

**Table: Median values of M.F.I of adhesion molecules.**

	CLL	MCL	MZL	p*
CD11a	167.9	257.9	401.6	<0.0001
CD11b	0	21.7	42.7	0.0011
CD11c	46.3	0	143.6	<0.0001
CD18	105.9	150.1	275.2	<0.0001
CD49c	75.5	0	0	0.0002
CD49d	97.9	248	362.1	<0.0001
CD29	117.6	116.6	236	0.0109
CD44	311.4	316.4	285	0.2600
CD54	223.4	238.1	317.2	0.0018
CD62	4.2	10.2	12.1	0.4895

\*Kruskall-Wallis test.

The Dunns post test was applied when the p value was <0.05. The comparison between CLL and MCL showed that CLL presented a higher expression of CD11c and CD49c, and a lower expression of CD11b and CD49d. When we compared the CLL with MZL, the CLL showed a higher expression of CD49c and lower expression of CD11a, CD11b, CD18, CD49d, CD29 and CD54. Finally, the comparison between MCL and MZL showed that the MZL had a higher expression of CD11a, CD11c, CD18, CD29 and CD54. The structure of normal lymphoid follicle in lymph nodes depends on appropriate association between the B-lymphocytes and the dendritic follicular cell through the interaction between CD11a and ICAM-1, as well as CD49d and VCAM-1. The lower expression of CD11a and CD49d on CLL cells could facilitate their detachment from the lymph node to invade the peripheral blood. A higher frequency of splenic involvement has been reported in cases of CLL with strong positivity to CD11c. However, in our series 82% of the MCL patients presented with an enlarged spleen, but showed the lowest expression of CD11c among the groups. Thus, our findings give support to the role of adhesion molecules in the determination of nodal or leukemic presentation in lymphoid malignancies.

**1394****SAFETY AND EFFICACY OF A COMBINATION REGIMEN CONTAINING PENTOSTATIN, CHLORAMBUCIL AND METHYLPREDNISOLONE IN ELDERLY PATIENTS WITH PROGRESSING CHRONIC LYMPHOCYTIC LEUKEMIA**

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**Background-Aim:** Chronic lymphocytic leukemia (CLL) is a neoplastic lymphoproliferative disorder diagnosed most commonly in the elderly. Alkylating agents used to be the traditional first line treatment, however their inferior response rate and the inability to prolong survival have resulted in purine nucleoside analogues (PNAs) being used as first- and second-line therapy for patients with CLL. Management decisions are more difficult in the elderly because of the increase in toxicity of PNAs in this population. Pentostatin has been proven to be effective and less myelotoxic compared to other PNAs. We wished to evaluate the safety and efficacy of an alternative chemotherapeutic regimen containing pentostatin, chlorambucil and methylprednisolone in CLL patients with progressive disease. **Patients and Methods.** Five elderly, previously multiply treated CLL patients with progressive disease (3 male, 2 female, median age 73.5 years) and one patient with progressing Waldenstrom's macroglobulinemia (WM) were enrolled in the study. Pentostatin was given intravenously at a dose of 4 mg/m<sup>2</sup>, days 1 and 15, chlorambucil was given orally at a dose of 10 mg/d, days 1-7 and methylprednisolone orally 32 mg/d, days 1-7. The cycle was repeated every 30 days. All CLL patients had stage C disease (Binet system). **Results.** Four out of five CLL patients responded to treatment. Response was manifested as normalization of the full blood count and significant reduction in lymphadenopathy and/or organomegaly when previously present. Response was noted at the end of the second cycle. One patient died after the first cycle due to refractory disease. The patient with WM did not respond to treatment and developed grade IV neutropenia leading to discontinuation of this treatment. From the four responders one patient developed grade IV mucositis and delayed further courses and one patient had 3 febrile neutropenic episodes requiring admission. All

patients developed at least grade III neutropenia and received GCSF support. **Conclusions.** Combination therapy with pentostatin, alkylators and steroids seems to be active in CLL patients with progressive disease. However due to increased toxicity especially in the elderly, we suggest that pentostatin should be given at a lower dose. Addition of appropriate antibacterial, antifungal and antiviral therapy and GCSF support is advisable in order to reduce infection risk in these patients.

