Objectives: Immunology of transplant is very complex and yet not well known. This involve polymorphism of HLA, minor histocompatibility antigens, KIR (activatory and inhibitory) allele, cytokine (homeostatic, inflammatory, anti-inflammatory), chemokine's and their receptors. Other non-HLA encoded genes, LA matching can have dualistic effect on transplant outcome: it reduces rejection but conversely, it may promote other HLA-restricted mechanisms of allograft injury. Methods: We have assessed eighteen pairs for allogeneic bone marrow transplantation for hematological malignancies. All related donors and recipients had 100% HLA alleles match (HLA A, B, C, DRB1, DQB1, DPB). All pairs were investigated for their cytokine gene polymorphisms and KIR haplotypes as long term prediction factors for graft versus host disease acute and chronic, complication of vascular origin (veno-occlusive disease, thrombotic microangiopathy), recurrence of the disease and develop of a second malignity. HLA typing was performed by high resolution sequence specific primers (SSP) method using Dynal (KIR Genotyping SSP Kit) high resolution SSP. HLA alleles ambiguities were resolved by Sequencing Based Typing (SBT) Allele SEQR, ABBOTT. Cytokine gene polymorphisms were performed by Dynal (Cytokine Genotyping SSP Kit) PCR SSP (IL1 alpha, IL1 beta, IL1ra, gammaIFN, TGF beta and TNF alpha). KIR genotyping for both donors and recipients were revealed using PCR SSP (Dynal). We try to correlate with leukocytes, platelets recovery presence of schistocytes, appearance of recurrence. Results: Short, our results: - following genes were identified: IL1alpha, IL1beta, IL1ra, gammaIFN, TGF beta1, IL4, IL6, IL10, IL12, gammaIFN, TGF beta and TNF alpha. - KIR genotype B in 31 patients (13 BB, 18 AB) A, in 5 patients. - HLA typing at 4 and 8 digits reveal as alleles secreted but not stable at the cell surface, find at soluble form, allele which contains a mutation outside the coding region (null allele) or low expressed allele at the cell surface. Conclusions: These differences in "perfect" matches, KIR genotype, the presence and fluctuation of cytokine influence the appearance and gravity of GVHD acute or chronic, vascular injury, leukocyte and platelets recovery, recurrence of disease and a late second malignity. Of course, that depends also from disease, conditioning regimen, treatments before and after transplant.