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Case Report
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Lung Involvement in Ulcerative Colitis: Bronchiectasis and Pleural Effusion

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Abstract

Ulcerative colitis is primarily a gastrointestinal autoimmune disorder that can have significant extraintestinal manifestations, including pulmonary involvement. We describe a rare case of a 78-year-old male ulcerative colitis patient with bronchiectasis and pleural effusion. The patient was admitted for progressive dyspnea in exertion, cough, sputum, and lassitude of three months. He had undergone total colectomy for ulcerative colitis 30 years ago and used anti-inflammatory therapy for flares of his primary disease since then. Serum biochemistry was within normal limits. Chest x-ray showed right sided pleural effusion. Thorax CT revealed proximal middle lobe bronchiectasis and right pleural effusion. Microbiological and cytological examination of the BAL and pleural fluid did not yield any results. Differential diagnosis did not reveal any infectious, pulmonary, or systemic disease that could lead to bronchiectasis and pleural effusion. This case highlights the need for vigilance for pulmonary findings in patients with inflammatory bowel disease. Variable manifestations of associated lung involvement emerging in such patients underscores the significance for pulmonary assessment. Because of the significant incidence of systemic involvement in ulcerative colitis, manifestations in any organ should be considered as extraintestinal involvement of the primary disease rather than as an association with another primary organ disease.

Keywords:
ulcerative colitis, lung, bronchiectasis, pleural effusion

Introduction

Ulcerative colitis may have extraintestinal manifestations that may affect up to 50% of the patients involving the skin, eyes, joints, and lungs while nearly 30% of the patients with inflammatory bowel disease experience at least one extraintestinal organ involvement. A variety of lung manifestations have been reported associated with inflammatory bowel diseases [1-4]. Although pulmonary involvement is rare, it can manifest as airway inflammation, interstitial lung disease, or pleural complications. As these manifestations are usually subclinical and unassociated with the inflammatory bowel disease activity, they are often related to a primary lung disease that are easily misdiagnosed or overlooked. These symptoms may be unnoticed and are not associated with the inflammatory bowel disease activity by physicians, they may emerge more common than previously thought [2-4]. Although pulmonary manifestations in patients with inflammatory bowel disease have been described in the literature [5-9], their existence comes out as a diagnostic and a therapeutic challenge that warrants further investigation. While pulmonary involvement is rare, it can manifest as airway inflammation, interstitial lung disease, or pleural complications. For the provision of appropriate treatment, a thoroughly detailed differential diagnosis should always be considered, particularly in terms of infection, other primary, or drug-induced lung disorders. In this article, we present a case of ulcerative colitis in remission that has been complicated by pulmonary involvement illustrating the complexity of diagnosis and treatment of such patients.

Case report

A 78-year-old male patient with a 40-year history of ulcerative colitis presented with progressive dyspnea in exertion, cough, sputum production, and lassitude over the last three months. The sputum was white and grey in color with a volume of approximately 50 ml per day. The patient did not have fever or inflammatory bowel symptoms at admission with a stable condition of his primary disease. He denied any previous pulmonary symptoms or other disease of concern, was not under any kind of treatment, and was a non-smoker without alcohol or drug abuse. His ulcerative colitis was previously managed with total colectomy 30 years ago. Treatment with mesalamine, and intermittent corticosteroids had achieved temporary remission with infrequent exacerbations. Family history revealed papillary thyroid carcinoma, hypertension, type II diabetes in the father, and hypertension in the mother. Vital signs comprised a temperature of 36.8°, a respiratory rate of 22 breaths/min, and an oxygen saturation of 94% on room air. Physical examination demonstrated bibasilar fine crackles with decreased breath sounds on the left lower hemithorax, and mild tenderness in the lower left abdominal quadrant without guarding or rebound. Serum biochemistry showed an ESR of 28 mm/hr, a CRP of 6.8 mg/dL, and a WBC count of 10.800 cells/µL.

ECG displayed a regular sinus rhythm of 94/min with a normal axis. Chest PA and lateral x-ray (Figure 1) showed a moderate sized left pleural effusion up to one third of the left hemithorax. Pulmonary function tests designated a mild obstructive pattern with a FEV_1/FVC ratio of 64%. Thorax CT (Figure 2) revealed proximal segmental and subsegmental bronchiectasis in the lower lobes with a left pleural effusion extending to one sixth of the hemithorax with small ground opacities of one to two cm in

the right middle and left lower lobes. Pleural effusion was an exudate with a 50% lymphocyte ratio while microbiologic and cytologic assessment of the fluid was not diagnostic. Serum TNF-α, interleukin-1, and interleukin-6 levels were markedly high. Fiberoptic bronchoscopy was normal revealing only white-greyish secretions in both lower lobes. Microbiologic evaluation of the BAL fluid did not yield any organisms while cytologic examination did not reveal a pathologic finding. Following a detailed and a comprehensive differential diagnostic assessment of the patient, a definitive diagnosis of bronchiectasis and pleural effusion associated with ulcerative colitis was reached. The patient showed clinical improvement in dyspnea and cough over the next three months. Follow-up HRCT (Figure 3) performed 6 months later demonstrated a completely resolved pleural effusion and a stabile permanent bronchiectasis.

