632. CML Therapy. Predictors of TKI discontinuation and TFR outcomes. 
Saturday 7 December, 7-9,30 h
OCCD Valencia D (W415D) Level 4

Moderator: Franck E Nicolini, MD,PhD
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25 Discontinuation of Tyrosine Kinase Inhibitor in Children with Chronic Myeloid Leukemia (JPLSG STKI-14 study)
Haruko Shima, MD, PhD

Background: Tyrosine kinase inhibitor (TKI) has now enabled patients with chronic myeloid leukemia (CML) to live a normal lifespan. However, its long-term side effects such as growth impairment are an issue of concern especially for children. Recent clinical trials in adults have suggested that a subset of CML patients with deep molecular response on TKI therapy may have chance to discontinue TKI without molecular relapse (Saussele et al. Lancet Oncol. 2018). However, the biology of CML in children may differ from adults with more aggressive presentation, and data of TKI discontinuation in CML children are limited (Hijiya et al. Blood. 2019; Bruijn et al. British Journal of Haematol. 2019).

Methods: The Japan Pediatric Leukemia/Lymphoma Study Group (JPLSG) CML committee recruited 22 Japanese patients diagnosed with CML-chronic or accelerated phase at < 20 years of age, treated with TKI for ≥ 3 years, and sustained molecular response (MR4.0) for ≥ 2 preceding years. Patients who relapsed after hematopoietic stem cell transplantation (HSCT) were included if total duration of TKI treatment was ≥ 3 years after rejection and relapse, and also met the criteria mentioned above. We prospectively analyzed treatment-free remission rate (TFR) at 12 months. Molecular relapse was defined as at least one loss of major molecular response (MMR), and TKI treatment was restarted as prescribed before discontinuation.
Results: All the patients were diagnosed with CML-chronic phase. Median age at diagnosis of CML was 9 years (range, 1 to 14 years), and median age at discontinuation of TKI was 16 years (range, 5 to 26 years). Initial TKI was imatinib in 21 patients, and imatinib was switched to second generation (2G)-TKI in 2 patients because of intolerance or poor response. One patient was treated only with 2G-TKI. Median treatment duration of TKI before discontinuation was 100 months (range, 42 to 178 months), and median duration of MR4.0 was 53.5 months (range, 25 to 148 months). TFR at 12 months was 50.0% (90% CI=31.7-65.8%). Eleven patients experienced molecular relapse and restarted TKI at median of 102 days (range, 67 to 166 days) after discontinuation of TKI (Figure. 1). No progression was observed during study, and all 11 patients reaehieved MR4.0 at median of 64 days (range, 25 to 196 days) after restart of TKI. All the patients who lost MR4.0 within 3 months after stopping TKI failed to maintain MMR thereafter and restarted TKI. On the other hand, a single patient who lost MR4.0 after 32 months of stopping TKI reached MR4.0 without TKI. Plasma trough concentration level of imatinib was determined just before stopping imatinib in 18 patients. In univariate analysis, plasma concentration level of imatinib was associated with TFR after stopping TKI ($p=0.005$). Higher plasma concentration of imatinib was associated with higher risk of relapse. In contrast, treatment duration of TKI, duration of MR4.0, Sokal, Hasford, or EUTOS scores had no impact on TFR. Of 3 patients who underwent HSCT, 2 patients succeeded in maintenance of MR4.0 without TKI. In terms of adverse effects, grade 1 musculoskeletal or joint pain observed in 3 patients, and grade 1 to 3 elevation of creatine phosphokinase observed in 6 patients regressed after discontinuation of TKI. No withdrawal syndrome as reported in adults was observed in this study. In 3 males who discontinued TKI before puberty (5, 9, and 13 years of age with testicular size < 3mL), slight recovery in growth associated with increase in both serum bone type alkaline phosphatase and urine N-Telopeptides of Type I collagen was observed soon after discontinuation of TKI. However, these 3 patients relapsed and restarted TKI within 4 months after discontinuation of TKI.

