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Case Report

Pulmonary Inflammation in Ulcerative Colitis

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Abstract

Pulmonary involvement in inflammatory bowel disease may emerge in any location of the respiratory system from larynx to pleura. Pulmonary manifestations almost always occur following bowel inflammation. We present a case of ulcerative colitis disease patient with severe pulmonary manifestations exhibiting simultaneous occurence with the exacerbation of the bowel disease.

A 74 year old male was admitted for fever, sputum, malaise, loss of apetite, pleural pain, dyspnea in exertion, and inflammatory discharge from a cutaneous fistula in the upper left abdomen. Physical examination displayed normal findings. Laboratory investigations were within normal limits except for mildly elevated ESR and CRP levels. Chest x-ray and thorax CT revealed peripheral pulmonary nodules in both lungs, bronchiectasis, and a left sided pleural effusion. Pulmonary function test and arterial blood gas values were normal. Pleural fluid was an exudate with a mild increase in leucocyte count.

Pulmonary complications of IBD include inflammation of small and large airways, pulmonary parenchymal disease, serositis, and pulmonary embolism. Pathogenetic mechanisms leading to pulmonary parenchymal and pleural disease consistent with inflammatory bowel disease is currently unknown. There is a great lack or gap of knowledge on the pathogenesis of inflammatory bowel diseases leading to lung dysfunction or involvement. The aim of this case report is to reveal that pulmonary involvement may emerge simultaneously with the inflammatory exacerbation of bowel disease. Our second aim was to shed light into the pathogenetic profile of the pulmonary complications of colitis ulcerosa. We suggest that inflammatory manifestations of lung involvement occur upon the same type of physiopathologic mechanisms involving the bowel in ulcerative colitis and may emerge simultaneously.

Keywords: *Pulmonary Inflammation; Colitis Ulcerosa; Inflammatory Bowel Disease*

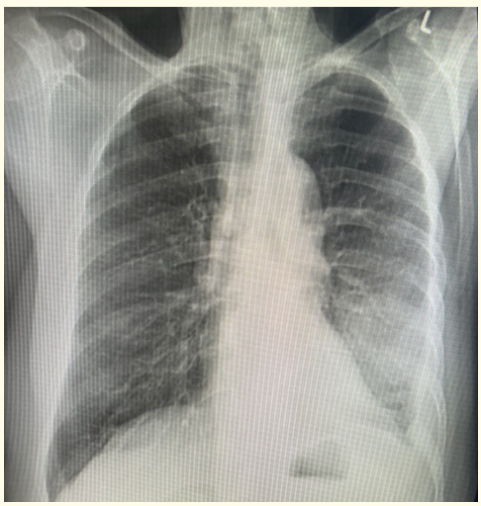
Introduction

A broad spectrum of extraintestinal manifestations, including the lung, may occur during the course of the two major inflammatory bowel diseases [1,2]. Involvement of the respiratory tract, although relatively rare, has been increasingly recognized in patients with inflammatory bowel disease with a propensity for presenting after colectomy that may present from a few hours or days to up to several years or decades after the surgical resection [3-5]. Bronchiectasis in these patients may lead to explicit airway inflammation with excessive

sputum production in the absence of a detectable bacterial infection. Although not fully understood, this inflammatory state appears to be a complex multifactorial process involving an overwhelming T helper type 2-like immune responses leading to mucosal injury in response to gut microbial dysbiosis in genetically predisposed patients [6]. The same pathogenetic mechanisms may appear to be responsible for the marked lung inflammation. As a rare complication of inflammatory bowel disease, serositis involving intrathoracic structures may develop in the form of pleural effusions, pericarditis, pleuropericarditis, and myopericarditis. Pathogenesis of pulmonary parenchymal and pleural inflammation associated with inflammatory bowel disease is currently unknown. To shed light on this issue, we present a patient with marked pulmonary inflammation and pleural effusion emerging simultaneously with ulcerative colitis execarbation. We suggest that the inflammatory pulmonary complications reflect the same profile with primary bowel disease itself.

