LETTER TO THE EDITOR

Clinical and molecular response of acute myeloid leukemia harboring non-canonical *FLT3* N676K driver mutations to contemporary FLT3 inhibitors

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Supplementary Materials for:

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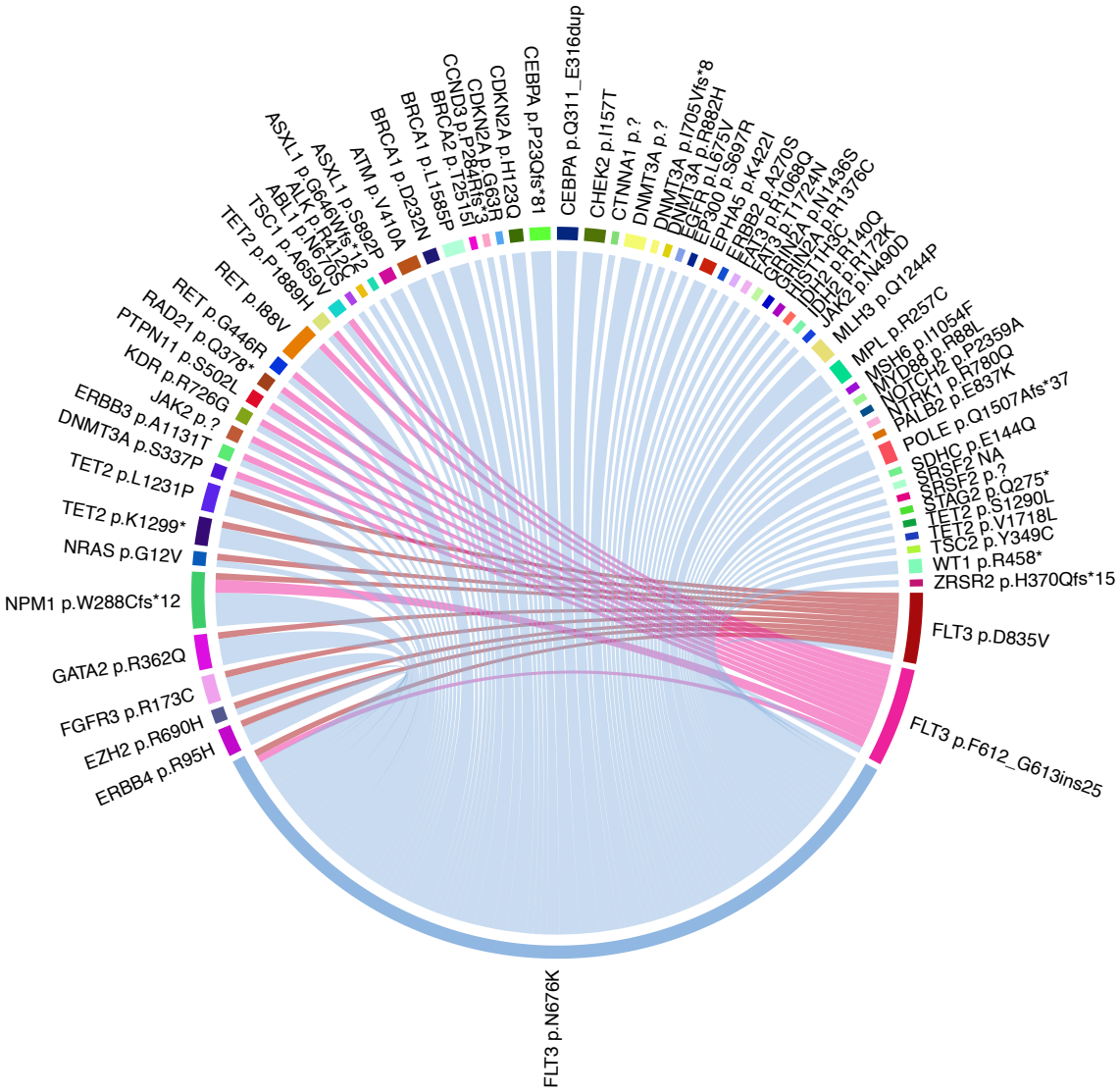

Figure S1. Network of comutations associated with FLT3 variants in the cohort. Circos plot of comutational networks of *FLT3* N676K-mutated AML patients in the analysis. Chord thickness reflects the number of co-occurrences between two genes.

