We did not find mutation in coding areas of genes for components of TGFbeta1 signaling pathway but we detected decreased or undetectable expression of these analysed genes. The decreased expression is probably caused by epigenetic changes, so by hypermethylation and deacetylation of promoter regions of these genes. Antiproliferative and apoptotic effect of TGF1 was analysed in AML cell lines (ML1, ML2, CTV1 and Kasumi1). ML2 cells resistance to inhibition of DNA synthesis by TGFβ1 is not caused by mutations of genes for components of TGFβ1 signaling pathway. We found that increased SnoN (Ski-like novel gene) expression on the level of corresponding mRNA and protein is probably accountable for this resistance. Kasumi1 and M2 cells were sensitive to induction of apoptosis caused by TGFβ1 treatment but in less extent than by proteasome inhibitor bortezomib. The difference of AML cells of different lines answers shows a great heterogeneity AML in AML patients. Prognostic factors analysis in AML with normal karyotype confirmed that CEBPA (CCAAT/enhancer binding protein alpha) mutations predict favourable prognosis but the elevated EVI1 (“Ecotropic Virus Integration Site 1“) and ERG (“ETS-related gene”) expression are connected with unfavourable prognosis. EVI1 is a negative marker for MDS as well. We did not confirm overexpression of MNI (“Meningioma 1“) as a negative prognostic factor.