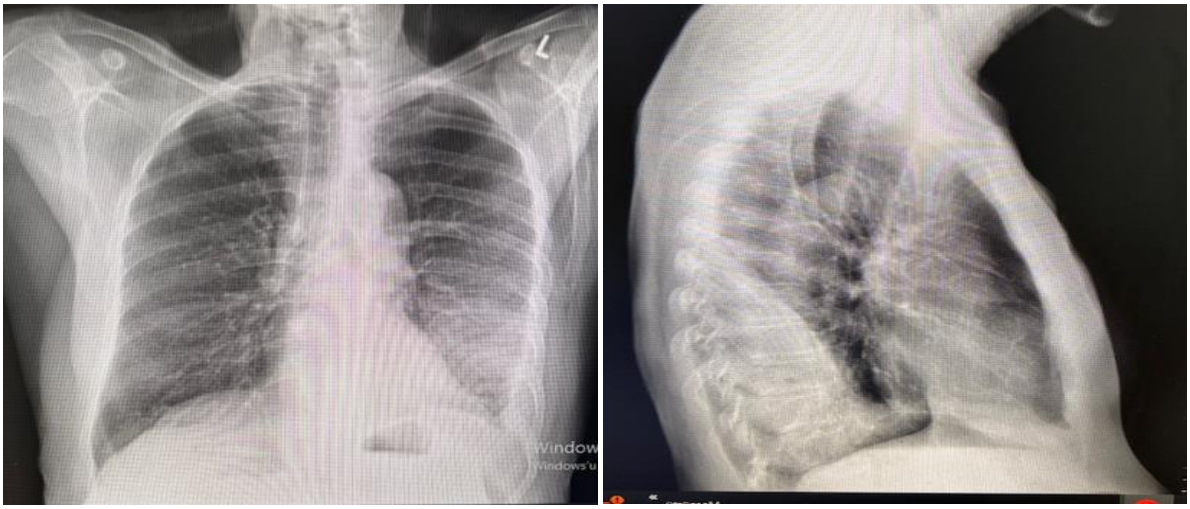

Figure 1: PA and lateral chest x-ray showing left pleural effusion.

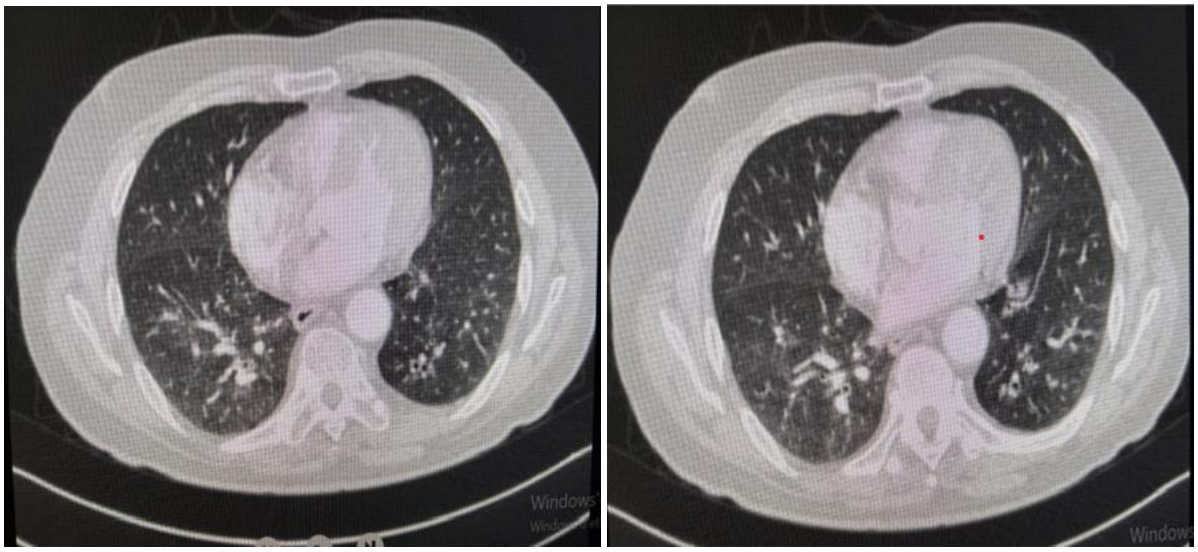

Figure 2: Thorax CT revealing left pleural effusion, segmental and subsegmental bronchiectasis in both lower lobes.