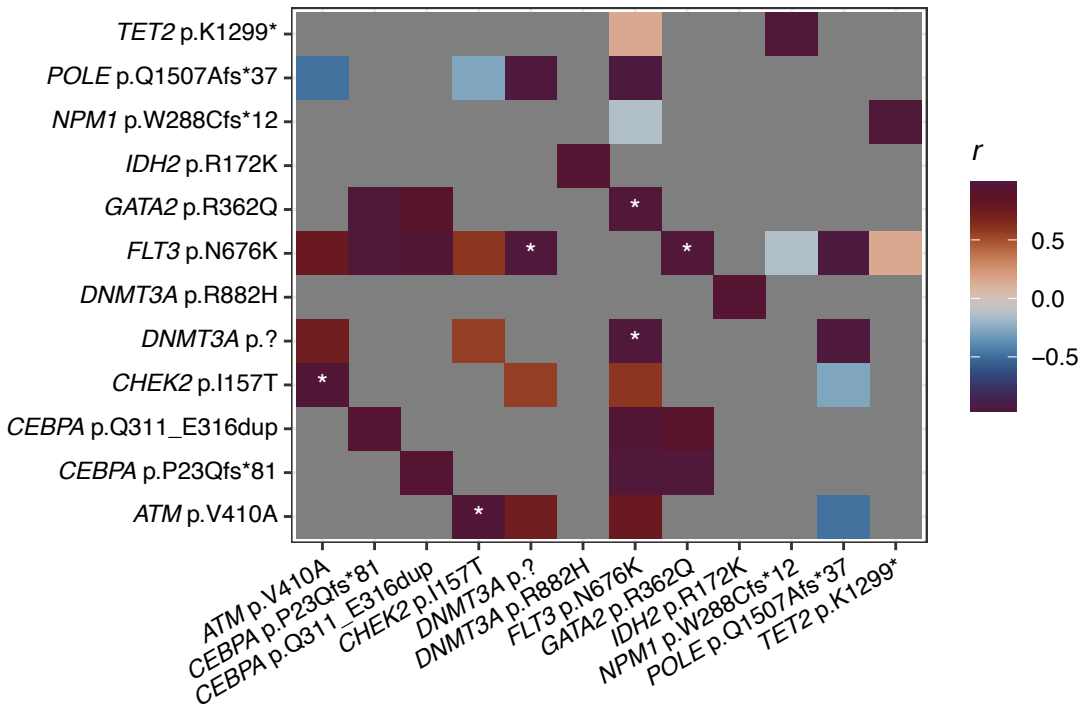

Figure S2. Comutational correlation analysis of longitudinal NGS data.
Pearson's correlation coefficient was across variant allele frequencies (VAFs) for all observed instances of comutations that co-occur at least twice in the dataset. Values approaching 1.0 on the heatmap indicate a strong propensity for mutational co-occurance while more negative values denote mutual exclusivity between two mutational events. White asterisks indicate $p < 0.05$.

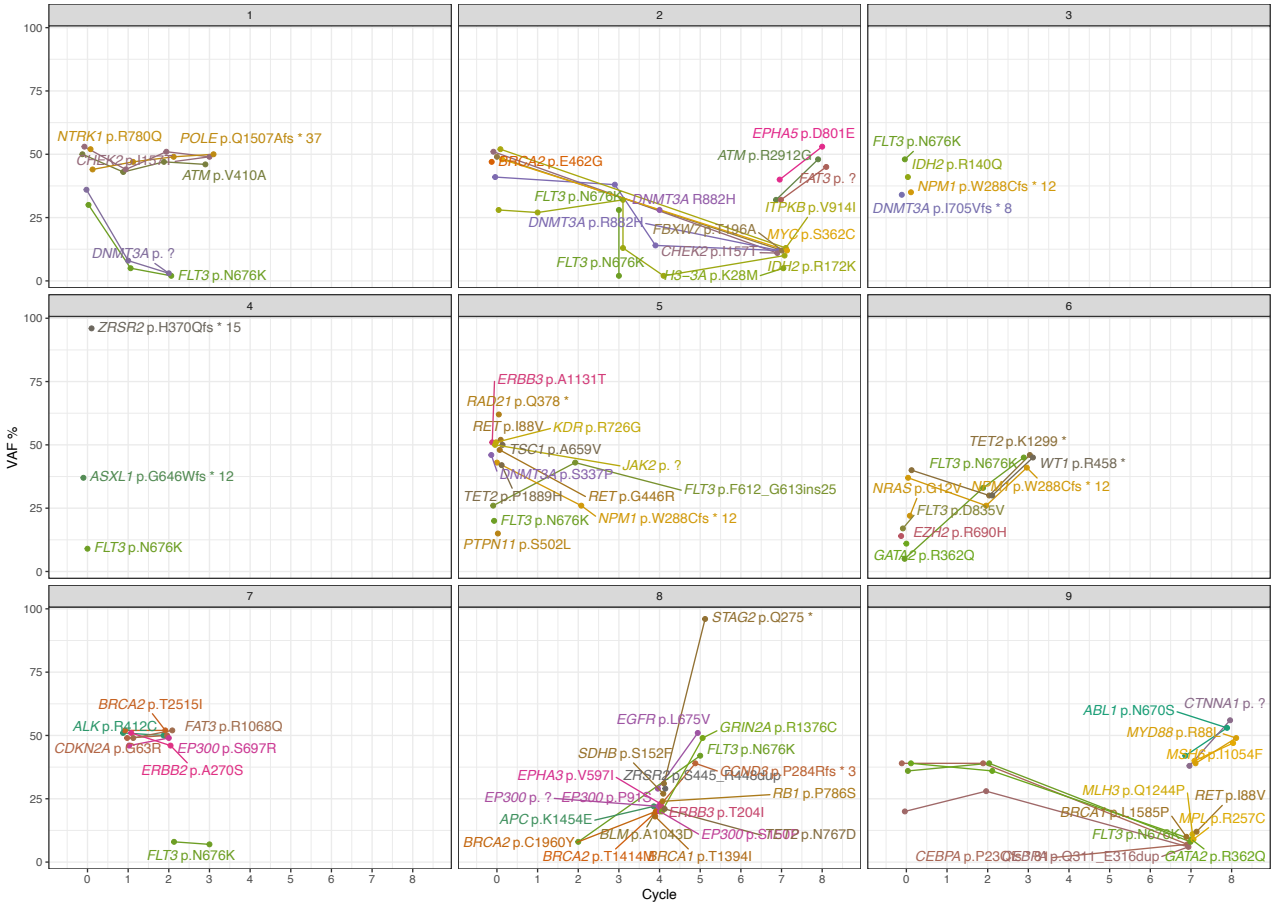

Figure S3. Intra-patient mutation profiles detected on longitudinal NGS.
Longitudinal NGS demonstrates VAF kinetics over time. Each cycle indicated on the x-axis represents a repeat NGS assessment over the clinical course.