crease during the conversion period. We also observed that there were no changes in leukocyte and lymphocyte counts. We found no studies that evaluated serum ADA activity during sputum conversion in the literature so there is no data to compare with our results. Also, serum ADA activity in drug-resistant cases is not known. In these cases, serum ADA levels are expected to be high as the sputum positivity continues. The relationship between ADA and lymphocytes may be clarified by new studies in drug-resistant cases. Finally, the serum ADA level is a highly specific parameter in pulmonary tuberculosis patients. Despite acute phase reactants during sputum smear conversion, serum ADA levels do not decrease. For this reason serum ADA may be a good parameter in the follow-up of a chronic disease like tuberculosis.

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References
1. Kuyucu N, Karakurt C, Bilaloglu E, et al. Adenosine deaminase in childhood pulmonary tuberculosis: diagnostic value in serum. J Trop Pediatr 1999;45:245-7.
2. Mishra OP, Yufas S, Ali Z, et al. Adenosine deaminase activity and lysozyme levels in children with tuberculosis. J Trop Pediatr 2000;46:175-9.
3. Canbulat O, Ulusoyduran S, Ozgen G, et al. The comparison of adenosine deaminase activity values with polymerase chain reaction results in patients with tuberculosis. J Clin Lab Anal 1999;13:209-12.
4. Collazos J, Espina P, Mayo J, et al. Sequential evaluation of serum adenosine deaminase in patients treated for tuberculosis. Chest 1998;114:432-5.
5. Seaton A, Seaton D, Leitch AG, Crofton & Douglas’s Respiratory Diseases 4th Ed. Oxford: Blackwell Scientific Publications, 1999:409-10.
6. Guisti G. Adenosine deaminase. In: Bergmeyer UH (ed). Method of enzymatic analysis. New York: Academic Press, 1974:1092-9.

Lung involvement in inflammatory bowel diseases

To the Editor: The rate of extraintestinal involvement in inflammatory bowel diseases (IBD) was reported as 21% to 41%.

Pulmonary involvement patterns include tracheobronchitis, tracheal stenosis, bronchitis, bronchiectasis, interstitial lung disease, necrobiosis nodule, serositis, and pulmonary vasculitis.1,2,4,5,6,7 Our aim was to evaluate lung involvement in IBD. Seventeen IBD patients were included in the study with the approval of a local ethics committee. IBD activity was evaluated by clinical, endoscopic, and histopathological findings. Patients with a previous history of lung disease were excluded. Pulmonary function tests (PFT) were carried out using a Jaeger Master Screen Pneumo device. Patients with normal PFT values were examined for bronchial hyperreactivity with methacholine. High-resolution computed tomography (HRCT) was obtained using a Siemens Emotion 2003 Spiral CT (Munich, Germany) device. Fiberoptic bronchoscopy was applied to 15 of 17 patients who accepted the procedure. Bronchoalveolar lavage (BAL) was performed by standard technique. Mucosal biopsies were also taken from the middle lobe through the lower lobe carina on the right, and the upper lobe through the lingua carina on the left. SPSS software was used for the analysis of the data. Fisher’s exact test was used for comparison of disease activity to other parameters.

Of the 17 patients, 15 had ulcerative colitis and 2 had Crohn’s disease. The mean age of 10 female (58.8%) and 7 male (41.2%) cases was 41.0±12.5 years and the mean duration of disease was 5.6±5.9 years. Six of the cases were regarded as active IBD. Respiratory symptoms were observed in 4 (23%) cases. PFT parameters were normal in all patients except one, who had restriction. Bronchial hyperreactivity was positive in 5 cases irrespective of respiratory symptoms. HRCT revealed pathology (air-trapping, emphysema, peribronchial thickening, bronchectasis, fibrosis, frosted glass, bullae) in 15 cases (88.2%). In BAL, the cell count of 7 cases (46.6%) indicated alveolitis (lymphocytic 40% and neutrophilic 6.6%) was present whereas in the mucosal biopsy of 2 cases (11.8%), submucosal inflammatory cell infiltration was observed. No relationship was found between disease activity and thoracic HRCT findings, PFT, and BAL values (P=0.5).

Despite the amount of research carried out on extraintestinal findings in IBD, the pathogenesis still needs clarification. In such diseases, since there is an impairment in the mucosal immune regulation of gastrointestinal system antigens, digestive enzymes, and bacteria in the luminal content; activation of immune regulatory cells by the systemic circulation occurs.8 Respiratory system pathologies can be classified as airway disease (upper airway obstruction, acute bronchitis, chronic bronchitis, bronchial suppuration, bronchiectasis, bronchiolitis), parenchymal disease (cryptogenic organising pneumonia, pulmonary infiltrates and peripheral
eosinophilia, interstitial lung disease, necrotic nodule), and serositis (pleural effusion, pericarditis, pleuropneumonitis, myocarditis).  

Respiratory pathologies generally arise after diagnosis of IBD. However, they may arise simultaneously or previously.  

