

eosinophilia, interstitial lung disease, necrotic nodule), and serositis (pleural effusion, pericarditis, pleuropericarditis, myocarditis).² Respiratory pathologies generally arise after diagnosis of IBD. However, they may arise simultaneously or previously.⁹

Abnormalities of PFT in IBD are not so common. Obstructive and restrictive disorders and bronchial hyperreactivity can be observed.^{2,4,10,11,12} 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.^{13,14} 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.^{4,9,10} 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.^{4,15,16} 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 be an extraintestinal manifesta-

tion of Crohn's disease and it responds very well to inhaled budesonide therapy. Camus et al have shown an intense infiltration of lymphocyte and plasma cells in mucosa biopsies.⁹ Karadag et al have identified lymphocyte infiltration, fibrosis and thickening in alveolar septas.⁴ Submucosal inflammatory cell infiltration was identified in 2 of our cases.

In conclusion, there was no relationship between the radiological and histopathological 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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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.^{1,2,3} To determine the influence of atopic

family history on cord blood IgE, a total of 201 cord blood samples were examined for total IgE concentration from 100 newborns with a family history of atopic disorders and 101 newborns with no history of atopic disorders, assigned as a control or low-risk group, from the Obstetric Ward at Hafez Hospital, Shiraz, Iran, from August to December 2002. All were ethnic Iranian families and informed consent was obtained from parents. This study was approved by our university ethics committee.

The atopic history of parents was collected in the third trimester by means of a standard questionnaire of asthma, allergic rhinitis, atopic dermatitis, and urticaria based on the 'International Study of Asthma and Allergies in Childhood' questionnaire.⁴ A last year medical student interviewed mothers and filled in the questionnaires.

The serum total IgE level of cord blood was determined by enzyme-linked immunosorbent assay (ELISA). A standard calibration curve was constructed. Elevated cord blood IgE values were cut off at the level of 0.5 IU/mL according to the previous reference cut off point.⁵ All data were processed using the SPSS program. There was no significant difference in mean age of mothers, mean gestational age, and mean birth weight between case and control groups. Fifty-one newborns in the case group and 65 newborns in the control group had values of cord blood IgE higher than 0.5 IU/mL ($P=0.158$, chi-square) (Figure 1). We found no significant difference for cord blood IgE levels between the case and control groups.

Our study demonstrated that a family history of atopy is not a

risk factor for elevated CB-IgE. The same result was achieved in a survey conducted in Taiwan, indicating that allergic history of parents does not correlate with elevated CB-IgE levels of neonates.⁶ However, another study conducted in the same region demonstrated that family history of atopy is a risk factor for elevated CB-IgE.⁷ In comparing the results of the present study with others one should consider the different methods for determination of IgE and different cut-off points used in the analysis, as well as differences in the population groups examined. Researchers have frequently argued about the bias from questionnaires of allergy history because the questionnaires were answered by mothers, who probably misinterpret the paternal allergic history.⁸ Certain studies have shown that a maternal history of atopy, but not paternal history correlates with the

elevated CB-IgE levels, suggesting that maternal and/or placental factors can significantly affect prenatal IgE synthesis.^{1,9} In a study conducted in Germany the influence of atopic family history on CB-IgE proved to be strong and the percentage of elevated IgE values increased significantly with the number of atopic family members.³ Kaan et al found that a maternal history of asthma was the most important determinant for high CB-IgE,² and Magnusson reported that infants with a positive immediate family history had a higher incidence of elevated cord IgE.¹ We tried to define clear criteria so as to eliminate questionnaire bias. To avoid contamination at sampling cord blood was collected using direct needle aspiration of the cord vein.¹⁰ Also, acceptable techniques were used in detecting CB-IgE levels and therefore existing variations in results with some other

