means of diagnosis in rectal lymphoma. It is also able to monitor the therapeutic response to the drug treatment and, during follow-up, to detect an unsuspected recurrence of the rectal disease.

To conclude we think that EUS should be performed routinely in the study of rectal lymphomas just as it is for the more common gastric lymphomas.

Margherita Gavioli, Alberto Bagni, Carlo Garoia°, Italo Piccagli, Andrea Biscardi, Gianni Natalini*

*Department of Surgery, Sassuolo Hospital, Modena; *Department of Pathology, Modena University School of Medicine; °Department of Medicine, Castelfranco Hospital, Modena, Italy*

Key words

Rectal lymphoma, endorectal ultrasonography.

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Correspondence

Margherita Gavioli, M.D., Divisione di Chirurgia, Ospedale Civile, via Prampolini, 42 41049 Sassuolo, Italy. Phone: international +39-0536-863311 – Fax: international +39-0536-863388.

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Hepatitis C virus infection and a new association with extrahepatic disease: multiple myeloma

Hepatitis C virus (HCV) is associated with various types of tumors. Our study examines the prevalence of hepatitis C infection in myeloma and compares the findings with a control group. Our results show a prevalence of HCV of 32% in the myeloma patients compared with a prevalence of 9% in the control group, with an odds ratio of 4.3, CI 95% 1.8-11.0 (*p*=0.001).

Sir,

Apart from hepatic cancer, hepatitis C virus is associated with thyroid tumors, and diverse processes originating from B-clonal lymphoid proliferation, such as mixed cryoglobulinemia and B-cell non-Hodgkin's lymphomas.[1-3] It is therefore possible that the virus favors the onset of other tumors correlated with immune disorders.[4] With these considerations in mind we evaluated the prevalence of hepatitis C in 41 patients (mean age, 62±9 years) suffering from multiple myeloma (23 men and 18 women) who were hospitalized in our Institute from 1997 to 1998. Patients who had no history of cancer or autoimmune disease but who were hospitalized in the same period as the cases were used as controls. They included 120 subjects (mean age, 59±10 years), 47 men and 73 women, most with the following diagnoses: breast dysplasia (33), fibroma or myoma of the uterus (26), prostate adenoma (22), hernia (8), lipomas (10), prostate hypertrophy (11), fistulas (6), moles (4). The following data were collected at recruitment: date of admission, date of birth, histologically confirmed diagnosis, diagnosis-age (grouped as ≤ 55, >55), marital status. The serologic diagnosis of hepatitis C virus infection was tested with a third generation ELISA test (Ortho Diagnostic System, Raritan, NJ, USA) and samples yielding positive results were re-tested with polymerase chain reaction (PCR, Amplicor qualitative, HCV test, Roche Diagnostic). The relationship between multiple myeloma and HCV infection was assessed by means of odds ratios (OR) and corresponding 95% confidence intervals. Unconditional logistic regression equations included terms for age, sex and marital status.

Our results showed a prevalence of HCV infection of 32% in patients with myeloma compared with a prevalence of 9% in the controls. After controlling for both age and sex the odds ratio was 4.3, CI 95% 1.8-11.0 (*p*=0.001). The prevalence of HCV infection in male multiple myeloma patients was 30% and in female patients 33%. This compares with prevalence of 7% in male controls and 13% in female controls. For the variable age, we found that the prevalence of HCV infection in over 55-year olds was 36% in those with myeloma and 8% in the controls (see Table 1).

Multiple myeloma is a relatively rare cancer (affecting older individuals) and its causes are still unknown;[5] the relationship between the viral agent (HHV-8) and multiple myeloma is controversial, but some studies have shown a positive correlation.[6] Moreover a high prevalence of HCV infection, not associated with mixed cryoglobulinemia, was detected in patients with multiple myeloma.[3]

The mechanism responsible for the oncogenetic role of HCV is not well understood. Despite an active immune response by the host, HCV has the ability to escape this response. It is believed that the quasi-species nature of HCV is one of the major mechanisms allowing the virus to develop chronic infection and co-factors influencing the outcome of the diseases, such as age, gender, environmental and immunologic genetic factors may play important roles.[4,7] HCV is a major health problem throughout southern Italy, with a prevalence of 12.6%;[8] the same area also has a high mortality rate for liver cancer,

Table 1. Odds ratio and corresponding 95% confidence intervals of multiple myeloma by hepatitis C virus (HCV) infection.

	HCV infection			OR	95% CI	p
	Positive n (%)		Negative n	Total		
All						
Controls	11	(9)	109	120	1*	
MM	13	(32)	28	41	4.3°	1.8–11.0	0.001
Total	24	(15)	137	161		
Male						
Controls	6	(13)	41	47	1*	
MM	7	(30)	16	23	2.4	0.7–8.4	0.1
Total	13	(18)	57	70		
Female						
Controls	5	(7)	68	73	1*	
MM	6	(33)	12	18	7.6	2.0–30.6	0.004
Total	11	(12)	80	91		
≤ 55						
Controls	5	(10)	43	48	1*	
MM	1	(12)	7	8	1.2	0.1 – 12.1	0.8
Total	6	(11)	50	56		
> 55						
Controls	6	(8)	66	72	1*	
MM	12	(36)	21	33	5.6	2.0 – 17.2	0.002
Total	18	(17)	87	105		

*MM: multiple myeloma; *reference category; °derived from unconditional mutliple regression equations including terms for sex and age and marital status.*

thyroid cancer and multiple myeloma although for myeloma these data have been less evident.[1,9,10] Our study strengthens the evidence of a correlation between HCV and lymphoproliferative disorders, but the role of HCV needs to be confirmed by both molecular biology and extensive epidemiologic studies.

Maurizio Montella, Anna Crispo,* Filippo Russo,° Domenico Ronga,# Vincenzo Tridente,# Mario Tamburini**

**Department of Epidemiology; °Unit of Hematology; #Transfusion Center, National Cancer Institute, "G. Pascale" Foundation, Naples, Italy*

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Correspondence

Maurizio Montella, M.D., National Cancer Institute, via M. Semmola, 80131 Naples, Italy. Phone: international +39-081-5903395 – Fax: international +39-081-5462900 – E-mail: legamont@tin.it or gderri@tin.it

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Kaposi's sarcoma after allogeneic bone marrow transplantation in a child

Kaposi's sarcoma (KS) has been increasingly diagnosed in patients who receive immunosuppressive treatment for organ transplantation.[1-4] Furthermore, four cases of KS in patients undergoing bone marrow transplantation (BMT) have been reported.[5-8] Herein we report a case of KS after allogeneic BMT in a child in whom an active infection by HHV-8, a Kaposi's sarcoma-associated herpes virus, was demonstrated.

Sir,

A 7-year old black girl with a severe form of sickle cell disease underwent allogeneic BMT from an HLA identical brother. She had received multiple transfusions. Serologic titers were positive for CMV and negative for HIV, herpes virus and EBV. The donor was also negative for HIV. Conditioning included busulfan, total dose 16 mg/kg, and cyclophosphamide, total dose 200 mg/kg. She received cyclosporin A (CsA) 3 mg/kg daily from day –1 for prevention of GVHD. On day +115, with an absolute neutrophil count > 1,000/µL, she developed extensive chronic GVHD with skin, mucosae and liver involvement. She did not respond to methylprednisolone 2 mg/kg daily and CsA 13 mg/kg, and antithymocyte globulin (45 mg/kg total dose) was added to the treatment. The patient showed a mild improvement after another course of the same regimen. On day +330, purple lesions in skin, hypertrophy of oral and tongue