28%), so we have a good survival at 5 years in P2 (OS: 75% vs 58%, EFS: 73% vs 55% DFS: 79% vs 68%).
Summary and Conclusions: Childhood ALL in our institut were characterized by a poor presentation at diagnosis: male sex, leucocytosis more than 100 G/L, T phenotype and bad response to prophase are frequent compared to the occident reports. EFS are acceptable in our study but still less than observed in literature (85-90%). We have noted an improvement in the survival rates during the second period P2 this could be explained by improved support treatment

PB1606

WHAT IF YOU FAIL? IMPACT OF UNSUCCESSFUL CYTOGENETIC ANALYSIS ON TREATMENT OUTCOMES IN ACUTE LYMPHOBLASTIC LEUKEMIA (ALL): A TERTIARY CARE SINGLE CENTRE ANALYSIS FROM INDIA

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Background: Cytogenetic findings are important in predicting prognosis; and are an obligatory tool in stratifying patients for treatment assignment in ALL. The large studies in ALL have reported between 10-30% of cytogenetic failure. The impact of cytogenetic failure on treatment outcomes in this group with ALL has not been sufficiently explored.
Aims: To determine the differences in clinical features and treatment outcomes in patients with ALL based on the success of karyotype analyses.
Methods: We undertook a retrospective study to evaluate the impact of cytogenetic failure on treatment outcomes in patients with a diagnosis of ALL (Burkitt-excluded) treated at our tertiary care center from January 2009 till December 2014. Cytogenetic failure was used to define analyses that could not be performed in the laboratory due to no mitoses or non-informative morphology. Risk stratification and treatment was based on the BFM95 protocol for patients' ≤15 years and GMALL in the older patients. Standard of care diagnostic tests and supportive care was administered to all patients. The cytogenetic data was retrieved from the original reports of the laboratory performing the analyses at our centre. Only cytogenetic studies sent to the laboratory were included for analyses. Clinical details were ascertained through patient admission records and discharge cards. The collected data was analyzed using SPSS.

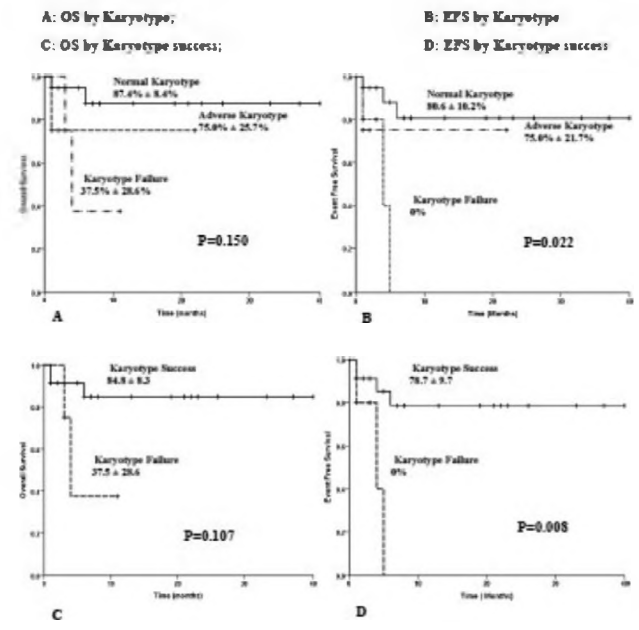

Figure 1.

Table 1. Socio-demographic characters, clinical features and laboratory parameters in newly diagnosed patients (N=54).

Variable	Cytogenetic Success (N=28) n (%)/Median (Range)/Mean ±SD	Cytogenetic failure (N=7) n (%)/Median (Range)/Mean ±SD	P value
Age (years)	16 (2-65)	17(1-30)	0.604
Sex (male)	16 (61.5)	3 (42.9)	0.203
ALL type (B-ALL)	20 (76.9)	3 (42.9)	0.092
Philadelphia (positive)	5 (20.8)	0 (0)	0.252
Steroid response (day8)	17 (89.5)	2 (40.0)	0.044
Post Induction Marrow (remission)	16 (80)	2 (50)	0.232
Infection in induction	7 (26.9)	1(14.3)	0.469
Treatment duration (Induction)	28 (27-41)	29 (26-35)	0.278
Haemoglobin (g/L)	82.0(24.0-137.0)	73.0 (52.0-153.0)	0.706
WBC count (x10 ^{9}/L)	68.2 (0.6-360.0)	33.2 (0.2-324.0)	0.327
Platelet count (x10 ^{9}/L)	43(5.4-344.0)	84 (3.0-307.0)	0.589
Absolute Blast count(x10 ^{9}/L)	24.1(0.12-229.4)	26.3(0-111.6)	0.912

