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DIFFERENTIAL THERAPY OF ACUTE LYMPHOBLASTIC LEUKEMIA (ALL) IN ADULTS

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Between 1987 and 1992 44 pts with ALL received differential therapy depending on the most significant prognostic signs. All pts were divided into 3 groups. Group I included pts with pre-pre-B ALL, WBC $< 50x10^9/l$ and Ph'-negative karyotype (low risk); group II (intermediate risk) included pts with Ia-like antigen on blast cells, TALL, WBC $> 50x10^9/l$ and Ph'-; group III (high risk) included pts with B- and pre-B ALL and all pts with Ph'+t(9;22). Pts in group I received Vcr, Pr, L-asparaginase, in group II Vcr, Pr, L-asp. and Adr, in group III ANLL type of therapy - AdoAP. CR was achieved in 92,3% in group I, 77,3% in group II and 33,3% in group III. Our previous results with the use of unificated program for all groups of pts (Vcr, Pr, L-asp. and Adr) were 100%,58% and 0% CR, resp. 3 years survival is 31%.Among the pts with CR 3 years disease-free survival is 35%(unificated program: 21% and 25%, resp.).

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HEPATIC INFILTRATION IN HAIRY CELL LEUKEMIA. EFFECTS OF THE TREATMENT WITH INTERFERON ALFA-2b (IFN) (INTRON A).

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Hepatic infiltration in HCL is frequent, but usually underdiagnosed because functional hepatic tests are not altered, and pre-treatment stadification procedures are not often performed. Though the effect of IFN Alfa-2b in the bone marrow in pts. with HCL is well known, there is little information about its effects on other HC infiltrated tissues. Between January 1980 and December 1992, 18 pts. with HCL were admitted in two Hematology units. The mean age was 48.8 years old and the M/F relation was 5/1. 14/18 pts. were treated with IFN Alfa-2b alone or combined with another treatment. Treatment dose was 3 MIU/day for 90 days, then 3 MIU three times a week until one year was completed. 5/18 pts. died; 2/5 due to disease progression before the IFN administration and 3/5 for sepsis. Pre-treatment hepatic biopsy was performed in 13/18 pts.: 2/13 showed normal hepatic parenquima without infiltration, while 11/13 (85%) showed different degrees of hepatic involvement due to the disease. Most frequent histologic findings were infiltration and sinusoid dilatation together with portal infiltration and fibrosis, all of which were present at different degrees in all pts. with hepatic injure. Seudopeliosis was observed in 7/11 pts. (64%). HBP was performed in 6 pts. after one year of treatment with Alfa-IFN, which proved the disappearance of the HC infiltration. We concluded that the HC hepatic infiltration is frequent and that Alfa-IFN is useful to elliminate hepatic infiltration after 12 months of treatment.

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TWO TREATMENT REGIMES WITH INTERFERON ALPHA 2bR (IFN) IN Ph+ CHRONIC MYELOID LEUKEMIA (CML) PTS

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Twenty-one previously untreated Ph+CML pts were randomized to receive (A):IFN 5MU/d sc 3 days-a week,or (B):IFN same dose 5 days-a-week, after induction therapy with IFN 5 MU/daily.Seventeen pts were evaluated for complete/partial hematologic & cytogenetic response (C,P,HR,CR),as for evolution according to initial risk. Group A: 7 pts,follow-up 14 mos (range 9-24), 2 sustained CCR, 2 transient CCR, 2 PCR, 1 blastic crisis. Group B: 10 pts, f-up 16 mos (range 10-21 mos), 2 sustained CCR,3 transient CCR, 3 PCR, 2 treatment failure. A good correlation between risk factors (especially cytogenetic abnormalities other than Ph')and response to therapy + evolution was observed in both groups. Despite the small # of patients evaluated and the short f-up time, it seems convenient to select different treatment regimens related to initial risk in order to achieve complete and sustained suppression of Ph' clone in Ph+ CML pts.

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MYOFIBROSIS IN CHRONIC MYELOID LEUKEMIA

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This study aimed to evaluate the incidence, degree, morphological aspects and possible clinical significance of myelofibrosis (MF) in 63 patients with chronic myeloid leukaemia. For the bone marrow biopsy preparation, the usual methods of fixation, decalcification, paraffin embedding and sectioning were used. The sections were stained with haematoxilyn-eosine and special stains for reticuline and collagene fibres. The degree of MF was assessed retrospectively and was defined based on a working histological (light microscopy) classification. MF was observed in 66,7 % of the patients ; 47,7 % had mild or moderate MF and 52,3 % a severe one. In 12 cases in which several bone marrow biopsies were performed, the histopathologic evolution could be assessed. MF can display different patterns : "network-like", linear, circular or mixed. The sudied histological aspects suggest the involvement of the cellular factors (especially those released from megakaryocytes) and of the vascular ones in the genesis of MF. The marked MF correlates with a significant splenomegaly, worsening anaemia and a higher percentage of blasts. The degree of MF influences the clinical, evolutive and haematological pecularities of each case, without influencing significantly the incidence of specific complications and the average survival period.

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ALBUMIN DECREASE IS A DELETEROUS PROGNOSTIC SIGN IN CHRONIC LYMPHACTIC LEUKEMIA (B-CLL).

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A study on 46 B-CLL patients(pts)with an extensive multivariate analysis of biological parameters including total WBC,B-CELL count,platelet count,hematocrit,complement fractions, immunoglobulin level and others has shown that Albumin(A)gave the strongest correlation to stage ($p < o,ooo45$).In a complementary study trying to correlate A level to outcome,2o pts were studied consecutively;9 of them died. A levels measured repeatedly over time showed that 7/9 pts had decreased levels ($< 4g/l$).All other pts had normal levels.Overall,in these 9 pts there was no clear decrease of A over the year preceding death,but in most a trend to decrease could be seen.Probably more pts will have to be studied to answer this particular point.A decrease in A level at any stage is a very poor prognostic sign with a significance of $p < o,oo46$.At this point a pathophysiological explanation is still conjectural.

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CYTOGENETIC ABERATIONS IN THERAPY RELATED MYELOID LUEKEMIA (T-ML) IN CHILDREN

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T-ML, and T-ALL developed in 5 and 2 children respectively, out of 543 survivors (>2 yrs) of childhood cancer treated in the Sambur Center for Ped. Onc. between 1969 to 1991. The cytogenetic findings and previous therapy of 3 children with T-ML is presented:

Age(y) Diag.	First MN	Treatment Irrad	Chemo	Latent Period(y)	T-ML	Chromosome
2.5	ALL	CR	Ctx	5.5	CMMOL	-7
1.3	NBL	MIBG	Mel,Ctx VP-16, Cis	5	MDS-AML	-7, inv(3) (q21q26)
1.5	ALL/BMT test-Rl Abd		VP16 VM26	2.5	(donor) MDS-AML	add(11)(p15)

Monosomy 7 and 3q21, 3q26 breakpoints involvement are assoicated with alkylating agents and radiotherapy. Epipodophyllotoxins lead to another type of T-ML, (M4,5), occurring earlier, associated with der(11q23), occasionaly with 11p15. In the presented case 11p15 occured as the sole chromosomal finding in T-ML that developed in BMT donor cells following VP16, VM26.