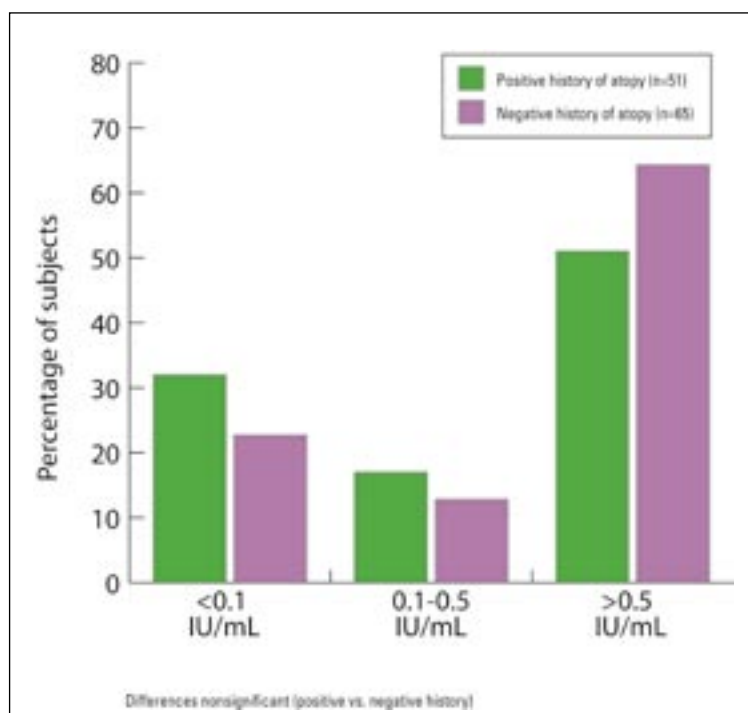


Figure 1. Distribution of cord blood IgE according to family atopic history.

reports should be a matter of ethnic variations. In conclusion, the influence of atopic history on cord blood IgE levels was not confirmed in this study.

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Chylothorax-complicated chronic lymphocytic leukemia

To the Editor: B-chronic lymphocytic leukemia (CLL) is the most common leukemia affecting adults and may infiltrate any organ. Chylothorax is an unfrequent complication of CLL. We describe a new case of CLL complicated by chylothorax and discuss the pathophysiology of this association. A 58-year-old man had been diagnosed five years ago with CLL of B cells. He was treated by chlorambucil (2 mg daily) and then by an association of chlorambucil-cyclophosphamide (Endoxan). Approximately 1 month prior to his hospitalization, the patient had dyspnea on exertion, which had an insidious onset and gradually progressed with a productive cough. On physical examination, the patient was pale, afebrile, with a respiratory rate at 36 breaths/minute, and had signs of pleural effusion over the right lung. An abdominal examination revealed hepatosplenomegaly and no lymphadenopathy was noted. A chest X ray (Figure 1) showed a large amount of effusion on the right side with retractile opacity in the third lower right lobe, and a nodular shadow in the left lower lobe, without mediastinal adenopathy.

The laboratory study found a WBC of 88 100/mm³ (lymphocytes 90%), hemoglobin 8.7 g/dL, platelet count 38 000/mm³, ESR of 85 mm/hr (first hour). Hypogammaglobulinemia was found by protein electrophoresis. Arterial blood gas measurement showed hypoxemia at 71 mm Hg,

transcutaneous saturation of oxygen at 93% with oxygen-therapy (2 L/mn). Thoracentesis revealed a milky pleural effusion with lymphocytes (100%), triglycerides 3.39 g/dL and cholesterol 2 mmol/L. Gram stain and culture were negative. Staining for acid-fast bacilli and culture for TB were negative in the expectoration and in the pleural fluid. Abdominal ultrasound revealed splenomegaly without lymphadenopathy or peritoneal effusion. A chest CT scan (Figure 2) showed a large effusion in the right pleura with condensation in the left lung. No abnormally enlarged lymph nodes were noted. Treatment was by conservative measures: oxygen, repeat thoracentesis and total parenteral nutrition. In evolution, a febrile episode with hypoxemia necessitated antibiotic therapy with a corticosteroid, but the patient died within 2 weeks in respiratory insufficiency.

Chylothorax results from a disruption of the thoracic duct and subsequent accumulation of chyle within the pleural space.¹ A triglyceride level greater than 110 mg/dL constitutes a diagnosis of chylothorax.² The most common etiology of chylothorax is malignancy (more than 50% of cases) and lymphoma accounts for 75% of cases, followed by lung carcinoma.^{2,3} Other lymphocytic tumors are rarely reported, with only 6 other cases of CLL complicated by chylothorax found in the English literature.⁴ The mechanism of how CLL causes chylothorax is not well understood and theories about its pathophysiology are lacking. The paucity of CLL-induced chylothorax probably is attributed to the typical lack of mediastinal lymphadenopathy in CLL.^{4,5} Unlike the cases reported by Zimhony et al,⁶