Fetal hemoglobin induction in azacytidine responders enlightens methylation patterns related to blast clearance in higher-risk MDS and CMML.

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Additional file 3: Supplementary figures_1

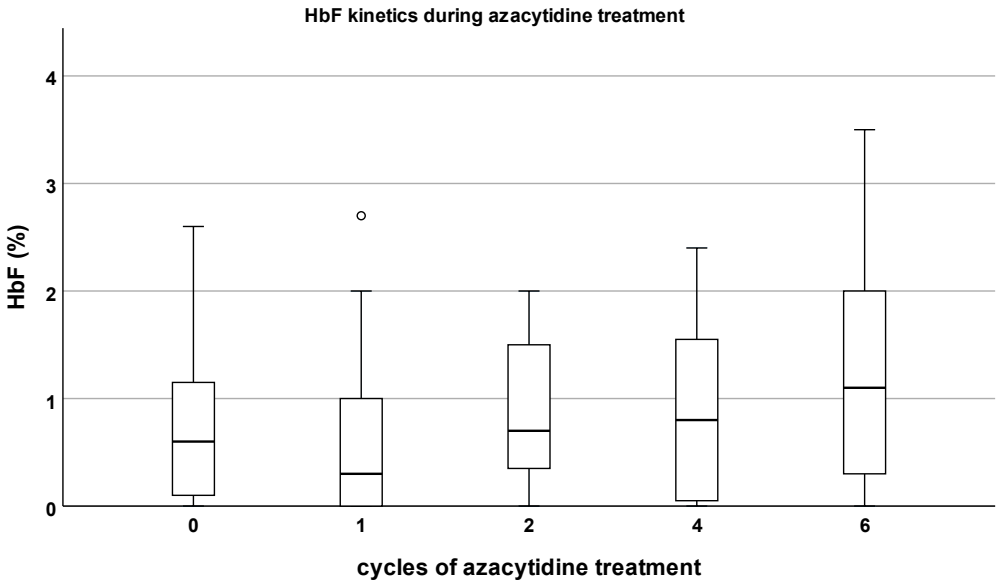


Figure S1. HbF kinetics in the whole 19 patients' cohort over time (after 1, 2,4 and 6 cycles of azacytidine treatment).

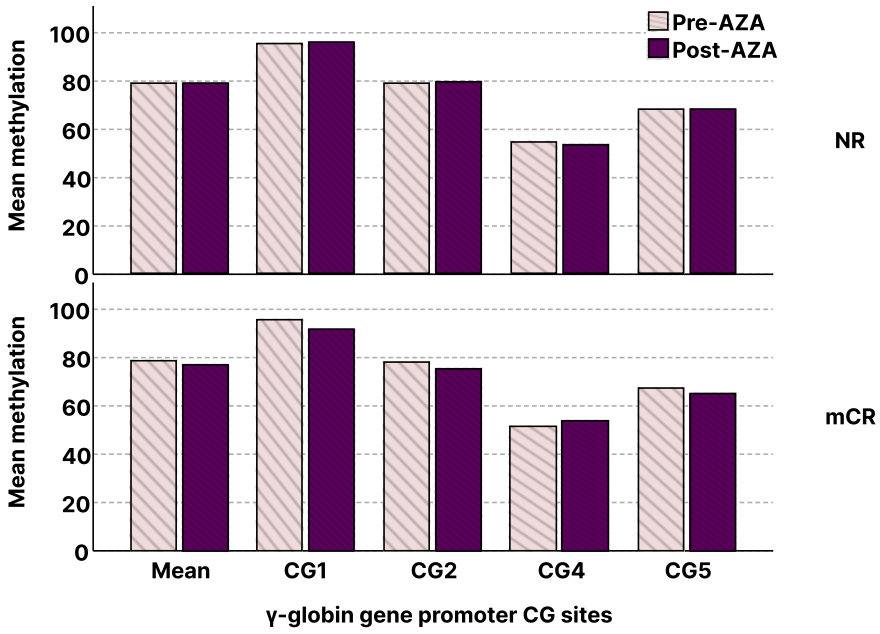


Figure S2. Methylation levels (%) of the γ-globin gene *(HBG2)* promoter at CG sites tested pre- and post-azacytidine (AZA) in mCR responders and NR patients.

