammatory bowel disease (IBD) is thought to be a rare disorder and needs to be separated from viral complications, drug side effects from treating IBD [1] (like mesalazine), and other more prevalent autoimmune lung diseases. Crohn's disease (CD) is a chronic inflammatory disorder commonly involving the gastrointestinal tract. However, several extra intestinal signs of Crohn's disease, including pyoderma gangrenosum, erythema nodosum, polyarthritis, episcleritis, pericholangitis, and thromboembolism, are also possible. Ulcerative colitis (UC) predominantly impacts the joints, skin, liver, and eyes in addition to its intestinal signs [2]. Despite the rarity of UC associated to pulmonary diseases in clinical settings, it is crucial to identify pulmonary problems as soon as possible in order to reduce patient mortality [3]. As shown in (Table 1), the range of lung involvement in IBDs ranges from asymptomatic abnormal involvement to interstitial lung disease (ILD) [4]. Patients with IBD reported obstructive respiratory disorders as asthma (8.6%) and chronic obstructive lung disease (8.7%) more commonly, followed by pleural diseases (1.9%), in a recent study [5]. Patients with IBD had a 46% higher incidence of bronchiectasis, a 52% higher incidence of pulmonary vasculitis and interstitial pneumonia, a 35% greater risk of lung nodules, a 16% higher incidence of pulmonary fibrosis, and a 5.5% higher incidence of asthma compared to the non-IBD sample. Patients with IBD who had CD had a 34% reduced age/sex-adjusted risk for bronchiectasis, a 56% lower risk for pulmonary vasculitis, a 14% lower risk for pleural illnesses, and so on, compared to those who had UC and approximately 30% higher risk for chronic obstructive pulmonary diseases [5] (Figure 1). Some of the following pathways have a role in the pathophysiology of IBD-related lung abnormalities: colonic and respiratory epithelia both feature columnar epithelia with goblet cells and submucosal mucus glands and share an embryonic origin from the primordial foregut; the lungs and gastrointestinal tract also have submucosal lymphoid tissue, which is important for host mucosal defense [6,7]. As a result of epithelial exposure to

common antigens by ingesting and inhalation, comparable pathologic alterations may occur that result in inflammation and sensitization of the lymphoid tissue [8]. Tumor necrosis factor (TNF)-, interleukin (IL)-1, IL-2, and IL-6, as well as other circulating cytokines, can all be produced by activated inflammatory cells in the gut tissues. These and other mediators can control the leukocyte movement, modulate endothelial cell adhesion molecules, promote the formation of damaging reactive oxygen metabolites, and cause lung parenchyma damage [9-11]. Due to reports of bronchopulmonary disorders developing after colectomy [12], a continuous intestinal irritation is not a requirement for the start of respiratory changes. Any area of the lungs might be affected by pulmonary abnormalities in IBD, which can appear years after the bowel disease first manifests. These could be overt or subclinical, and they are unrelated to how long IBD has been diagnosed (Figure 2).

Table 1 [16-20].

Airway disease (panbronchiolitis, bronchiolitis obliterans organizing pneumonia and bronchiectasis)
Inflammatory tracheal stenosis
Pulmonary vasculitis
Thromboembolic disease
Pleural disease
Apical fibrosis
Sarcoidosis
Enteric-pulmonary fistulas
Langerhans cell histiocytosis
Manifestations resembling adverse drug toxicities
Pulmonary bullae
Lung cysts
Pulmonary function test abnormalities, in the form of restrictive, obstructive airway, and small airway disease

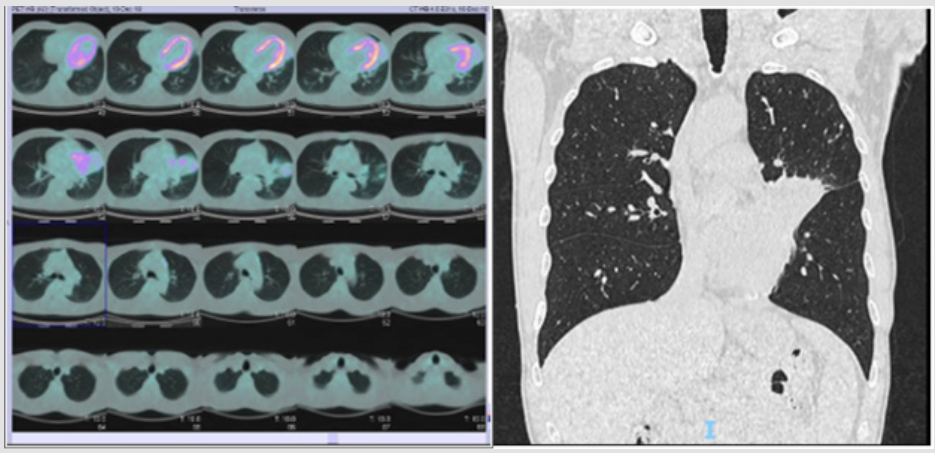

Figure 1.

