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PB1767 OUTCOME OF RELAPSED CHILDHOOD ACUTE LYMPHOBLASTIC LEUKEMIA IN THE SOUTH OF TUNISIA SINGLE CENTER EXPERIENCE

Topic: 02. Acute lymphoblastic leukemia - Clinical

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Background: Relapsed acute lymphoblastic leukemia (ALL) has remained challenging to treat in children, with survival rates lagging well behind those observed at initial diagnosis. Although there have been some improvements in outcomes over the past few decades, only ⊠50% of children with first relapse of ALL survive long term, and outcomes are much worse with second or later relapses.

Aims: In this study we describe clinical characteristics and therapeutics results of first relapse of childhood ALL.

Methods:

Our study included children under 16 years on first relapse of ALL treated according to the EORTC 58951 protocol between January 2000 and December 2020 in the Hematology department of Hedi Chaker Hospital of Sfax. The marrow relapse was defined as the reapparition in the peripheral smears and/or the increase of blasts (>20%) in bone marrow (BM) after a period of complete remission (CR). A combined relapse included another extra medullary location, neurological (N) or gonadal (G). Relapse treatment was based on the COOPRALL 1997 protocol and since 2008, COPRALL 2007 protocol or the VHR group of the 58951 EORTC protocol.

Results:

Among the 262 Childs in CR, 81 was relapsed (31%) after a medium follow-up of 127 months. There are 27 females and 54 males. Median age was 7 years and 6 months. Relapse's frequencies in the groups low risk, average risk 1, average risk 2 and high risk were respectively 4, 39, 18 et 39% of cases. Median of relapse timing was 12 months (2 to 83). Cumulative incidence of relapse at 1, 2 et 3 years after complete remission (CR) were respectively 51, 67 et 84%. Three relapses were occurred after 5 years of CR. Timing of relapse was not correlated with treatment group (p=0.8). The relapse was only medullary in 72% of cases, only neurological in 5% of cases and combined in 19% of cases (M+N= 6%, M+G= 9% et M+N+G= 4%). Second course was delivered to 57% of these patients. Protocol followed was the COOPRALL 1997 in 12 cases (26%), COOPRALL2007 in 28 cases (61%) and EORTC VHR in 6 cases (13%). The second CR (CR2) was get in 55% (44 patients) of treated patients, among them only 6 were allograft (24%). Toxic death before CR had occurred in 19% of treated cases. Among relapsed patients without curative treatment, only one was vivant (neurological relapse). Survivors after first relapse were less than 20%.

Summary/Conclusion:

In our cohort, relapse's rate was more than rates reported in other literature series (31% vs 10 to 15%). Relapse's prognostic factors validated by the literature were the ALL's phenotype, cytogenetic abnormalities (phi +), timing of relapse, combined relapses (medullary is more serious) and intensity of treatment. Those factors were not been find in our cohort because of the little number of cases. CR2 rates was less than literature (55 vs 80%); the revision of followed relapse's protocol is to be discussed. As well, the survival of our patients after ALL relapse was less then reported rates (less than 20% vs 35% at three years). This is due to the abstention of treatment in patient in precocious relapse haven't a sibling familial HLA donor.