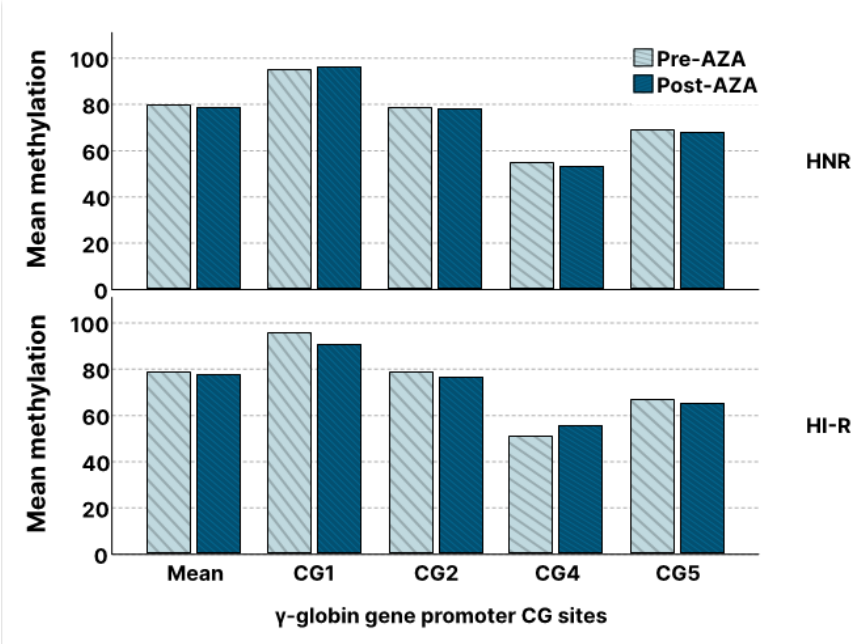


Figure S3.Methylation levels (%) of the γ-globin gene *(HBG2)* promoter at CG sites tested pre- and post-azacytidine (AZA) in HI-R responders and HNR patients.

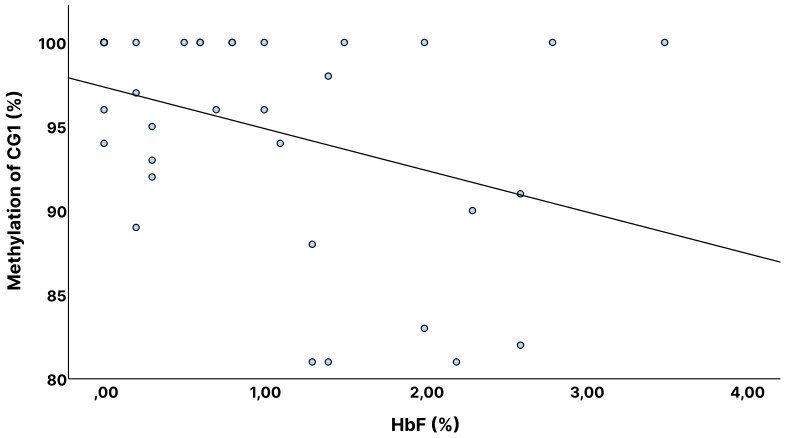


Figure S4. Methylation levels of CG1 of *HBG2* are related to HbF levels (all simultaneous methylation and HbF measurements included, both pre- and post-AZA, p_1=0.042, r_s=-0.341).