Case Report

A 74 year old male presented with fever, sputum, malaise, loss of apetite, pleural pain, dyspnea in exertion, and inflammatory discharge from a cutaneous fistula in the left upper abdomen. The patient was a non-smoker. Thirty years earlier the patient had underwent colectomy for ulcerative colitis and had type II diabetes mellitus for ten years. Family history did not reveal any significant disease. The patient described increased sputum production and dyspnea on exertion arising contemporaneously during the occasional exacerbations of ulcerative colitis several times a year. Physical examination displayed normal findings except a fistulae in the left upper abdomen near the umbilicus and decreased breath sounds in the left lower lung. Laboratory showed normal complete blood count, serum biochemistry, liver, and renal function tests. Urine analysis was normal and fecal occult blood test result was negative. ESR was 42 mm/h, CRP was 35.78 mg/dL, procalcitonin was 0.067 µg, and albumin was 2.57 gr/dL revealing mild inflammation. Blood glucose was 134 mg/dL. ECG showed 84/min sinus rhytm with a normal cardiac axis. Tuberculine test was negative. Chest x-ray dsiplayed a moderate left pleural effusion (Figure 1) while CT revealed bronchiectasis in the lower lobes, peripheral pulmonary nodules in both lower lungs, and a left sided pleural effusion (Figure 2). Pulmonary fuction tests were within normal limits. Ultrasound US detected a 4.5 cm anechoic loculated pleural effusion separated by echogenic septa. Thoracentesis of the pleural fluid revealed an exudative effusion composed primarily of neutrophils. Smear and culture of the fluid was negative. Bronchoscopy showed normal findings while bronchial lavage did not grow any microorganisms including bacteria, *M. tuberculosis*, or fungus.



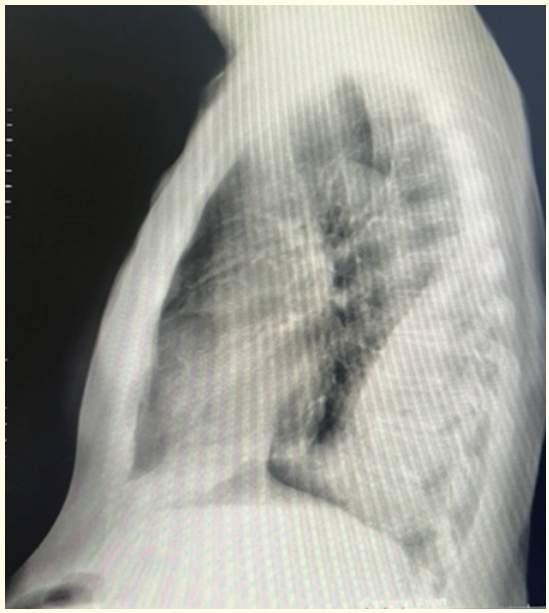
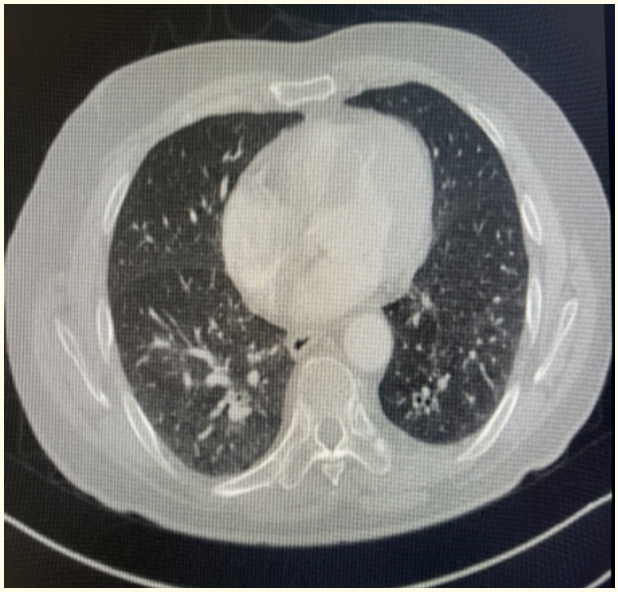


Figure 1: *PA and lateral chest x-ray showing left pleural effusion.*



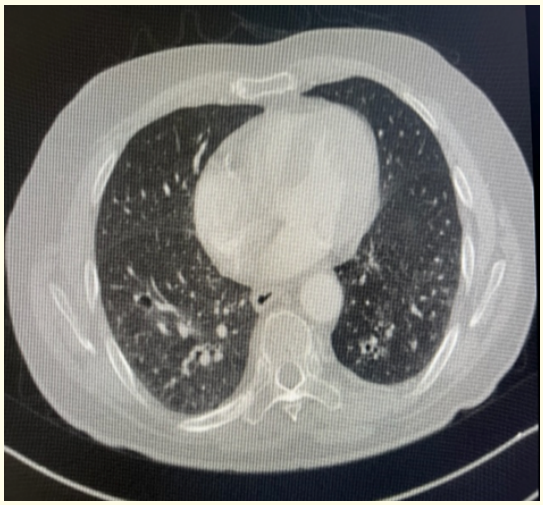


Figure 2: *Chest CT revealing pulmonary nodules, bronchiectasis in the lower lobes, and a left sided pleural effusion.*

