Hematopoietic stem cell transplantation for children with severe aplastic anemia: Single center experience
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Aplastic Anemia (AA) is a life threatening hematological disease that can be cured with hematopoietic stem cell transplantation. We have transplanted 13 children with severe AA from fully matched sibling donors between April 2011 and June 2014. They were followed up for a median duration of 3 years. All patients received ATG and Cytoxan conditioning regimen and PBSC. 11 patients were engrafted. Neutrophil and platelet engraftment occurred after a median duration of 13 and 17 days respectively. Complete donor chimeras (>98%) in all patients; except one who showed partial chimera that followed with progressive decreasing chimera and rejection. Chronic GVHD (ocular) occurred in one patient 9 months post-transplant, CMV activation in four patients and skin infection in another two; All responded well to therapy. Immune reconstitution was studied by flow cytometry quantification of T and B-cell lymphocyte subsets and measurement of immunoglobulins IgM, IgG and IgA at 6, 12 and 18, and 24 months. NK (CD56+/16+) cells recovered in all patients by 6 months. Mean CD4 remained low in most of the patients up to 18 months and normalized in 3 patients at 2 years. CD19 B cell, CD3 T cell and CD8 T cell, as well as IgM and IgG immunoglobulins production were early and complete by one year in almost all patients. Young age, use of ATG and absence of chronic GVHD besides the presence of complete chimeras are responsible for the patients' good quality of life.

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Association of MDR1 gene polymorphism (G2677T) with Imatinib response in Egyptian chronic myeloid leukemia patients
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Background: Despite the excellent efficacy results of IM treatment in CML patients, resistance to IM has emerged as a significant problem. Genetic variations in genes involved in drug transportation might influence the pharmacokinetic and metabolism of IM. The genotype of a patient is increasingly recognized in influencing the response to the treatment.

Aim: To investigate the genotype frequencies of SNPs G2677T in CML patients undergoing IM treatment and to determine whether different genotype pattern of these SNPs have any influence in mediating good response and resistance to IM.

Methods: A total of 96 CML and 30 control samples were analyzed for MDR1 gene (G2677T) polymorphism using PCR-RFLP technique.

Results: Genotype distribution revealed increase in GG, GT (34.4%, 46.9%) and significant decrease in TT (18.8%) genotype frequencies in CML patients compared to controls (p=0.257, 0.326, 0.017 respectively). Patients in accelerated and blastic phases had higher GT genotype frequency compared to patients in early phases (p<0.001). The resistance incidence correlated with G allele. GG and GT genotypes were higher in CML patients showing imatinib resistance compared to sensitive CML patients (P=0.893, 0.002). Meanwhile TT genotype was shown significantly to be higher among imatinib sensitive group with (p<0.001). GT genotype was found to be a significant predictor of IM resistance risk (p=0.002). GG genotype was not proved to be significant indicator of resistance risk (p=0.893). On the other hand, TT may be a protective factor against imatinib resistance in CML patients (P<0.001).

Conclusion: Determination of G2677T MDR1 polymorphisms might be useful in response prediction to therapy with imatinib in patients with CML.

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