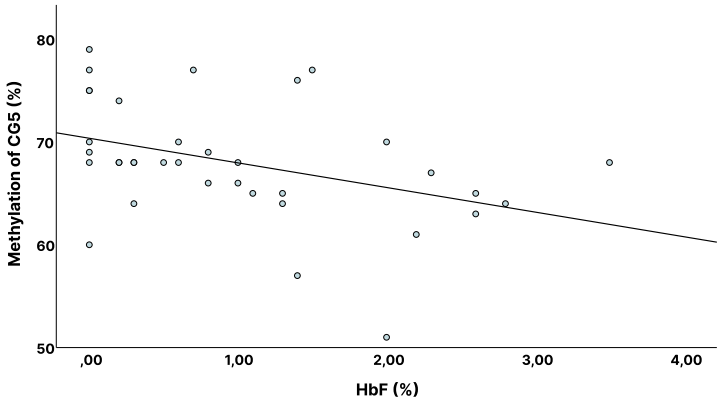
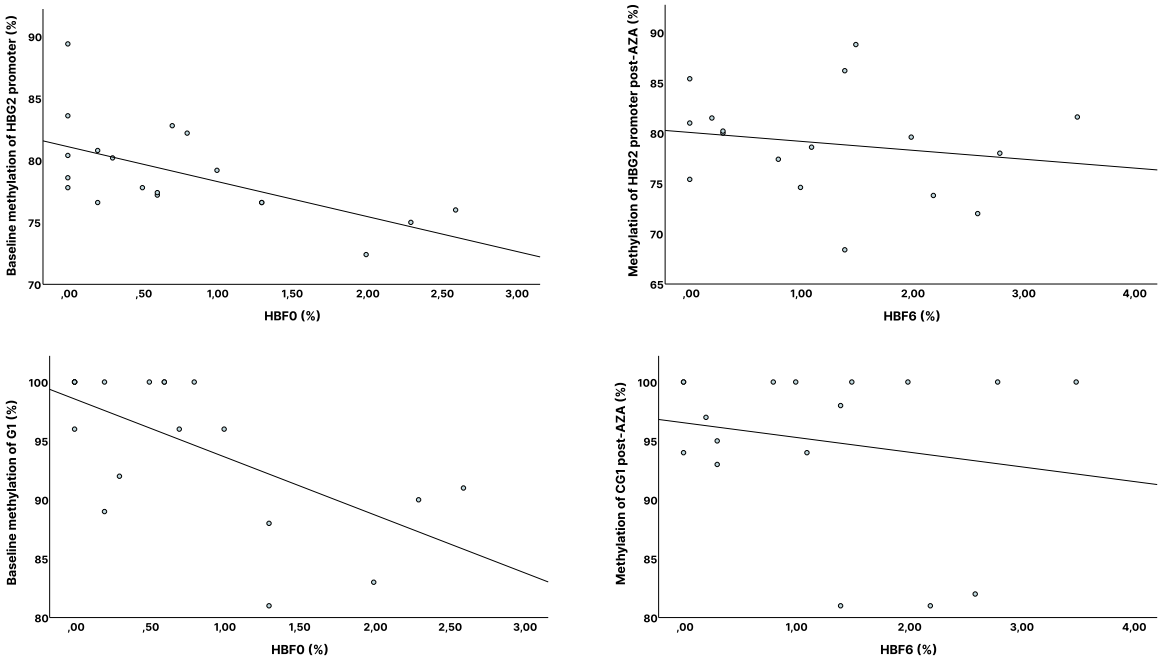


Figure S5. Methylation levels of CG5 of *HBG2* are related to HbF levels (all simultaneous methylation and HbF measurements included, both pre- and post-AZA, p_5=0.005, r_s=-0.461).



