Introduction

Ulcerative colitis and Crohn’s disease are traditionally grouped together under the term inflammatory bowel disease (IBD). It has been known for some years that lung disease can exist in patients with IBD. 

Diseases of the central airway, bronchi, and lung parenchyma are among the most common forms of lung involvement in IBD patients. Bronchiectasis is frequent and almost always appears after the onset of disease and in close association with inflammatory activity. However, reports of the appearance of bronchiectasis following colectomy for an exacerbation of IBD have been rare. 

We report the case of a 36-year-old man with severe ulcerative colitis who, following a total colectomy, developed bilateral bronchiectasis 12 months after surgery and for whom both preoperative and postoperative computed tomography scans were available.

Key words: Bronchiectasis. Ulcerative colitis. Inflammatory bowel disease.
total colectomy with total ileostomy due to an exacerbation of ulcerative colitis with toxic megacolon. He was also diagnosed with hereditary thrombophilia (factor V Leiden mutation) after suffering a pulmonary thromboembolism in 1998 and deep venous thrombosis in 1999 following withdrawal of an oral anticoagulant; a brother of the patient was also subsequently diagnosed with the genetic defect. In March 2002 a HRCT scan was taken in the emergency department of our hospital to confirm or rule out suspected pulmonary thromboembolism, but no signs of pulmonary thromboembolism or alterations of the lung parenchyma were observed (Figure 1).

In September 2003 he was admitted to our department with right-sided subscapular pleuritic pain and frank hemoptysis of 15 mL that had begun 24 hours earlier, with no clinical picture of infection or fever. The patient had had no previous episodes of hemoptysis, although he presented a clinical picture of abundant mucopurulent morning expectoration, with no infection, dating from January 2003. On examination the patient was found to be well perfused and without fever or dyspnea at rest. Arterial blood pressure was 120/70 mm Hg and arterial oxygen saturation 92%. Heart and lung auscultation revealed only rhonchi in the apical and medial regions of the right hemithorax and isolated crackles in the right-mid axillary line. Examination of the abdomen revealed only a laparotomy scar and the ileostomy bag. The lower limbs showed no edema or signs of deep venous thrombosis. Biochemistry was normal and the hemogram showed no evidence of anemia, leukocytosis, or neutrophilia. Coagulation testing showed an international normalized ratio of 2.4 with prothrombin activity of 34%. D-dimer assay results were negative. Arterial blood gases measured at an inspiratory oxygen fraction of 21% showed a PaO$_2$ of 62 mm Hg, PaCO$_2$ of 42 mm Hg, pH of 7.41, and arterial oxygen saturation of 92%. The chest x-ray taken upon admission revealed an alveolar infiltrate with an air-bronchogram sign in the right upper lobe. Sputum smear examinations and cultures were negative. A HRCT scan of the chest revealed signs of bilateral bronchiectasis with upper-lobe predominance, although the lower lobes were also involved. Bleeding bronchiectasis was found at the apex of the right lung (Figure 2). Bronchoscopy yielded no significant findings, and the microbiological samples obtained were negative for common pathogens, acid-fast alcohol resistant bacilli, opportunistic pathogens, and fungi. Bronchoalveolar lavage showed an elevated neutrophil concentration. Lung function tests were performed with the following results: forced expiratory volume in 1 second (FEV$_1$), 70% (2.93 L); forced vital capacity (FVC), 88.2% (4.41 L); ratio of FEV$_1$ to FVC, 66%, and maximal midexpiratory flow rate, 30%. Carbon monoxide diffusing capacity was normal, and a methacholine challenge test was negative. Clinical signs had disappeared 24 hours after admission. The patient was discharged and prescribed a 6-month course of systemic corticosteroids at doses higher than those he had been using, together with inhaled corticosteroids and bronchodilators. The HRCT findings persisted at the 6-month follow-up visit, as did the signs of obstructed breathing observed in spirometry, although the daily sputum production had decreased.

**Discussion**

While it has been known for years that lung disease may be present in patients with IBD, the true incidence of lung involvement is unknown. True IBD-associated lung disease must be distinguished from lung disease produced as a consequence of the immune suppressant effects of treatment (mainly with sulfasalazine, in which lung involvement usually takes the form of eosinophilic pneumonia). True IBD-associated lung disease includes diseases of the airway, of lung interstitium, or other rarer conditions, such as serositis, necrobiotic nodules, and pulmonary vasculitis. In 85% of cases, pulmonary manifestations appear after the onset of IBD, and in 28% of cases symptoms begin following colectomy. The Table shows the wide spectrum of IBD-associated lung diseases

Camus et al say that the appearance of IBD-associated bronchiectasis has been described in several articles that generally show it to be more frequent in patients with ulcerative colitis than in those with Crohn’s disease and to appear in a period of time ranging from days to years following the onset of IBD.
symptoms. Frequency is not greater in any particular age group. The most common symptom is bronchial hypersecretion, and onset is usually insidious, but benign. The course of the disease usually parallels that of IBD, with episodes of respiratory symptoms occurring together with episodes of intestinal disease. This has led to the hypothesis that the disease might arise in the lung, as both the digestive tract and the lung have the same embryonic origin. Cases of bronchiectasis following colectomy lend support to this hypothesis; in such cases, when the organ that is the target of the inflammatory process is removed, the respiratory system becomes the next target of inflammatory damage. Foci of bronchiectasis are usually sterile and only show elevated levels of neutrophils in bronchoalveolar lavage.

A MEDLINE search located only 2 reports of bronchiectasis following a colectomy in a patient with IBD, and in neither case was a preoperative tomography scan available. In the case we report, given that a HRCT scan had been taken before the colectomy, we were able to compare the regions of the lung parenchyma before and after surgery.

IBD-associated bronchiectasis generally responds well to corticosteroid therapy, usually given in conjunction with treatment for the underlying disease. High-dose inhaled corticosteroids are the first treatment option, followed by oral prednisone (1-2 mg/kg/day). No evidence is available to support an optimal duration of treatment. Cases have been reported in which the development of side effects and poor control of the respiratory disease led to the use of serial bronchial lavages with a corticosteroid solution. Response to therapy in those cases was good. In the case we report, corticosteroid treatment led to a clinical but not to a radiologic or functional response.

The case we describe contributes to a better understanding of the pathogenesis of IBD-associated lung disease, especially in relation to colectomy. It also underscores the need for patients with IBD who develop respiratory symptoms to be referred to a pneumologist as soon as possible, as delay in defining the clinical picture and establishing a diagnosis can make repair of the lung parenchyma impossible even with proper treatment.

**REFERENCES**