Results: A total of 76 admitted patients were diagnosed with ALL. Of these 54 (71.1%) continued with treatment. Bone marrow was sent for cytogenetic analyses in 33 of these patients. There were no significant differences observed in terms of socio-demographic and baseline laboratory parameters among the two groups based on the success of cytogenetic analyses (table1).The Event Free Survival (EFS) and Overall Survival (OS) were lesser in patients with cytogenetic failure than in those who had a successful karyotype analyses (Figure 1). With a mean follow up of 32 months, the EFS at one year in those with cytogenetic failure was significantly lower than those with a successful karyotype analyses (P=0.008). When compared against the outcomes of patients with adverse karyotype (t (9; 22) or >3 abnormalities), those with cytogenetic failure had a significantly lower EFS (P=0.022).
Summary and Conclusions: Risk stratification in ALL has not defined the optimal strategy in patients with cytogenetic failure. Unsuccessful cytogenetics likely predicts a poor outcome. This needs to be further explored by facilitating the inclusion of these patients as a group in clinical trials.

PB1607

THE FREE RADICAL OXIDATION ROLE IN THE DEVELOPMENT OF ANTHRACYCLINE-INDUCED CARDIOTOXICITY IN PATIENTS WITH ACUTE LEUKEMIA IN THE PRESENCE OF CONCOMITANT ISCHEMIC HEART DISEASE

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Background: Anthracycline antibiotics (AA) are included into the most modern acute leukemia (AL) treatment regimens. The AA assignment in polychemotherapy (PCT) programs contributes to the clinical-hematological remission percentage growth, and the improvement of pts survival. However, the formation of anthracycline-induced cardiotoxic effects can be significant limiting factor of PCT full dose conducting, which certainly leads to reduced effectiveness of anticancer therapy. In this aspect particularly important becomes the assessment of the heart tissue injuries potential risks. The incidence of cardiotoxicity depends on the cumulative dose (CD) of AA. The generally toxic AA CD is 550 mg/m^2 for doxorubicin. An additional risk factor for anthracycline cardiotoxicity is considered to be the concomitant ischemic heart disease (IHD). The imbalance between generation and inactivation mechanisms of aggressive free radicals as a risk factor of heart tissue AA-induced injury in patients with AL with concomitant ischemic heart disease remain insufficiently studied.
Aims: To assess the prooxidant-antioxidant imbalance status in patients with AL in the dynamics of AA treatment taking into account concomitant IHD.
Methods: The study involved 41 patients with acute leukemia (acute lymphoblastic leukemia-13 pts, acute myeloid leukemia-28 pts), aged 16–72 years, 23 (56%) men and 18 (44%) women, which PCT included AA. Patients were divided into two groups according to the presence of concomitant IHD: I (n=24) – without concomitant IHD; II (n=17) – with the concomitant IHD. The general condition assessment of pts of the both groups was performed twice: before specific therapy and after reaching AA cumulative dose from 100 to 200 mg/m^2. The POL processes activity was determined by the malondialdehyde (MDA) level, the antioxidant protection (AOP) – the serum catalase concentration.
Results: Before treatment in patients of group I without concomitant IHD the MDA concentration in serum exceeded the upper limit of normal in 1.2 times, the catalase level – in 1.1 times. In patients of group II with concomitant IHD the MDA level was increased in 1.46 times with the simultaneous tendency to reduce the catalase concentration in serum compared to normal, indicating that the exhaustion of antioxidant protection on the IHD background. Upon reaching the AA CD of 100-200 mg/m^2 MDA concentration in serum was in 1.54 times higher in pts of group II compared with pts of group I (4.81±0.38 mmol/l vs 3.12±0.28 mmol/l; P<0.05). Simultaneously, with the presence of concomitant IHD in pts of group II the catalase level in serum was in 2.1 times lower in comparison with pts of group I (72.5±8.7 mkkat/l vs 33.8±3.2 mkkat/l; P<0.05).
Summary and Conclusions: Therefore, concomitant ischemic heart disease in patients with AL during the treatment AA is an additional risk factor for cardiotoxicity, due to exhaustion antioxidant protection system and deepening imbalance between the formation and inactivation of free radicals.

PB1608

OUTCOME OF ADOLESCENTS AND YOUNG ADULTS WITH ACUTE LYMPHOBLASTIC LEUKEMIA TREATED WITH PEDIATRIC PROTOCOL: 62 CASES

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Background: Several retrospective studies have confirmed that adolescents and young adults (AyA) with acute lymphoblastic leukemia (ALL) treated with pediatric protocols have better outcomes than similarly aged patients treated with adult protocols. We reported results and feasibility of a pediatric-based protocol (EORTC 58951) in adolescents and young adults.