**1395****RISK OF CANCER IN PATIENTS WITH B-CELL CHRONIC LYMPHOCYTIC LEUKEMIA. EXPERIENCE AND REPORT FROM A RETROSPECTIVE STUDY IN HEMATO-ONCOLOGY DEPARTMENT, UNIVERSITY HOSPITAL OLOMOUC**J. Vondrakova,<sup>1</sup> R. Urbanova,<sup>1</sup> T. Papajk,<sup>2</sup> Z. Holusova,<sup>2</sup> E. Faber,<sup>2</sup> L. Raida,<sup>2</sup> K. Indrak<sup>2</sup><sup>1</sup>University Hospital Olomouc, OLOMOUC, Czech Republic; <sup>2</sup>University Hospital, OLOMOUC, Czech Republic

**Backgrounds.** B-cell chronic lymphocytic leukemia (B-CLL) is characterised as a chronic indolent disease with an immunodeficiency. It is the most common leukemia of adult people, especially in the elderly. It is known that immunodeficiency and age can be the risk factors of cancer. Based on these facts we analyzed retrospectively our own data from patients with B-CLL who were diagnosed in our centre from 1994 to 2004. **Patients and Methods.** We analyzed group consisting of 215 patients, male/female 129/86. The median age of patients was 64 (35 - 91), in the clinical stage Binet A 123 (57%) patients, Binet B 55 (26%) patients, Binet C 37 (17%) patients. There were 19 patients who had a malignancy before the diagnosis of B-CLL was determined such as melanoma (4), Grawitz's tumor (3), colorectal cancer (2), basal cell carcinoma (2) and squamous cell carcinoma (1), uterus carcinoma (1), cancer of breast (1), lung (1), stomach (1), prostate (1), parotid gland (1), osteochondroma (1), leiomyosarcoma (1) and urinary bladder papiloma (1). Two patients had two of these tumors as listed above. There were 15 patients who developed second solid tumors after a diagnosis of B-CLL was established and 4 of them did not receive any chemotherapy for B-CLL. These second tumors involved basal cell carcinoma (3), colorectal carcinoma (2), cancer of the thyroid gland (2), lung (2), kidney (2), prostate (1), squamous cell carcinoma (1), uterus carcinoma (1) and bone metastasis (1). The incidence of all solid cancer was in 34 patients (16%), ratio male/female - 20/14 with a median age of 69. Most of these solid tumors were diagnosed in the clinical stage Binet A in 18 patients (53%), Binet B in 8 patients (24%), Binet C in 8 patients (24%). **Conclusion.** The development of second solid cancers in B-CLL diagnosed patients represents a high risk factor and a complication among long term survivors. Longer follow-up is needed to assess proper anamnesis, physical examination (skin lesion included) and differential diagnosis.

**1396****MULTIPLE MYELOMA IN A PATIENT WITH CHRONIC LYMPHOCYTIC LEUKEMIA. EVIDENCE OF A COMMON PATHOLOGICAL CLONE**

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The coexistence of chronic lymphocytic leukemia (CLL) and Multiple Myeloma (MM) in the same patient is very rare. It is uncertain whether the myeloma cell represents a clonal evolution of the CLL cell or a totally different cell population. We present one female, 62 years old patient suffering from CLL. From 1998 to 2005 she has received therapy for the CLL (chlorambucil and fludarabine in combination with cyclophosphamide). On March 2005 she was presented with pancytopenia and the diagnosis of multiple myeloma was established. In the bone marrow aspirate a diffused infiltration from 40% λ- monoclonal myeloma cells and 40% interstitial infiltration from CLL cells was found. The immunophenotype showed the same light chain in both the MM and CLL bone marrow cells examined. The G-banding conventional karyotype and the molecular cytogenetic analysis by M-FISH and M-BAND showed two different clones. One clone with 45 chromosomes and t(13;14)(p11;p11) and an other with the same chromosomal abnormality and additional complex chromosome rearrangements such as deletion of one chromosome 4, t(4;9), t(4;9;15), t(6;9;15), t(8;11), t(8;17), and t(16;21). The patient underwent chemotherapy with thalidomide plus dexamethasone and had a short partial remission of both diseases. Finally, she died on August 2005. **Conclusions.** The fact that the neoplastic cells carried the same light chain and the presence of translocation