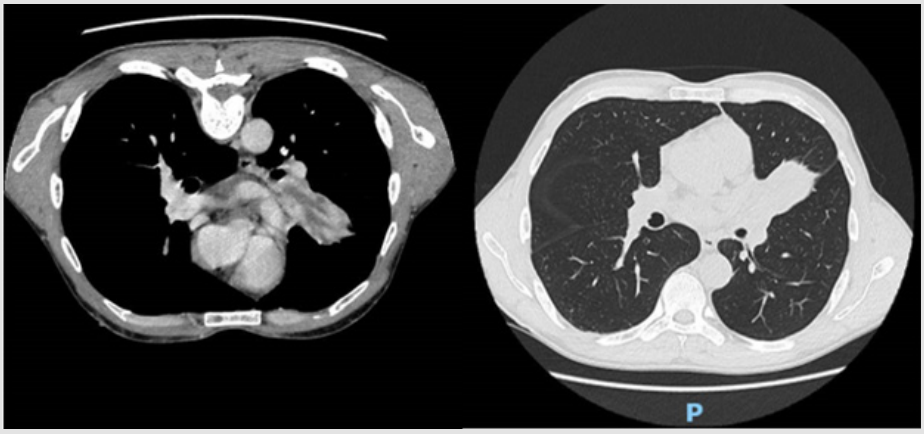

Figure 2: Thoracic CT scan.

The frequency of the pulmonary symptoms has been observed to differ depending on the stage of the disease, whether it is active or not, and even in individuals who have had a colectomy [13,14]. Patients who have diffuse ground-glass shadows in pulmonary computer tomography (CT) pictures, increasing dyspnea, and dry cough should be cautiously watched. Patients with central airway involvement frequently experience chest pain, wheezing, shortness of breath, difficulty exercising, purulent expectoration, and hemoptysis (Figure 3). Anti-asthmatic therapy is frequently initiated once asthma is diagnosed, but the rate of symptom control varies [15]. It is important to choose the glucocorticoid therapy carefully and to prevent against lung opportunistic infections. Combining cyclophosphamide and globulin may be a successful therapy approach [12].

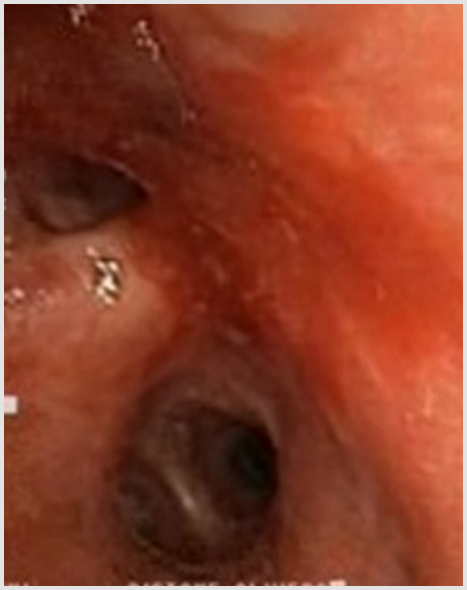

Figure 3: Left upper lobar bronchus in Bronchoscopy.

Case Presentation

A 49 year old male presented to the emergency department because of persistent cough and fever. A thoracic radiography and then a CT scan revealed radiologic infiltrates localized in the left lung, persistent after antibiotic therapy. The subject was non-smoker, working as a welder, with a history of Crohn's disease, under therapy with mesalazine, and rheumatoid arthritis, without therapy. Pulmonary function test showed a mild obstructive dysfunction with predicted values of FVC 130%, FEV1 109%, FEV1/FVC 0.67, TLC 119%, RV 111%. Diffusion capacity for CO was in range (114% of predicted). A CT/PET total body scan showed a metabolic activity in the lingula indissociable from the near thoracic pleura and the pericardium, in close contiguous relationship with hilar structures, and other multiple focal areas of metabolic activity in the right retromandibular, left paratracheal and sub-carina sites, probable lymph nodes. Then the patient underwent bronchoscopy which revealed a cicatricial stenosis of the left upper lobar bronchus; during the procedure multiple transbronchial biopsies were obtained of the known left radiologic infiltrates PET +. The anatomopathological report indicate the presence of histiocytes, with an infiltrate of lymphocytes; no multinucleated giant cells or parenchymal granulomatous lesions was seen. (Figure 4) One year later the patient performed another CT/PET total body which showed a small size reduction of the known left radiologic infiltrates with metabolic activity, while the other focal areas didn't show metabolic activity. (Figure 5) Then the patients underwent another bronchoscopy with transbronchial biopsies, the histological findings were stackable with the last biopsies. This year, after three years, the patient repeated a thoracic CT scan, which showed an increase in size of the known bronchial stenosis in the lingula and atelectasis of its distribution territory. A few months later he presented at the emergency department because of an isolated episode of hemoptysis (Figure 6). Two years later the patient repeated a bronchoscopy with bronchial biopsies and bronchoalveolar lavage at the left upper lobar bronchus; because of the cicatricial stenosis it was impossible to explore its branches. The histological findings were negative for bronchial infection and showed normal lung tissue with septal fibrosis and granulocyte, without appearance of granulomatosis or malignities, stackable with the last biopsies [16-20].

