



# haematologica

the hematology journal

14<sup>th</sup> Congress of  
the European Hematology Association  
Berlin, Germany, June 4 - 7, 2009  
**ABSTRACT BOOK**

**2009 | s2**

ISSN 0390-6078

Official Organ of the European Hematology Association  
Published by the Ferrata-Storti Foundation, Pavia, Italy  
Volume 94, supplement no. 2, June 2009  
[www.haematologica.org](http://www.haematologica.org)  
[www.ehaweb.org](http://www.ehaweb.org)



study suggests that the SC route of administration of alemtuzumab is not as effective as IV in the treatment of T-PLL; a series of 16 patients receiving 1st line therapy with IV alemtuzumab has shown an OR of 94% with 88% CR. It is therefore advisable that IV treatment should be used. Pentostatin is an effective agent to augment response. Allogeneic HPCT should be used as consolidation therapy when possible.

## 0932

### INCLUSION OF TOTAL BODY CT SCAN IN THE INITIAL WORK-UP OF CLL PATIENTS WITH EARLY-STAGE ON CLINICAL GROUNDS: PRELIMINARY RESULTS OF A PROSPECTIVE, MULTICENTER O-CLL1- GISL STUDY

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**Background.** The clinical staging systems proposed by Rai and Binet represent the backbone for assessing prognosis in patients with Chronic Lymphocytic Leukemia (CLL). However, staging systems are not devoid of some limitations, among the most significant of which is the lack of recognition of early-stage patients who will progress. Unlike the guidelines for assessing the response to therapy for most other types of non-Hodgkin's lymphomas, the widely-used NCI-WG guidelines for patients with CLL do not incorporate use of computed tomography (CT) scans in the algorithm. However, two recent retrospective study challenged this notion, highlighting the importance of prospective validation of CT scans before routine inclusion in CLL work up. **Aims.** In the present study, we investigated whether total body CT scan allowed to individuate among Binet stage A CLL patients, included in the prospective multicenter O-CLL01 GISL study, cases in more advanced stage and whether this subgroup showed a different expression of clinical and biological prognostic markers. **Patients.** Up to date, 275 patients have been enrolled in the trial started in April 2007 and total body CT scan were available in 87 patients. Fifty-two patients (60%) were male and the median age was 61 years (range, 33 to 71 years). All patients are in Binet stage A, while 83 patients were at low risk (0-I stages) and 4 at intermediate risk (II stage) by Rai classification. LDH was elevated in 11.5% of cases and B2-microglobulin in 24%. Twenty-eight patients (33%) were IgVH unmutated, 31 patients (36%) had a high ZAP-70 expression, 17 patients (20%) were CD38 positive (>30%). Fluorescence *in situ* hybridization (FISH) data are available in 61/87 cases; the most frequent abnormality was del(13)(q14) (29 pts 33%), followed by trisomy 12 (5 pts, 6%), del(17p13) (4 pts 5%) and del(11q22.3) (2 pts 2%), 21 cases (24%) were normal. Cytogenetic abnormalities were clustered in 3 risk groups [i.e. low (del(13q14) and normal), intermediate (trisomy 12) and high risk (del(11q22) and del(17p13))] as suggested by others. **Results.** Considering total body CT scan, 22 out of 83 analyzed (25%) patients were converted into Binet stage B. Notably, 64% were male, LDH was elevated in 18% of cases and B2-microglobulin in 18%, 41% were IgVH unmutated, 27% had a high ZAP-70 expression, 27% were CD38 positive, 4,5% showed a high-risk FISH. Both main clinical characteristics and biological prognostic markers failed to correlate with a more advanced stage. In fact, no statistically different distribution of gender, age, LDH and B2-microglobulin, such as IgVH mutational status, CD38 and ZAP-70 expression and cytogenetic abnormalities were observed

between Binet A cases and Binet B. According the Rai classification 14/83 (17%) low risk patients became at intermediate risk with the integration of total body CT scan. Also this subset of patients did not show a statistically different expression of all prognostic markers, but a higher rate of cases with elevated B2-microglobulin ( $p=0.003$ ), than patients at low risk. Finally, total body CT scan allowed to early individuate a second neoplasia in 2 cases (lung cancer 1 pt, renal cell carcinoma 1 pt). **Conclusions.** In line with literature information, our preliminary data indicate that the integration of total body CT scans in the clinical staging allowed to individuate among Binet A CLL cases on clinical grounds 25% of cases with a more advanced stage. Although a more advanced stage did not correlate with both clinical and biological variables reflecting bad prognosis. A longer follow-up will allow to demonstrate whether the inclusion of total body CT scan in the initial work-up of patients with early-stage on clinical grounds provide relevant prognostic information.

## 0933

### CLL WITH BURKITT-TYPE TRANSLOCATION: A SUBGROUP WITH AN AGGRESSIVE DISEASE COURSE

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**Background.** Balanced translocations are uncommon in chronic lymphocytic leukemia (CLL), and their significance remains poorly understood. **Aims.** The aim of the present study was to identify the characteristics of CLL patients with a Burkitt-type translocation. **Methods.** Clinical and cytogenetic files from patients referred to Belgian and French institutions between 1990 and 2009 for cytogenetic characterisation of CLL, and displaying a t(8;14)(q24;q32) or a variant translocation, were reviewed. FISH was performed to confirm involvement of MYC gene and to analyze other prognostically significant aberrations, i.e. those affecting 17p, 11q, 12 and 13q. IgVH mutational status was additionally assessed. **Results.** 16 patients displayed a t(8;14) or variant (representing < 0.5% of CLLs referred during the period of study). These were mainly males (ratio M/F: 14/2) with a median age of 69 years (range 46-84). Six patients presented with Binet stage A, 3 with stage B and 7 with stage C. Lymphadenopathy and splenomegaly were present in 9/16 and 10/15 patients, respectively. Anemia and thrombocytopenia were present at diagnosis in 3 and 4 patients, respectively. In 3/10 patients, a monoclonal paraprotein was observed. Morphology was compatible with "typical" CLL in 2 cases, "atypical" CLL in 9 cases (i.e. presence of occasional prolymphocytes (< 10%) in 4, and CLL/PL (10-55%) in 2 cases) and PLL in 3 cases (i.e. > 55% prolymphocytes). Morphological data could not be obtained for 2 cases. Immunophenotypical data matched a Matutes-Catovsky score of > 3 in all but one cases. CD38 was expressed (i.e. > 30%) in 9/15 patients. The Burkitt-type translocation was the sole aberration in 2 cases. The translocation most frequently involved IGH, but variant cases involving the IGH or IGL locus were observed in 4 and 4 cases, respectively. The Burkitt-type translocation was associated with one other abnormality in 7 cases. Complexity (> 3 changes including the t(8;14) or variant) was observed in 7 cases. Associated recurrent aberrations included del(11q) (n=6), trisomy 12 (n=5) cases and del(6q) (n=2). VH was mutated in 6 and unmutated in 3 cases. V3 and V7 family were used in 6 cases and 1 case, and V3-21 was never used. There were no recurrent VDJ combinations. Follow-up data was available in all patients over a median period of 23.5 months (range 1-168). Median time to treatment (TTT) was 0.5 months (range 0.5-36). 11/16 received therapy; 4 were refractory and none of the remaining achieved complete remission. Disease-related death occurred in 8 patients, whereas 6 are still alive and 2 are lost to follow-up. **Conclusions.** CLL with Burkitt-type translocation represents an extremely rare entity with an aggressive disease course, an advanced stage at presentation, adverse cytogenetic findings (i.e. del(11q), complexity), short TTT and poor response to therapy.