EDİTÖRE MEKTUP/LETTER TO THE EDITOR
J Turgut Ozal Med Cent 2013:20(3):287-288

Journal Of Turgut Ozal Medical Center
www.jtomc.org

Familial Chronic Lymphocytic Leukemia Patients in Eastern Anatolia Region

Doğu Anadolu Bölgesindeki Ailesel Kronik Lenfositik Lösemili Hastalarımız

İlhami Berber, Mehmet Ali Erkurt, Mustafa Köroğlu, İrfan Kuku, Emin Kaya, Recep Bentli

İnönü Üniversitesi Tıp Fakültesi, Hematoloji Bilim Dalı, Malatya, Türkiye

Dear Editor;

Chronic lymphocytic leukemia (CLL) is qualified by the accumulation of mature-appearing lymphocytes in the blood, marrow, lymph nodes, and spleen. It is the most common form of leukemia found in adults in western countries, accounting for approximately 30% of all leukemias in the United States of America. The etiology of CLL is unclear. However, some agents associated with CLL have been identified. While some environmental factors (such as farm related exposures and occupational chemicals) may increase risk of CLL, results of epidemiological studies have been generally inconsistent and well-defined extrinsic risk factors are unknown but inherited susceptibility to CLL has been recognized for decades. New molecular and genetic markers have been recognized in CLL. Although early studies do not show unique patterns of these markers in cases with a family history versus cases without a family history (1). In this letter, we have presented familial CLL patients.

First family; Y. K and İ.K are two brothers. Y.K, 67 year old male was diagnosed with CLL and was given several treatments; unknown drugs in 2008, 6 cycles of fludarabine, cyclophosphamide (FC) in 2011 and 6 cycles of rituximab, cyclophosphamide, vincristine, prednisolone (R-CVP) in 2012. The patient is still in remission. İ.K, 57 year old male was diagnosed with CLL and patient was given several treatments; unknown drugs in 1994 and in 2000, 6 cycles of FC in 2011. This patient is also in remission.

Second family; E.K and S.Y are siblings. E.K, 79 year old female was diagnosed with CLL in 2005 and after one year, patient was in stage IV. He was treated with several therapies; 6 cycles of CVP in 2006, 6 cycles of FC in 2007, 6 cycles of chlorambucil, prednisolone in 2011. The patient is still in remission. S.Y, 65 year old male was diagnosed with CLL in 2005 and was given chlorambucil, prednisolone. Then patient was treated with several therapies such as CHOP, kladribin and alemtuzumab. He died from sepsis in 2008.

Familial tendency in CLL has been proven by epidemiological and family studies. In epidemiological studies, the risk of developing CLL and other lymphoproliferative disease in relatives of CLL patients was found. In a study of Gunz et al. increased risk of developing CLL was found 2.8-3 fold in CLL patients' 1st degree relatives, 2.3 fold in more far relatives and 2.5 fold in total (2). The roles of major histocompatibility complex genes have not shown in the development of familial CLL (1). In our cases four CLL patients are only two different families and they have first degree familial relatives (First family is brother-brother and second family is sister-brother).

Several studies have been conducted in high-risk CLL families to screen the genome-wide localization that contribute to susceptibility but no gene mutations have been identified yet (3). In another study, it was revealed that 11 of the 13 genes in the candidate region were expressed in immune tissues, supporting their functional relevance in investigations of familial CLL (4). The ability to conduct large scale genomic studies will play an important role in detecting susceptibility genes in familial chronic lymphocytic leukemia. CLL is a heterogeneous disease according to survival. Some patients have a long-term survival and no need for treatment. In some cases, the disease is characterized aggressive and need intensive treatment regimens (5). We were not able to examine molecular and gene mutation screenings of our patients.

In conclusion, we think that, understanding the familial CLL biology may assist to elucidate CLL pathogenesis. Early screening in patients with familial CLL may contribute significantly for early diagnoses and prevention of complications. Molecular research could facilitate the elucidation of mechanisms of predisposition and progression in CLL and ultimately lead to targeted molecularly designed treatment, as has been evidenced in the case of chronic myelogenous leukemia.

KAYNAKLAR

1. Bevan S, Catovsky D, Matutes E, Antunovic P, Auger MJ, Ben-Bassat I and et al. Linkage analysis for major

histocompatibility complexrelated genetic susceptibility in familial chronic lymphocytic leukemia. Blood 2000;96:3982-4.
2. Gunz FW, Gunz JP, Veale AM, Chapman CJ, Houston IB. Familial leukemia: a study of 909 families. Scand J Haematol 1975;15:117-31.
3. Lynn R. Goldin, Susan L. Slager. Familial CLL: genes and environment. Hematology Am Soc Hematol Educ Program 2007:339-45.
4. Ng D, Toure O, Wei MH, Arthur DC, Abbasi F, Fontaine L et al. Identification of a novel chromosome region, 13q21.33-q22.2, for susceptibility genes in familial chronic lymphocytic leukemia. Blood 2007;109:916-25.
5. Cappio-Caligaris F. What we are learning from familial chronic lymphocytic leukemia? Leukemia Research 2002;26:779-80.

Received/Başvuru: 30.11.2012, Accepted/Kabul: 07.01.2013

Correspondence/İletişim

İlhami BERBER
İnönü Üniversitesi Tıp Fakültesi, İç Hastalıkları Anabilim Dalı, Hematoloji Bilim Dalı, MALATYA/TÜRKİYE
E-mail: drilhamiberber@hotmail.com

For citing/Atıf için:

Berber I, Erkurt MA, Koroglu M, Kuku I, Kaya E, Bentli R. Familial chronic lymphocytic leukemia patients in eastern anatolia region. J Turgut Ozal Med Cent 2013;20(3):287-288
DOI: 10.7247/jtomc.20.3.22