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VIRTUAL EDITION 2020

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Virtual Edition 2020

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25th Congress of the European Hematology Association

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CML patients who experienced relapse, after imatinib discontinuation, based on the probability of molecular recurrence free survival (MRFS) at 12 months in patients treated by nilotinib for 2 years with a sustained MR4.5 for at least 1 year. Secondary objectives included the rate of MR4.5 achievement and the tolerance of the drug.
Methods: Fine and Gray model and Kaplan-Meier method were used to assess the cumulative incidence of adverse events on nilotinib and to estimate MRFS respectively.
Results: Between April 2013 and February 2018, 31 patients from the EURROSKI (n = 16) and STIM2 (n = 9) clinical trials, or who were treated according to the STIM2 protocol (n = 6) were included. MolRec after imatinib discontinuation was defined by loss of major molecular response (MMR) in 29 patients or increase of 1 log of BCR-ABL1 transcript in 2 patients. Among the 31 patients included, 7 patients experienced adverse events (AE) after the initiation of nilotinib leading to treatment discontinuation: myocardial infarction at 2 months (n = 1), pain of left hypochondrium at 4 days (n = 1), coronary atherosclerosis at 3 months (n = 1), extrasystoles at 1 month (n = 1), grade 3 skin rash at 1 month (n = 1), headache, vomiting and asthenia at 3 months (n = 1) and grade 3 liver toxicity at 6 months (n = 1). All required hospitalization except the 2 last AEs. The cumulative incidence of adverse events leading to nilotinib discontinuation is 22% at 6 months (CI 95%:12-41) (Fig1a). During treatment, one additional patient wishing to become father was excluded at 9 months although he was in MR4.5. Among the 23 patients treated with nilotinib during 2 years, all reached MR4.5 and 22 (96%) maintained their molecular response during at least 1 year of nilotinib (median: 21.7 months: min-max: 13-23 months) and stopped nilotinib. Among the 20 patients with at least 6 months of follow-up after discontinuation, 7 (35%) patients experienced a MolRec, defined as loss of MMR. Median time to MolRec was 4.1 months (min-max: 2-12.3 months). MRFS at 12 months after nilotinib discontinuation is 35% (CI 95%: 2-76) (Fig 1b).

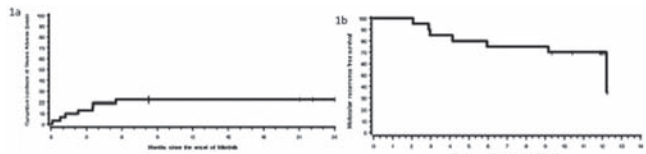

Figure 1: Cumulative incidence of adverse events leading to nilotinib discontinuation (1a) and molecular recurrence free survival of the 20 patients who experienced a 2nd attempt, with at least 6 months of follow up after discontinuation (1b)

Summary/Conclusion: Nilotinib treatment induces a high rate of sustained deep molecular response and can lead to successful second TFR attempt in patients having previously failed a first stop of imatinib. However, MRFS is not better than those already reported (Legros et al. 2017), even if this preliminary result has to be confirmed. Several adverse events including 3 cases of cardiovascular toxicities were observed in this study. They might be explained by the lack of patient selection according to cardiovascular risk factors and of guidelines for their management at the time of Nilo Post-STIM trial onset.

