Materials and Methods We present data from the ENESTnd study. In ENESTnd, a phase 3, multicentre, open-label, randomised study, patients treated with nilotinib demonstrated higher and faster rates of major molecular response (MMR), more profound molecular response (MR), and complete cytogenetic responses (CCyR) compared with imatinib by 12 and 24 months. 282 adult patients were randomly assigned to receive nilotinib 500 mg twice daily, 281 to receive nilotinib 400 mg twice daily and 283 to receive imatinib. Patients were eligible if they had been diagnosed with chronic phase, Philadelphia chromosome-positive CML within the previous 6 months.

Results By 24 months after the start of treatment, significantly more patients had a MMR with nilotinib than with imatinib (201 with nilotinib 500 mg twice daily, 187 with nilotinib 400 mg twice daily and 124 with imatinib; \( p < 0.0001 \) for both comparisons). Significantly more patients in the nilotinib groups achieved a complete molecular response at any time than did those in the imatinib group (74 with nilotinib 500 mg twice daily, 59 with nilotinib 400 mg twice daily and 29 with imatinib; \( p < 0.0001 \) for nilotinib 500 mg twice daily vs. imatinib, \( p = 0.0004 \) for nilotinib 400 mg twice daily vs. imatinib).

Conclusions Nilotinib continues to demonstrate superiority vs. imatinib with faster and more profound molecular responses. These results support nilotinib as a first-line treatment option for patients with newly diagnosed Philadelphia chromosome-positive and chronic myeloid leukaemia.

No conflict of interest.

Results The study included a total of 40 patients. The mean age of the patients was 38.66 ± 13.98 years and 61.1% were female. The distribution for genotypes was 81.6% AA, 15.8% GA and 2.6% GG. Significant correlation wasn’t found between genotypes or alleles of this polymorphism and clinical response to infliximab. Instead, statistically significant differences were shown for approximately 6 months of treatment when comparing patients with genotypes GG and GA/AA and a positive response (\( p = 0.047 \)) when considering the biological response. Similarly patients with a G allele had a more frequent negative response than those with the A allele (\( p = 0.043 \)). On the other hand, significant correlation was found between patients carrying the A allele and the positive response, at 3, 6 and 12 months based on biological response distribution.

Conclusions The results of our study show an association of this polymorphism with response to infliximab. Worst response rates are observed in patients carrying allele G diagnosed with CD. We need more studies on this polymorphism and with a larger sample size to confirm these findings.

No conflict of interest.