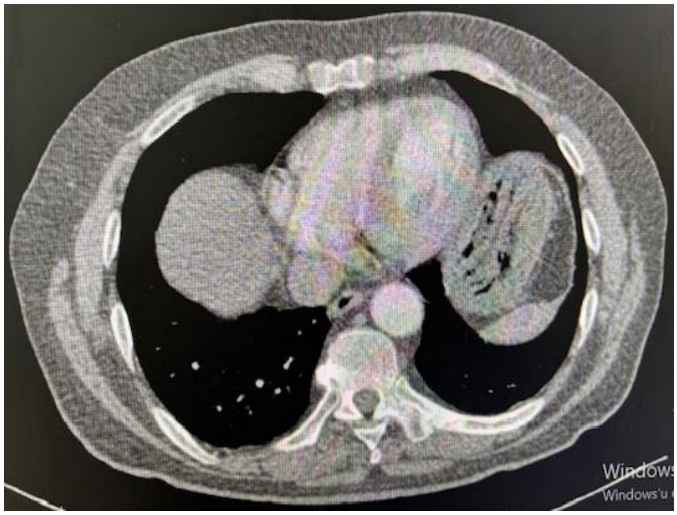

Figure 3: Chest x-ray and thorax CT revealing complete resolution of the pleural effusion.

Discussion

Ulcerative colitis is a chronic inflammatory bowel disease primarily affecting the colonic mucosa. However, it is increasingly recognized as a systemic disease with numerous extraintestinal manifestations involving the skin, joints, liver, eyes, and the lung [1-6]. Pulmonary involvement is one of the less commonly described organ involvements of ulcerative colitis but clinically significant lung manifestations comprising pleural effusion, bronchiectasis, interstitial disease, and airway inflammation may emerge [7-10]. This case highlights two pulmonary complications, pleural effusion and bronchiectasis, in a patient with a known history of ulcerative colitis and total colectomy, underscoring the complex interplay between systemic inflammation and organ-specific pathology.

Pulmonary manifestations of ulcerative are rare but can lead to a significant impact on morbidity [6-8]. An estimated 30-50% of patients with inflammatory bowel disease develop extraintestinal organ involvement during their lifetime often involving the skin, joints, and the eyes [2,3]. Lung disease associated with ulcerative colitis was first described by Kraft [1]. Since then, various manifestations have been reported, ranging from fatal interstitial lung disease to subclinical lung involvement or drug induced pulmonary disorders. Pulmonary involvement in ulcerative colitis is rare, with a reported prevalence ranging from 0.2% to 0.5% [7-10,12]. The underlying mechanisms are not fully understood but are believed to stem from shared immunopathogenic pathways between the gut and the lungs. Both tissues are lined by mucosal surfaces rich in lymphoid tissue and share common embryologic origins, which might explain the propensity for parallel inflammatory processes. Inflammatory cytokines such as tumor necrosis factor-α (TNF-α), interleukin (IL)-1, and IL-6 play key roles in driving systemic inflammation, potentially leading to pulmonary involvement in ulcerative colitis patients [6-9]. Storch et al, described lung involvement associated with inflammatory bowel disease as drug-induced, anatomic, overlap syndromes, autoimmune disease, lung function abnormalities, and other pulmonary manifestations [5]. Radiologic imaging may reveal parenchymal lesions in up to 37–53% of patients [12,13] while only 10% of the affected patients have been reported to have respiratory symptoms in retrospective studies [5-10].