Conclusion: These data indicate that as in adults, TKI may be safely discontinued also in children younger than 15 years of age at diagnosis, treated with TKI for ≥ 3 years, and sustained MR4.0 for ≥ 2 years, including patients who relapsed after HSCT. We suggest that plasma trough concentration level of imatinib may predict TFR for children with CML and sustained MR4.0 with imatinib. Further study of TKI discontinuation in children with CML is expected.
26 Introducing a Predictive Score for Successful Treatment Free Remission in Chronic Myeloid Leukemia (CML)

Simone Claudiani, MD

**Background:** Treatment free remission (TFR) is now a realistic goal of treatment for CML. Approximately 50% of patients (pts) who discontinue tyrosine kinase inhibitors (TKI) after achieving deep molecular responses (DMR) are able to remain off treatment without losing major molecular response (MR3). Data from the largest available TKI stopping trial, EURO-SKI, showed that the most important variable associated with prolonged TFR is the duration of DMR. However, to date no clinical tool exists to guide clinicians and patients in predicting the likelihood of success of TFR attempt.

**Methods:** We performed a retrospective analysis of clinical data from 172 pts with CML in whom treatment was discontinued in 6 hospital centres in order to identify
factors associated with TFR. Data analysis started with a training set (TS) derived from pts treated at a single centre which was then validated on a validation set (VS) derived from the 5 other centres. Eligibility criteria included diagnosis of CML in chronic phase, a minimum duration of treatment with TKI of 3 years and discontinuation of TKI after achievement of confirmed ≥MR4. Patients diagnosed in accelerated phase CML and/or who underwent prior allogeneic stem cell transplant were excluded. Kaplan-Meier method was used for univariate analysis, with log-rank test for group comparison. A Cox proportional hazards model was employed with the purpose of choosing the most influential prognostic predictors on the probability of TFR in MR3 (pTFR3) and TFR in MR4 (pTFR4) on the TS. Variables with a p-value ≤0.1 entered in the multivariate analysis (MVA). Proportional hazard assumptions were tested for the final model. A prognostic TFR score was built from the combination of the predictors identified by the Cox model and validated on the VS.

**Results:** The TS included 118 pts, while the VS 54 pts (Table 1). In the TS, the 2-year pTFR3 was 67.4% (95% CI 66.5-68.3%) and the 2-y pTFR4 was 56.8% (95% CI, 55.9-57.7%). The median time to MR3 loss was 3.8 months (range 1-31), and for MR4 loss was 3.2 months (range 0.8-24.5). After loss of MR4, the 1-year probability of MR3 loss was 77% (95% CI, 70.8-73.2%). However, 10 pts (8.5%) resumed TKI after MR4 loss and were not evaluable for time to loss of MR3. In univariate analysis, the variables most significantly associated with higher pTFR3 and pTFR4 were age at diagnosis >40 years (p=0.029 and p=0.002), absence of previous TKI resistance (p=0.003 and p= 0.068), longer duration of MR4 (p=0.003 and p<0.0001) and ≥MR4.5 at stopping (p=0.026 and p= 0.004). Variables entered into the MVA were age at diagnosis, BCR-ABL1 transcript type, Sokal score, dose of TKI at stopping, previous TKI resistance, duration of MR4 at stopping, depth of response at stopping. The Cox model suggested the inclusion of the following variables, for both pTFR3 and pTFR4: duration of MR4, previous TKI resistance, age at diagnosis and transcript type. Using these variables we developed a predictive score (Figure 1a), which was able to identify a good risk population (2-y pTFR3 81.8%, 2-y pTFR4 80%); intermediate (66.6% and 61.5%) and poor risk (42.3% and 30.8%) (overall log-rank test p=0.00092 and p <0.0001 for pTFR3 and pTFR4, respectively)(Figure 1b). The score was tested on the VS of 54 pts. In this population, the overall 2-y pTFR3 and pTFR4 were 61.3% (95% CI, 59.8-62.7%) and 42.6% (95% CI, 41.2-44%), respectively. Despite the small sample size, our
score was still able to predict different 2-y TFR probabilities (Figure 1c) in the three risk groups. Of the pts who lost MR3 in the TS (n=39), 37 regained ≥MR3 after resuming TKI; 1 patient did not restart TKI and died from an unrelated cause;

1 patient had only 2 months follow-up after TKI resumption. In the VS, 15 of 21 pts losing MR3 achieved ≥MR3 again after TKI resumption; 3 pts had a follow-up <3 months after MR3 loss, 2 were lost to follow-up and 1 had not yet re-gained MR3 6 months after restarting TKI. In both cohorts no case of disease progression had occurred at last follow-up.