Abnormalities of PFT in IBD are not so common. Obstructive and restrictive disorders and bronchial hyperreactivity can be observed.1-3,6,10,11,12 These findings become obvious especially in the activation period of the disease. A restrictive disorder was present in only one (5%) of our patients. However, the majority (64%) of our patients were in a remission period. Bronchial hyperreactivity in the range of 17% to 45% was detected in different studies.1-3,14 We detected hyperreactivity in 29% of our cases. Abnormalities such as bronchiectasis, air-trapping, tree-in-bud appearance, and ground glass opacity may be observed in HRCT even when there is no respiratory symptoms in IBD.1,3,9,16 Songur et al found no relationship between HRCT pathologies and PFT.7 Our study also yielded no correlation between HRCT pathologies and PFT.  

Alveolitis may be lymphocytic, neutrophilic, or eosinophilic according to the presence of bronchiectasis, associated granulomatous disorders, drug usage and smoking.4,13,16 Our study demonstrated lymphocytic and neutrophilic alveolitis in 40% and 6.6%, respectively. Ground glass opacity, neutrophilic alveolitis, and restrictive-type PFT were observed all together in one patient. Tracheobronchial involvement has been defined in tracheal mucosa biopsies of patients with Crohn's disease.6 Tracheobronchitis may become obvious especially in the activation period of the disease.  

In conclusion, there was no relationship between the radiological and hystopathological findings of the respiratory system in IBD patients. In our study, this may be due to the small number of cases. However, even in the absence of respiratory symptoms, cases with IBD should be evaluated for pulmonary involvement because extraintestinal involvement is frequently observed in IBD.

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References  
1. Storch I, Sachar D, Katz S. Pulmonary manifestations of inflammatory bowel disease. Inflamm Bowel Dis 2003; 9(2): 104-115.  
2. Shepeytycky M, Sciberras D, Sharma S. Lung involvement in inflammatory bowel disease: A case report and review of literature. Clinical Pulmonary Medicine 2004; 11(2): 92-100.  
3. Casey MB, Tzeleras HD, Myers JL, Humminglehke GW, Kalka S, Kaira SX, et al. Noninfectious lung pathology in patients with Crohn's disease. Am J Surg Pathol 2003; 27(2): 212-219.  
4. Karagöz F, Özkan M.H., Akcicek E, Gunel O, Alper H, Veral A. Is it possible to detect ulcerative colitis-related respiratory syndrome early? Respirology 2001; 6: 341-346.  
5. Kraft SG, Earle RH, Rossler M, Estery JR. Unexplained bronchopulmonary disease with inflammatory bowel disease. Arch Intern Med 1976; 136: 454-459.  
6. West JE, Adair NE. Tracheobronchitis associated with Crohn's disease. J Bronchol 2004; 11: 194-197.  
7. Songur N, Songur Y, Tuzun M, Dogan I, Tuzun D, Ensari A, et al. Pulmonary function tests and high-resolution CT in the detection of pulmonary involvement in inflammatory bowel disease. J Clin Gastroenterol 2003; 37: 292-296.  
8. Şahan C. Pulmonary involvement in inflammatory bowel disease. Guncel Gastroenteroloji 2003 7(2): 141-145 (Turkish).  
9. Camus P, Piard F, Ashcroft T, Gal AA, Colby TV. The lung in inflammatory bowel disease. Medicine 1993; 72: 151-183.  
10. Mahadeva R, Walsh G, Flower CDR, Shmeerson JM. Clinical and radiological characteristics of lung disease in inflammatory bowel disease. Eur Respir J 2000; 15: 41-48.  
11. Douglas JD, McDonald CF, Leslie MJ, Gillon J, Crompton GK, McHardy DJ. Respiratory impairment in inflammatory bowel disease: does it vary with disease activity. Respir Med 1989; 83: 389-394.  
12. Kuruz K, Vivecka A, Prikazska M, Drugda B, Hronec J, Senkova A, et al. Pulmonary complications in patients with inflammatory bowel disease. Hepatogastroenterology 1999; 46: 1714-1719.  
13. Ceyhan BB, Karakurt S, Cevik H, Sungur M. Bronchial hyperreactivity and allergic status in inflammatory bowel disease. Respiration 2003; 70(1): 60-66.  
14. Louis E, Louis R, Drion V, Bonnet V, Lamproye A, Raddermeer M, et al. Increased frequency of bronchial hyperresponsiveness in patients with inflammatory bowel disease. Allergy 1995; 50: 729-733.  
15. Bonnere P, Wallaert B, Cortot A, Marchandise X, Riou Y, Tonnell AB, et al. Latent pulmonary involvement in Crohn's disease: biological, functional, bronchoalveolar lavage and scintigraphic studies. Gut 1988; 27: 919-925.  
16. Wallaert B, Colombel JF, Tonnell AB, Bonnere P, Cortot A, Paris JC, et al. Evidence of lymphocyte alveolitis in Crohn's disease. Chest 1989; 87: 363-367.  

Influence of atopic history on cord blood IgE  

To the Editor: An increase in cord blood IgE (CB-IgE) is considered an effective immunologic predictor which can be used to screen neonates for the risk of atopic diseases.1,2 Maternal atopic diseases and family atopic history have been proposed as two important risk factors determining CB-IgE in neonates.1,2 To determine the influence of atopic...