Pleural effusion is an uncommon complication of ulcerative colitis that may arise due to several mechanisms, including immune-mediated inflammation, hypoalbuminemia secondary to malnutrition, or as a complication of drug treatment. In this case, the pleural fluid analysis revealed an exudative effusion with lymphocytic predominance, aligning with an inflammatory etiology rather than a transudative process from hypoalbuminemia. Pleural fluid examination did not reveal any infectious agent, malignant, or inflammatory disease, drug or an environmental factor that could cause the effusion, so it was evaluated as a sequela of ulcerative colitis. Bronchiectasis is another rare but recognized pulmonary manifestation, with prevalence rates higher in patients with ulcerative colitis compared to the general population. The pathogenesis is multifactorial and includes chronic systemic inflammation, immune dysregulation, and recurrent airway infections [5-11]. Inflammation within the bronchial walls, perpetuated by the release of proteases and other inflammatory mediators, led to structural damage and irreversible airway dilation in this patient. Exclusively detailed differential diagnostic assessments performed on our patient did not detect any infectious, granulomatous, autoimmune, or vasculitis etiology that could lead to pleural effusion and bronchiectasis. The final diagnosis was ulcerative colitis associated pleural effusion and bronchiectasis due to the autoimmune inflammatory induction of the primary disease. Complete spontaneous resolution of the pleural effusion without any treatment is evidence for its occurrence due to the autoimmune inflammatory mechanisms of the ulcerative colitis itself. Inflammation within the bronchial walls caused by the release of proteases and other inflammatory mediators may have led to bronchiectasis due to structural damage with irreversible airway dilation. In the absence of infection, primary autoimmune, vasculitis, other systemic disorders, or any other etiologic factors, ulcerative colitis was determined to be the exclusive mechanism for the development of bronchiectasis in this patient.

The pulmonary manifestations of inflammatory bowel disease in some patients may only become clinically significant after surgery as Kelly et al has suggested an association between surgery for inflammatory bowel disease and development of lung disease, particularly bronchiectasis, in susceptible patients and the withdrawal of medical treatment [14]. However, similarities between pathogenesis, sharing significant environmental risk factors, and genetic susceptibility suggest that there is a complex interplay between the inflammatory bowel disease and the airway involvement. Recent evidence of inflammatory bowel disease among patients with airway diseases and the higher than estimated prevalence of subclinical airway injuries among these patients support the hypothesis of a two-way association [15,16]. This patient clearly demonstrates all the autoimmune factors that lead to lung involvement associated with inflammatory bowel disease.

The pathologic distinctive feature of lung involvement for inflammatory bowel disease is cytokine release while genetic susceptibility and environmental factors may play a contributory role. Cytokine release in this patient is probably the exclusive mechanism for lung involvement in this case as the high serum levels justify this assumption. Acute cytokine induced inflammatory involvement like pleural effusion may have resolved spontaneously without any treatment while parenchymal structural changes such as bronchiectasis tend to persist in their irreversible state due to its long-term existence due to an indelible permanent damage of the lung tissue. Contribution of pulmonary infections in ulcerative colitis associated bronchiectasis cannot be disregarded considering that pulmonary infections may develop much more easily in a destructed lung parenchyma but any clinical or laboratory evidence of pulmonary infection was not detected in this patient. It is highly probable that the notably high serum cytokine levels such as TNF-α, interleukin-1, and interleukin-6 is the hallmark mechanism for lung involvement in this patient. Environmental risk factors, lung infection, genetic susceptibility, and other unknown factors such as pulmonary manifestations becoming after surgery or treatment withdrawal may have also contributed or played a role in the development of pulmonary involvement.

Conclusions

Despite a detailed differential diagnostic assessment for other potential or presumptive etiologic disorders, a causative factor that would lead to a pleural effusion and bronchiectasis was not identified in this case. Inflammatory cytokines such as tumor

necrosis factor-α (TNF-α), interleukin (IL)-1, and IL-6 appear to be the hallmark in driving systemic inflammation that led to pulmonary involvement in ulcerative colitis patients. There appears to be an implexuous pathologic mechanism between inflammatory bowel disease and lung involvement. Although inflammatory cytokines play the hallmark role in pulmonary involvement, contributory or the additive influence of environmental risk factors, genetic susceptibility, and other unknown factors such as pulmonary manifestations emerging after surgery or treatment withdrawal cannot be ignored. The probability of inflammatory bowel disease leading to lung involvement should be considered following the detection of pulmonary manifestations, pulmonary function test, or imaging abnormalities while the differential diagnosis is a great challenge for the clinician. Pleural effusion may resolve spontaneously while bronchiectasis may remain irreversible due to the persistence of permanent pathological changes caused by long-term existence of the associated inflammatory mechanisms. Lung disease associated with ulcerative colitis is a significant challenge for clinicians that requires an extensive and a thoroughly detailed differential diagnostic assessment. Early recognition, exclusion of other etiologies, and targeted therapy are critical for optimal outcomes. Further research into the pathogenesis and management of ulcerative colitis associated pulmonary involvement is warranted to guide evidence-based care.

Author contributions

Cuneyt Tetikkurt contemplated and wrote the case report.
Muammer Bilir prepared the laboratory findings of the patient.
Umit Seza Tetikkurt wrote the pathologic mechanisms of the case.

Conflicts of interest

All authors declare that they do not have any conflicts of interest associated with this case report. Authors confirm that there does not exist any supporting or funding agencies for this manuscript.

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