**Conclusions:** This retrospective study confirms the safety of TFR attempt and identifies variables strongly associated with prolonged TFR. The resulting predictive score presented here, if validated in larger patient cohorts, might help in tailoring the choice of TKI discontinuation to the individual patient. Also, most pts who lose MR4 inevitably lose MR3, suggesting the importance of a more intense monitoring strategy in this subgroup.
Table 1a. The TFR prognostic index

The final risk model, for each patient \( i = 1, \ldots, 138 \) of the TS had the following form:

\[
\lambda_i(t) = \exp(b_0 + b_1 \text{Age}_i + b_2 \text{PreviousTKI}_i + b_3 \text{Stage}_i + b_4 \text{BMI}_i + b_5 \text{TranscriptType}_i)
\]

The prognostic index score was then taken to be the linear predictor of the risk model:

\[
\text{Score}_i = b_0 + b_1 \text{Age}_i + b_2 \text{PreviousTKI}_i + b_3 \text{Stage}_i + b_4 \text{BMI}_i + b_5 \text{TranscriptType}_i
\]

where \( b_0, b_1, b_2, b_3, b_4, b_5 \) are the actual coefficient values estimated from the risk model.

![Figure 1a. The TFR prognostic index](image)

**Figure 1b.** Hammersmith cohort - TS

**Figure 1c.** External cohort - VS

![Figure 1c. External cohort - VS](image)
27 A Report on 114 Patients Who Experienced Treatment Free Remission in a Single Institution during a 15 Years Period: Long Term Follow-up, Late Molecular Relapses and Second Attempts

Philippe Rousselot, MD

Background. The A-STIM (According to Stop IMatinib, NCT1038732) observational study established the loss of major molecular response (loss of MMR, BCR-ABL1 IS >0.1%) as a practical and safe criterion for restarting therapy in patients with CML who had stopped tyrosine kinase inhibitors after a prolonged (≥2 years) and sustained deep molecular response (J Clin Oncol. 2014;32(5):424-30). We focus now on the long-term prospective follow-up of a cohort of 114 patients from a single institution included in the A-STIM observatory in order to describe late events (late molecular relapses after 2 years or more in TFR and second TFR attempts).

Methods: Adult chronic phase CML patients treated with tyrosine kinase inhibitors and in sustained (≥2 years) MR4.5 (BCR-ABL1 IS ≤0.0032%) were eligible. Patients with a previous history of resistance or mutations of the BCR-ABL tyrosine kinase domain were excluded. Molecular relapses were defined by loss of MMR. After TKI discontinuation, BCR-ABL transcripts were monitored monthly during the first 12 months, every 2-3 months during the 2nd year and every 3-6 months thereafter. Median follow-up from diagnosis of CML was 15.9 years. Results. Over a 15 years period, 114 patients followed at the Centre Hospitalier de Versailles were registered. Median age at diagnosis was 48.2 years, sex ratio was 0.5 and Sokal score distribution was 54%, 26% and 20% for the Low, intermediate and high-risk categories respectively. Median duration of TKIs before the first TFR attempt was 7.4 years. Thirty-six patients (31%) were previously treated with interferon, 62 (54%) received imatinib only and 52 (46%) were on 2G-TKIs at the time of discontinuation (13 as first line therapy and 39 after a switch for sub-optimal response or intolerance). Median follow-up in TFR1 was 5.4 years. TFR1 rates were 57.6% at 1 year, 53.8% at 3 years, 51.6% at 5 years and 44.5% after 7 years. The longest duration of ongoing TFR1 is 14.9 years. The duration of TKIs and the duration of MR4.5 were associated with a higher TFR1 rate; a trend was observed for previous exposure to interferon. Patients on 2G-TKIs (first or second line) had similar TFR1 rates as compared to patients on imatinib. Fifty-seven patients relapsed including 8 patients (14%) experiencing late molecular relapses. Of those, 4 patients relapsed after 5 years. The latest molecular relapse was observed after 6.4 years. In late relapsing patients, MR4.5 was lost after 10 months in median and MR4 after 22 months with a long-lasting period of fluctuations of the BCR-ABL1 ratio in-between MR4 and MR3 (a focus on patients with fluctuations of the BCR-ABL1 ratio will be presented at the congress). Out of the 57 patients who restarted a TKI, 31 patients (54%) experienced a second attempt. Median duration of TKIs between TFR1 and TFR2 was 2.9 years and total exposure to TKIs before TFR2 was 9 years. Fifteen patients (48%) were on imatinib before
TFR2 whereas 16 where on 2G-TKI (52%). Median follow-up in TFR2 was 3.4 years. TFR2 rates were 53.9% at 1 year, 45.6% at 3 years and 39.9% after 5 years.