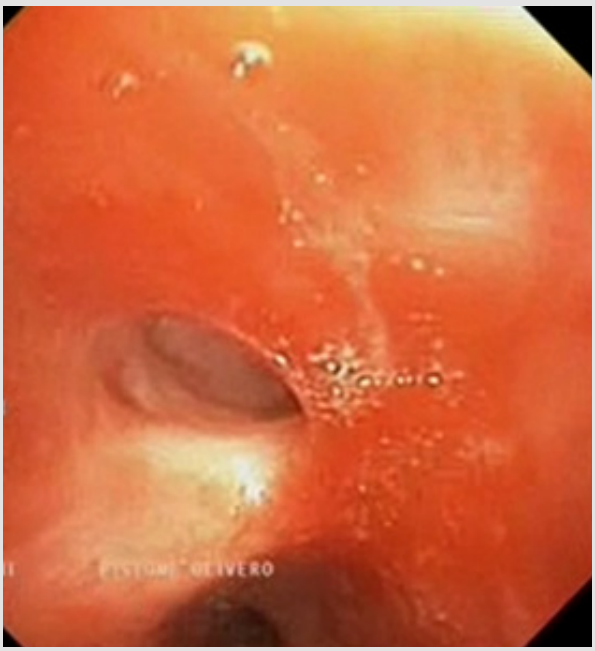

Figure 4.

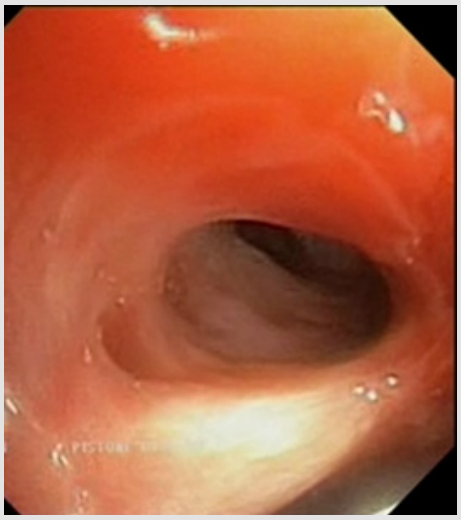

Figure 5: Left upper lobar bronchus in Bronchoscopy.

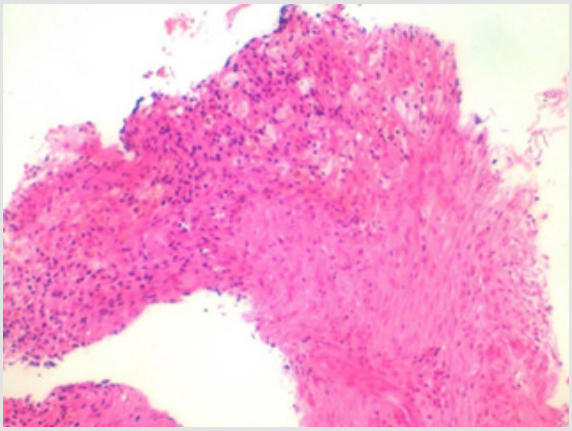

Figure 6.

Discussion

The spectrum of lung involvement in IBDs is broad, spanning from subclinical abnormal involvement to interstitial lung disease. Previously reported cases of airway stenosis related to Chron's disease involve the trachea [21,22] and the bronchi [23]. In Crohn's disease, the same types of respiratory lesions are described as in sarcoidosis: lymphocytic alveolitis and interstitial disease with tracheobronchial or parenchymal granulomatous lesions; large airway involvement with stenosis, bronchitis, bronchiectasis and pulmonary suppuration. This patient did not undergo local treatment for the cicatricial stenosis of the left upper lobar bronchus because we speculated that it was probably related to Chron's disease and the patient was therefore treated with biological therapy (Figure 7).

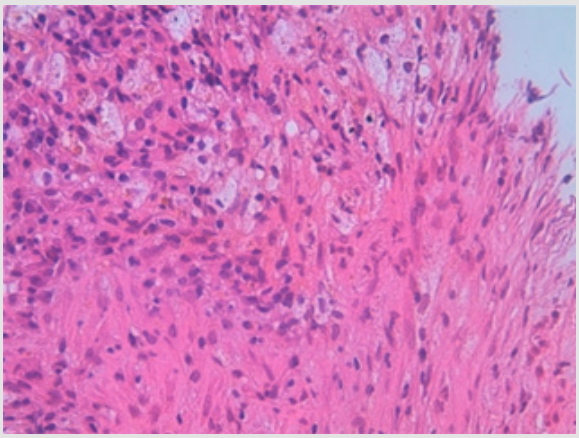

Figure 7: Anatomopathological aspect of the bronchial stenosis.

Conclusion

This case shows the importance of collecting an accurate anamnesis of patients with respiratory symptoms, in particular about comorbidities, in order to discover as soon as possible the underlying disease requiring a specific treatment. Moreover, this case highlights the importance of thinking about airway stenosis in patients with Chron's disease with pulmonary symptoms, as hemoptysis, or pulmonary radiological findings, as lung atelectasis.

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