Discussion and Conclusion

Pathogenesis of pulmonary parenchymal and pleural complications associated with inflammatory bowel disease is unknown. However, the more common airway inflammatory changes are thought to represent the same type of inflammatory changes that occur in the bowel [1,3]. Involvement of the respiratory tract with unexplained chronic purulent sputum, although relatively rare, has been increasingly recognized in patients with inflammatory bowel disease [7-11]. As a rare complication, serositis involving intrathoracic structures may occur in the form of pleural effusions, pericarditis, and pleuropericarditis [8-10]. The aforementioned inflammatory changes are thought to come out due to the same type of inflammatory pathogenetic mechanism that occur in the bowel [11]. We present a patient to elucidate the inflammatory mechanisms associated with inflammatory bowel disease leading to pulmonary complications.

Our patient was admitted for bronchiectasis and left pleural effusion. Laboratory findings other than mildly elevated ESR and CRP were within normal limits suggesting an inflammatory process. Inflammatory bowel disease develops as a consequence of impaired barrier function of the intestinal mucosa characterized by increased permeability and defective regulation of tight junctions [12]. Current pathogenetic mechanism involves impairment of the mucosal immune regulation of the gastrointestinal system concerning intraluminal bacterial antigens in genetically predisposed persons and respiratory disease in these patients may arise as these products cross-react with common antigens outside the bowel in the human body [13,14]. Lung involvement may also come out due to an aberrant homing of inflammatory cells to the lungs from the primary site of chronic inflammation [15] that might explain the fact that large airway disease is frequently not cured by colectomy [16]. Consequently, the inflammatory process would shift from the gastrointestinal tract to the airways and is asscoiated with the same pathogenetic mechanisms of bowel inflammation [17-19].

Our patient presented with an infectious disease profile along with a simultaneous exacerbation of ulcerative colitis while all markers of infection were negative. The patient had bronchiectasis and an exudative pleural effusion that emerged as a complication of ulcerative colitis. Purulent discharge from the abdominal fistula confirmed the exacerbation of ulcerative colitis while sputum, bronchial lavage,

pleural fluid smear, and culture did not grow any organisms. The patient stated that emergence of purulent sputum accompanied each ulcerative colitis exacerbation that occured several times every year and no source of infection had been identified during the episodes exhibiting purulant sputum. Our case demonstrates that the exacerbation of bowel disease may lead to a noninfectious inflammatory process in the lung simulating a pulmonary infection. Clinicians should be aware that enflaming exacerbation of ulcerative colitis is the fundamental phenomenon for the occurence of lung manifestations such as purulent sputum and pleural effusion. Pulmonary complications including inflammation of small and large airways, pulmonary parenchymal disease, and serositis are usually associated with the underlying inflammatory bowel disease that almost always become manifest with the exacerbation of the primary disorder.

The pathogenesis of inflammatory bowel disease leading to lung inflammation may be associated with the colonic and respiratory epithelia sharing an embryonic origin from the primitive foregut and have columnar epithelia with goblet cells and submucosal mucus glands. The lungs and gastrointestinal tract contain submucosal lymphoid tissue that play crucial roles in host mucosal defenses [20,21]. The similarity between the mucosal immune profile of both systems leads to sensitization and inflammation in the lymphoid tissue as a result of the exposure of the usual antigens taken by inhalation and ingestion to the epithelium [22]. Activated inflammatory cells in the bowel produce several circulating cytokines such as interleukin (IL)-1, IL-2, IL-6, and tumor necrosis factor (TNF)-α that regulate the endothelial cell adhesion molecules, alter leukocyte migration, increase production of damaging reactive oxygen metabolites, and lead to the damage of lung parenchyma [23-26].

Pulmonary complications of inflammatory bowel disease is a diagnostic challenge for the clincian. The patient profile frequently simulates pulmonary infection along with the laboratory and radiologic manifestations. Differentiation of a pulmonary infection and an acute exacerbation of bowel disease in such patients is an absolute necessity for accurate diagnosis and treatment. Occurence of purulent sputum production, pleural effusion, and fever is probably associated with the same pathogenetic mechanism relevant to inflammatory bowel disease exacerbation since there does not exist any underlying etiology such as infection or any other pathology to define the cuurent clinical profile of our patient. Another crucial issue is the difficulty in distinguishing between pulmonary infection and inflammation exacerbation of bowel disease due to the extremely similar laboratory findings that usually point out to the presence of both pathologic phenomenon. In such a diagnostic dilemma, discrimination can only be achieved with an elaborative and a meticulous assessment of the current clinical manifestations. The simultaneous occurrence of acute exacerbation of lung and the inflammatory bowel disease symptoms in our patient and the absence of any etiological factor in the differential diagnosis of pulmonary disease appears as the fundamental evidence that both phenomenon occur due to the same inflammatory physiopathologic mechanism of the bowel itself.