The longest TFR2 is 9 years. No factor was associated with TFR2 duration, a switch to 2G-TKIs did not provide any advantage. Seventeen patients relapsed including 3 patient (17%) experiencing late molecular relapses. Anecdotally, 5 patients went to a third TFR attempt and 1 is in TFR3 for 5.2 years. Conclusion. Based on a 15 years’ experience we were able to report on long term follow-up in TFR1 and in TFR2. Among patients experiencing molecular relapses, we observed 14% and 17% late relapses after more than 2 years after TFR1 and TFR2 respectively, suggesting that a long-term molecular follow-up is mandatory for CML patients in TFR.

Long-term TFR rates in 114 patients over a 15 years period

![Graph showing long-term TFR rates](image-url)
28 The TKI-Free Duration after a First Discontinuation Attempt That Failed in CP CML Patients Is a Predictive Factor of TKI-Free Remission after a Second Attempt

Laurence Legros, MD, PhD

Background: Tyrosine kinase inhibitors (TKIs) are able to induce, in some chronic myeloid leukemia (CML) patients in chronic phase (CP), long-term molecular response 4.5 (MR4.5) and several studies have now demonstrated that TKIs could be safely discontinued in those patients with a Treatment-Free Remission (TFR) rate reaching ~50%. The French CML group had recently demonstrated that a failure of the first TKI discontinuation attempt does not preclude a 2nd successful attempt (RE-STIM study, Legros et al. Cancer 2017).

Methods: The RE-STIM study is a national observational multicentre study collecting all cases of 2nd TKI discontinuation attempt of regardless the type, the duration of TKI, the duration of MR4.5 and the reason of discontinuation. CP-CML Patients in failure of a 1st attempt, had to recover a 2nd sustained MR4.5 on TKI to be eligible for this new analysis of the enlarged database (n=106). Loss of MMR loss was the trigger for therapy re-introduction.

Results: At the time of analysis (1st June 2019), 106 patients (median age: 55 years (range: 25-81 years)) were included with 41 months (2-131) of follow-up after 2nd discontinuation. Fifty males and 56 females were enrolled. The Sokal risk score was low in 45%, intermediate in 26.5%, high in 20% and unknown in 8.5% of patients. The majority of patients (95%) were treated with imatinib as first-line, and the others with a 2nd generation TKI. The median total time on TKI prior to a 2nd discontinuation was 104 months (range: 38-235) and the median duration of a 2nd MR4.5 prior to a 2nd discontinuation was 68 months (range: 20-176). After a 1st discontinuation attempt, the reason for TKI re-challenge was in majority a loss of MMR (66%), a loss of MR4.5 in 33% of patients (missing data in 1%).

The TFR rates after a 2nd discontinuation attempt were 44.3% [95% CI 35.48-55.41] at 24 months, 38.5% [95% CI 29.65- 50.09] at 36 months and 33.2% [95% CI 24.31- 45.39] at 48 months. In univariate analysis, we failed to find any association between TFR and: age, gender, Sokal score, prior exposure to IFN, TKI in combination versus monotherapy, TKI type, TKI treatment duration and uMR4.5 duration before the 1st and 2nd discontinuation attempts, and type of molecular relapse after the 1st discontinuation attempt (MR4.5 versus MMR loss). However, the speed of molecular relapse after the 1st TKI discontinuation remains a factor significantly associated with outcome. In patients who remained in uMR4.5 at 3 months after the 1st discontinuation, the TFR rate at 48 months was 53% [95% CI: 35.32-79.31] and 26% [95% CI: 16.88-40.28] for others. Another factor significantly associated with outcome is the TKI-free duration after the 1st attempt (Figure). The TFR rate at 48 months was 45 % [95% CI: 28.64- 69.62] in patients who remained without treatment more than 6 months after their 1st attempt and 27% [95% CI: 17.57- 41.34] for others.
All patients are alive at last follow-up except 2 who died from CML-unrelated reasons. One patient developed a sudden blast crisis at 4 years from 2nd discontinuation. The last previous molecular biology 3 months before transformation was MR4. In patients in TKI re-challenge (n=63), median TKI-free duration was 6 months (2-64), 55% of patients regained their MMR within 3 months (0-35) and 41% regained MR4.5 within 5 months (2-53).