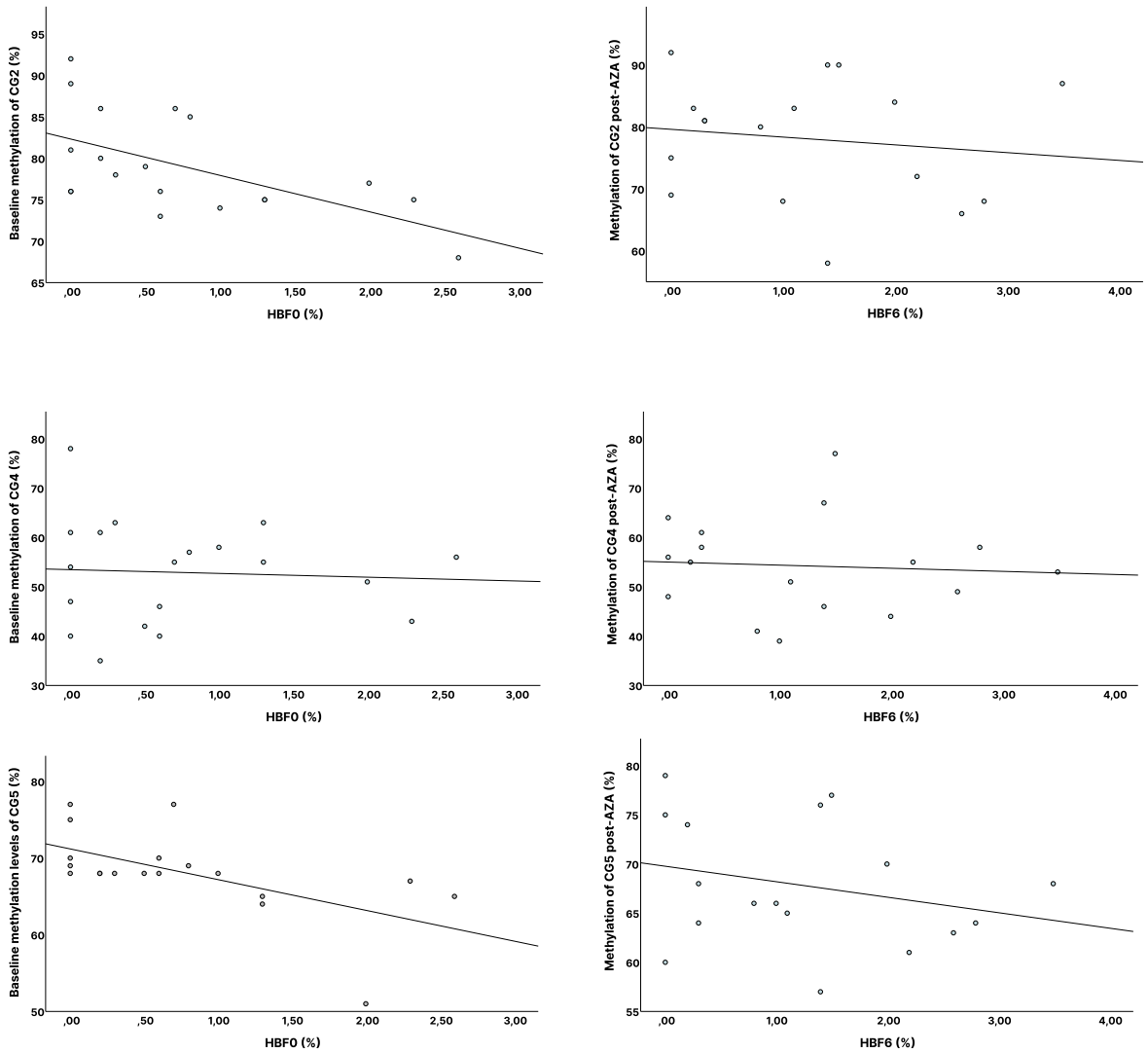


Figure S6. Scatter plots of CG sites of *HBG2* showing the relation between methylation and HbF levels pre- (baseline) and post-AZA (HbF0 and HbF6, respectively).

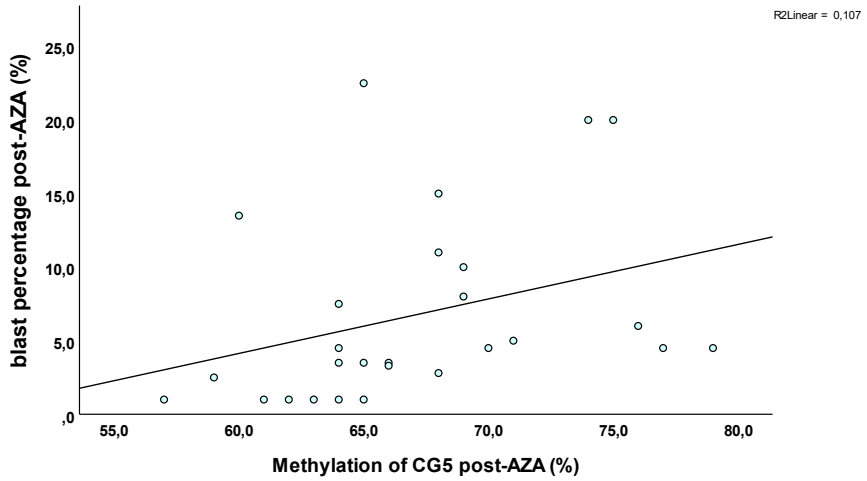


Figure S7. Lower methylation of CG5 of *HBG2* promoter post-AZA (after 5-7 cycles) is related to lower blast percentage post-treatment.