Conflicts of Interest

The author declare no conflicts of interest.

Bibliography

1. Camus P and Colby VT. "The Spectrum of Airway Involvement in Inflammatory Bowel Disease. Chalmers JD, edition. Bronchiectasis". *Clinics in Chest Medicine* 43.1 (2022): 141-155.

2. Olpi JD., *et al*. "Beyond the bowel: extraintestinal manifestations of inflammatory bowel disease". *Radiographics* 37 (2017): 1135-1160.

3. Camus P., *et al*. "The lung in inflammatory bowel disease". *Medicine* 72 (1993): 151-183.

4. Alcazaar BN., *et al*. "Bronchiectasis following colectomy in a patient with ulcerative colitis and factor Vleiden mutation". *Archivos de Bronconeumología* 41 (2005): 230-232.

5. Kelly MG., *et al*. "Inflammatory bowel disease and the lung: is there a link between surgery and bronchiectasis". *The International Journal of Colorectal Disease* 21 (2006): 754-757.

6. Ordás I., *et al*. "Ulcerative colitis". *Lancet* 380.9853 (2012): 1606-1619.

7. Kraft SC., *et al*. "Unexplained bronchopulmonary disease with inflammatory bowel disease". *Archives of Internal Medicine* 136 (1976): 454-459.

8. Black H., *et al*. "Thoracic manifestations of inflammatory bowel disease". *Chest* 131 (2007): 524-532.

9. Storch I., *et al*. "Pulmonary manifestations of inflammatory bowel disease". *Inflammatory Bowel Disease* 9 (2003): 104-115.

10. Rosenbaum AJ., *et al*. "Pleurisy during the course of ulcerative colitis". *Journal of Clinical Gastroenterology* 5 (1983): 517-521.

11. Higenbottam T., *et al*. "Bronchial disease in ulcerative colitis". *Thorax* 35 (1980): 581-585.

12. Turner JR. "Molecular basis of epithelial barrier regulation: from basic mechanisms to clinical application". *The American Journal of Pathology* 169 (2006): 1901-1909.

13. Bamias G., *et al*. "American College of Physicians; American Physiological Society: New concepts in the pathophysiology of inflammatory bowel disease". *Annals of Internal Medicine* 143 (2005): 895-904.

14. Bouma G and Strober W. "The immunological and genetic basis of inflammatory bowel disease". *Nature Reviews Immunology* 3 (2003): 521-533.

15. Birring SS., *et al*. "Idiopathic chronic cough: association with organ specific autoimmune disease and bronchoalveolar lymphocytosis". *Thorax* 58 (2003): 1066-1070.

16. Spira A., *et al*. "Large airway disease associated with inflammatory bowel disease". *Chest* 113 (1998): 1723-1726.

17. Kinnear W and Higenbottam T. "Pulmonary manifestations of inflammatory bowel disease". *Internal Medicine Subspecialties* 4 (1983): 104-111.

18. Mahadeva R., *et al*. "Clinical and radiological characteristics of lung disease in inflammatory bowel disease". *European Respiratory Journal* 15 (2000): 41-48.

19. Ji X., *et al*. "Pulmonary manifestations of inflammatory bowel disease". *World Journal of Gastroenterology* 20.37 (2014): 13501-13511.

20. Van Lierop PP., *et al*. "Role of the innate immune system in the pathogenesis of inflammatory bowel disease". *Journal of Pediatric Gastroenterology and Nutrition* 48 (2009): 142-151.

21. Higenbottam T., *et al*. "Bronchial disease in ulcerative colitis". *Thorax* 35 (1980): 581-585.

22. Tzanakis NE., *et al*. "Pulmonary involvement and allergic disorders in inflammatory bowel disease". *World Journal of Gastroenterology* 16 (2010): 299-305.

23. Williams H., *et al*. "Extraintestinal manifestations of inflammatory bowel disease". *Current Gastroenterology Reports* 10 (2008): 597-605.

24. Gionchetti P., *et al*. "Bronchopulmonary involvement in ulcerative colitis". *Journal of Clinical Gastroenterology* 12 (1990): 647-650.

25. Marvisi M and Fornasari G. "Is the lung a target organ in inflammatory bowel disease?" *Recenti Progressi in Medicina* 92 (2001): 774-777.

26. Sostegni R., *et al*. "Pulmonary manifestations of inflammatory bowel disease". *Digestive and Liver Disease* 39 (2007): 239-241.

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