Conclusions: This larger cohort confirms that TKIs could safely and successfully be discontinued a 2nd time in CP CML patients despite a 1st failure. The speed of molecular relapse after the 1st TKI discontinuation and TKI-free duration remain major factors significantly associated with TFR outcome.

29 Increased Tumour Burden over a 36 Month Period in Chronic Myeloid Leukemia Patients Following Imatinib Discontinuation: Role of Digital PCR

Elisa Diral, MD

Introduction: Discontinuation (D/C) of Imatinib or other TKIs in Chronic Myeloid Leukemia (CML) represents an important issue in the management of this disease. It is generally accepted that relapse develops (and treatment must be resumed) when patients (pts) lose Major Molecular Remission (MMR), i.e. when the amount of the BCR-ABL1 transcript, measured in peripheral blood by RT-PCR, exceeds 0.1 %. The Imatinib Suspension And Validation (ISAV) study, which started in 2011, enrolled pts with CML treated with Imatinib who showed no evidence of BCR-ABL1 transcript for at least 18 months before enrollment.
Methods: A digital PCR (dPCR) for BCR-ABL1 was performed at the time of Imatinib D/C while a second dPCR was performed when non-relapsed patients exited the study, 36 months later. dPCR experiments were performed by the QX200 system (BioRad) in the same lab and using the same methodology. The BCR-ABL1 fusion and ABL1 transcripts were quantified using DigiDrop P210 MasterMix and DigiDrop P210 Positive Control (Bioclarma), according to manufacturer’s protocol. The target concentration in each sample was expressed as percentage of BCR-ABL1/ABL1.

Results: A total of 107 pts were enrolled in the ISAV study. Relapses occurred in 54 pts (52%); among the 53 non relapsed pts, 41 (77%) presented at least one positive RT-PCR result following Imatinib D/C, and only 12 (23%) maintained PCR negativity throughout the duration of the study. Among the non-relapsed pts dPCR performed at treatment D/C showed positivity in 20.6% of cases (95% confidence interval [C.I.] 9-36%), while 91.1% of pts (95%, C.I. 80-97%) evaluated 36 months later showed a positive dPCR value, although no patient resumed treatment. The evaluation of non relapsed pts by dPCR showed that mean values at D/C were 0.00143% +/- 0.0006 (SE); when tested at study exit, the same pts showed average dPCR values of 0.0115 % +/- 0.002.

This difference is statistically highly significant and corresponds to a change of approximately 1 log in the residual tumor burden: from 2x10^7 to 2x10^8 cells. There was no correlation between the results of RT-PCR performed during the study and the dPCR status at study exit: pts who tested negative by RT-PCR during the study were uniformly negative in dPCR at D/C but tested positive at study exit in 83.3% of cases; pts who showed at least one positive RT-PCR during the study showed positivity by dPCR in 25% at D/C and in 89.3% at study exit. Finally, half of the pts who tested negative by dPCR at study exit showed dPCR positivity when tested at the time of Imatinib D/C.

Conclusions: These results show that during a three year period, the D/C of Imatinib led to the increase of approximately 1 log in the tumour burden of non-relapsed pts, although none of them lost MMR and resumed treatment.

These data strongly indicate the need for a long-term monitoring of pts who D/C Imatinib; they also suggest that the functional status of residual CML cells rather than their number could represent the critical factor to predict the tumour load present after 3 years of Imatinib D/C.
30 Prognostication of Molecular Relapses after Dasatinib or Nilotinib Discontinuation in Chronic Myeloid Leukemia (CML): A FI-LMC STOP 2G-TKI Study Update

Delphine Rea, MD, PhD

Background: Providing achievement and sustainability of deep molecular responses (DMR), patients (pts) taking tyrosine kinase inhibitors (TKI) against CML may discontinue therapy. The STOP 2G-TKI observational study showed that dasatinib and nilotinib could be safely stopped and prior suboptimal response or resistance to imatinib was an adverse prognostic factor for treatment-free remission (TFR). We present updated results with a specific focus on the risk of relapse using post-baseline information during follow-up.

