HEPATITIS C VIRUS INFECTION ASSOCIATED WITH B-CELL NON-HODGKIN'S LYMPHOMA IN HUNGARIAN PATIENTS

We read with great interest the paper by Cucuianu et al (1999) on hepatitis B and C virus infection in Romanian non-Hodgkin's lymphoma patients. Recently, a large amount of data has suggested the role of some infections as aetiological factors in different lymphoproliferative disorders: Helicobacter pylori in mucosa-associated lymphoid tissues (MALT) lymphoma. Epstein—Barr virus in Burkitt's lymphoma, human T-cell leukaemia/lymphoma virus in adult T-cell leukaemia/lymphoma and human herpesvirus 6 in plasma cell diseases. Hepatitis C virus (HCV) has hepatotropic and lymphotropic

activities, and has a role in the pathogenesis of B-cell non-Hodgkin's lymphoma (Ferri et al, 1997). The different prevalence of hepatitis C infection described in these pathological conditions varies from $4\cdot3\%$ (Ellenrieder et al, 1998) to $38\cdot4\%$ (Mazzaro et al, 1996), which indicates a need for further seroepidemiological studies in this field.

We performed a similar survey in Hungary, a neighbouring country to Romania. A total of 42 consecutive B-cell non-Hodgkin's lymphoma (NHL) patients (24 men and 18 women, mean age 54·14 years, range 22–80 years).

Table I. Hepatitis C virus infection in various lymphomas.

		Number of patients positive/total	
Lymphoma subtype		HCV antibodies by ELISA	HCV-PCR positive
Indolent	Chronic lymhocytic leukaemia (CLL)	2/10	4/6
(low risk)	Follicular lymphoma grade I or II	0/5	2/3
	Extranodal mucosa-associated lymphoid tissues (MALT) B-cell lymphoma	0/1	0/0
	Total	2/16 (12.5%)	6/9 (66.7%)
Aggressive	Diffuse large B-cell lymphoma (DLCL)	4/12	2/5
(intermediate risk)	Mantle cell lymphoma (MCL)	0/5	1/3
	Multiple myeloma (MM)	0/7	1/1
	Primary mediastinal large B-cell lymphoma (PMLBL)	0/1	0/0
	Total	4/25 (16%)	4/9 (44.4%)
Very aggressive	Burkitt's lymphoma	0/1	_

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498 Correspondence

classified as 16 with indolent (nine men, seven women), 25 with aggressive (14 men, 11 women) and one woman with very aggressive Burkitt's lymphoma, according to the modified REAL classification (Hiddemann et al, 1996), were investigated. All cases were tested by enzyme-linked immunosorbent assay (ELISA) for HBsAg and anti-HCV, and, in 18 cases, the polymerase chain reaction (PCR) technique was used to detect HCV RNA. None of the 42 patients examined was HBsAg positive, whereas anti-HCV was found in 6 out of 42 patients (14·3%) and PCR revealed HCV RNA positivity in 10 out of 18 (55·6%) patients. Table I shows the results according to the different subtypes of lymphomas.

Our results regarding the prevalence of HCV infection anti-HCV or HCV RNA in 10 out of 42 patients (23.8%) in B-cell lymphoma did not differ from the 29.5% prevalence of HCV infection in lymphomas observed by Cucuianu et al (1999), but there was a significant difference in HBV infection - 0% in Hungary vs. 30.8% in Romania. We found a similar prevalence of anti-HCV positivity in indolent and aggressive lymphomas (12.5% and 16%), but it was much lower than in Romania (44·4% and 19·5%). Pár et al (1992) described a significant difference in the prevalence of hepatitis virus infections between Hungarian and Romanian patients. In chronic alcoholic liver disease, HBsAg occurred in 16% of Hungarian patients and in 40% of patients from Romania respectively. HBsAg carriership occurs in 0.7% of Hungarian blood donors, the anti-HCV positivity rate is 1.3%; these data also differ from the Romanian healthy population (6.3% and 4.9% respectively).

. The PCR technique is mostly of importance in detecting HCV infection in patients with humoral immundeficiencies. The studies by Cucuianu $et\ al\ (1999)$ and ourselves show that HCV infection might play a role in the aetiopathogenesis of B-cell non-Hodgkin's lymphoma, but there are geographical differences in the prevalence of this causative environmental agent.

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