EP752 PONATINIB AT A LOWER DOSE IS A GOOD OPTION IN CHRONIC MYELOID LEUKEMIA (CML) PATIENTS INTOLERANT TO PREVIOUS TKIS

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Background: Ponatinib is a third-generation tyrosine kinase inhibitor (TKI) with proven efficacy in CML patients resistant to previous TKIs. However, some warnings emerged concerning its cardiovascular (CV) safety profile when used at the recommended starting dose of 45 mg/day. It was suggested that ponatinib may cause endothelial dysfunction through its multikinase inhibitory properties affecting targets such as vascular endothelial growth factor receptor (VEGFR) and promoting the expression of proatherogenic surface adhesion receptors. Recently, it was reported a relationship between CV adverse events (AEs) and the dose, but a reduction of the daily dose has been suggested for patients who have already achieved at least a MCyR. Indeed, very limited data have been reported in the literature so far on the use of low dose ponatinib after intolerance to previous TKIs.
Aims: The primary objective was to evaluate the safety and efficacy profile (molecular response rate) of low dose ponatinib when used in the real-life setting of CML patients intolerant to previous TKIs.
Methods: We evaluated 46 consecutive CML patients in the real-life setting, treated with ponatinib because of intolerance to previous TKIs, between May 2012 and December 2019 in 13 Italian hematological centers. Responses to TKIs were evaluated according to the current European LeukemiaNet recommendations. Hematological and extra-hematological toxicity was graded according to the Common Terminology Criteria for Adverse Events (CTC-AE).
Results: Male:female ratio was 2:1, with a median age at ponatinib start of 64.5 years (range, 34.5 - 81.0). Among comorbidities at baseline, hypertension was present in 19 (41.3%), diabetes in 6 (13%) and dyslipidemia in 4 (8.7%) patients. All of the cases started ponatinib because of intolerance to at least one TKI, most frequently dasatinib (31 patients, 67.4%), followed by nilotinib (11, 23.9%) and bosutinib (4, 8.7%). Median time from diagnosis to ponatinib treatment was 5.0 years (range, 0.7 - 23.4). Median starting dose of ponatinib was 22.5 mg daily, administered as a second-line in 19 (41.3%), third-line in 18 (39.1%) and fourth-line in 9 (19.6%) cases. At a median follow-up from ponatinib start of 22.2 months (range, 0.4 – 93.5), 35 (76.1%) patients increase the depth of molecular response (including 8 patients with MMR and 15 with DMR), with the remaining patients maintaining the same level of molecular response already achieved before ponatinib start. Interestingly, among patients who obtained a DMR during ponatinib, treatment-free remission was successfully achieved in one case. Concerning instead the safety profile, AEs were reported in 17 (36.9%) patients, including pancreatitis in 5 (10.9%), hypertension in 4 (8.7%) and cardiovascular events in 4 (8.7%) cases [including one acute myocardial infarction (IMA) and one ischemic stroke], the latter both registered in patients with pre-existing cardiovascular risk factors and who were initially treated with ponatinib at a daily dose of 30 mg. In addition, the patient who suffered from IMA received also nilotinib as a previous treatment.
Summary/Conclusion: Our data highlight the efficacy and safety of low dose ponatinib in the setting of CML patients who were intolerant to previous TKIs. A reduced incidence of specific AEs was recorded, although the efficacy has not been affected. This treatment strategy could represent a possible alternative for intolerant patients: further prospective studies are needed to assess in the long-term the benefits of dose reduction.

EP753 LONG-TERM FOLLOW UP OF CHRONIC MYELOGENOUS LEUKEMIA PATIENTS WITH CLONAL CHROMOSOMAL ABERRATIONS IN PHILADELPHIA NEGATIVE CELLS TREATED WITH TYROSINE KINASE INHIBITORS

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Background: The emergence of novel clonal aberrations (NCAs) in Ph-metaphases was rare in chronic myelogenous leukemia (CML) patients treated with interferon-a, whereas it is significantly higher in those treated with tyrosine kinase inhibitors (TKIs). The biological and clinical implications of NCAs are unclear.
Aims: We investigated the frequency of the occurrence and the prognostic significance of this phenomenon among 200 CML patients treated with TKIs.
Methods: We studied 200 CML patients who were treated with TKIs and monitored according to the ELN 2013 recommendations. Cytogenetic analyses were conducted on bone marrow cells to assess the degree of cytogenetic response (CyR).
Results: 21/200 patients (10, 5%) with a median time from diagnosis to their last follow-up (FUP) of 156 months (range 20-240), developed NCAs in Ph- cells, with a median time from the beginning of treatment of TKIs 58months (range 3-204). 14 patients received only imatinib, 1