Methods: Adult CML pts treated with dasatinib or nilotinib without a history of allogeneic stem cell transplantation (ASCT) or progression to advanced phase stopped TKI provided that: (1) BCR-ABL transcripts were of the major type, (2) total TKI treatment duration was ≥36 months, (3) uMR4.5 had been achieved and maintained for ≥24 months (undetectable BCR-ABL with ≥32000 copies of ABL). Relapse was defined by loss of major molecular response (MMR: BCR-ABL IS >0.1%) on a single occasion and triggered TKI reintroduction. The primary objective was TFR at 12 months. After TKI discontinuation, BCR-ABL transcripts were monitored monthly during the first 6-12 months, every 3 months during the 2nd year and then every 3-6 months. Data as of July 1, 2019 are reported in 104 pts (median follow-up 55 months (range: 6-70)).

Results: Median age at inclusion was 56 years (range: 21-82) and 65.4% of pts were female. Sokal risk score was low in 49%, intermediate in 31%, high in 16% and unknown in 4%. 2G-TKIs were given after imatinib intolerance in 47% of pts, suboptimal response or resistance to imatinib in 22%, lack of DMR on imatinib in 3% and as 1st line treatment in 28%. Median duration of TKI, 2G-TKI and uMR4.5 was 74 months (range: 36-163), 49 months (range: 19-112) and 31 months (range: 24-72), respectively.

Overall, 43 pts (41%) lost MMR within a median time of 5 months (range: 1-59). Overall 60-month TFR was 56% (95% CI: 45.8-66.3) but TFR probabilities increased up to 64% (95% CI: 53.3-74.8), 76.7% (95% CI: 65.9-87.5), 86.2% (95% CI: 76.3-96.2), and 92.1% (95% CI: 83.4-100) for pts still in MMR at 3, 6, 12 and 18 months, respectively (Figure 1).

Prior suboptimal response or resistance to TKI was confirmed as the strongest adverse baseline prognostic factor with a 60-month TFR rate of 29.8% (95% CI; 10.8-48.7) (median TFR 12 months) versus 63.6% (95% CI: 52.1-75.2) (median not reached) in pts without such history (logrank p=0.0012). This was explained by significantly higher risk of early relapses (within 6 months but not later) in pts with prior suboptimal response or resistance to TKI (cumulative incidence of
relapses by 6 months 47.8% (95% CI; 31.2-73.2) versus 20.9 (95% CI; 13.7-32) in other pts (p=0.00879)).

Landmark analyses at specific time points were performed to study the prognostic value of molecular responses categories after TKI discontinuation. All pts in MMR but not deeper at 3 months relapsed by month 9 (median time to relapse 4 months) while pts in ≥MR4 (BCR-ABL IS ≤0.01%) had 12- and 60-months probabilities of 86.8% (95% CI; 79.1-94.4) and 74.9% (95% CI: 64-85.7), respectively (logrank p<10-4). All pts but 1 in MMR but not deeper at 6 months relapsed (median time to relapse 12 months) while pts in ≥MR4 had 12- and 60-months probabilities of 95% (95% CI; 89.6-100) and 87.5% (95% CI: 78.7-96.2), respectively (logrank p<10-4).

Forty three pts restarted treatment including 1 who lost MR4.5 but not MMR and 42 who lost MMR. When treatment was reintroduced, 42 pts were in CHR and all regained MMR after a median time of 3 months (range: 1-11). The remaining pt lost MMR but not CHR 5 month after 1st line nilotinib cessation and was found in sudden myeloid blast crisis at the month 6 TKI reintroduction visit. No BCR-ABL mutation was found but an inversion of chromosome 3 at karyotyping analysis. The pt underwent ASCT after chemotherapy + ponatinib and is alive in remission 29 months later.

**Conclusion:** 2G-TKI may be successfully stopped in CML pts with long-lasting MR4.5. Those without a history of suboptimal response or resistance have greatest chances of success. Sudden blast crisis is rare but unpredictable. Post-TKI discontinuation estimates of TFR change overtime. Together with that of molecular response type at specific time points, they represent important dynamic prognostic measures of outcome. They may also help individualizing molecular monitoring programs after TKI cessation.
Figure 1:
Kaplan-Meier estimates of TRR at TKI discontinuation (black line) overlaid by TRR estimates in pts still in MMR at 3 months (pink line), 6 months (blue line), 12 months (green line) and 18 months (red line)