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ABSTRACT BOOK

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The origin of a name that reflects Europe's cultural roots.

Ancient Greek

αἷμα [haima] = blood
αἵματος [haimatos] = of blood
λόγος [logos]= reasoning

Scientific Latin

haematologicus (adjective) = related to blood

Scientific Latin

haematologica (adjective, plural and neuter, used as a noun) = hematological subjects

Modern English

The oldest hematology journal, publishing the newest research results.
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affect disease progression, we analyzed the risk of incurring into death for any cause, BT and HSCT, according to the genotype. In the composite outcome model, the MCP-1 high-risk group showed a significantly shorter time to event (HR 2.56; 95%CI 1.19-5.55; p=0.016). In conclusion, homozygosity for the MCP-1 SNP appears to identify PMF at high risk of a more severe disease presentation, a pro-inflammatory state and a higher chance to progression.

C020

RISK FACTORS FOR PROGRESSION TO BLAST PHASE IN PATIENTS WITH MYELOFIBROSIS TREATED WITH RUXOLITINIB: AN ITALIAN STUDY ON 589 PATIENTS

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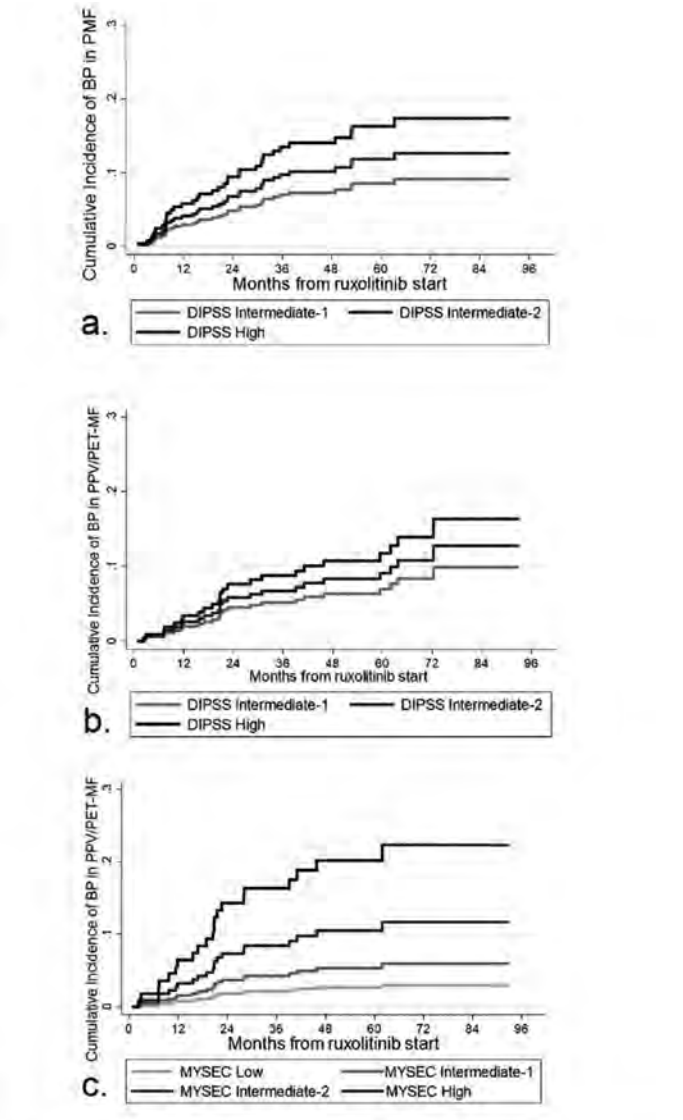
In the pre-ruxolitinib (RUX) era, several risk factors for progression to blast phase (BP) have been proposed in myelofibrosis (MF) patients (pts), namely peripheral blasts, thrombocytopenia, unfavorable cytogenetics, and high risk category. However, predictors of BP during ruxolitinib (RUX) therapy are unknown. To evaluate incidence and risk factors of BP in RUX-treated MF pts, we retrospectively collected clinical/laboratory data of 589 pts who received RUX in 20 European Hematology Centers. A time-to-event (BP) analysis was conducted with Fine & Gray model with death/time of stem cell transplant as competing risks. Cumulative Incidence Function among risk categories for DIPSS and MYSEC-PM was calculated applying the Gray's model. Of 589 MF pts [PMF, n.303, 51.5%; post-Polycythemia Vera MF (PPV-MF, n.164, 27.8%); post-Essential thrombocythemia MF (PET-MF), n.122], 65 developed BP. In 61 pts, BP caused RUX withdrawal after a median time of 1.2 yrs (0.6-6.8); in 4 pts BP occurred after RUX stop (median time: 2.6 yrs). BP incidence rate was 3.6 x100 pt-yrs and was comparable in PMF and PPV/PET-MF (p=0.1). In multivariable analysis, the probability of BP evolution for the entire cohort was significantly associated with PLT <150x10^9/l (HR[95% CI]: 2.03[1.10-3.74], p=0.02), spleen length ≥10 cm (HR[95% CI]: 2.56[1.21-5.42], p=0.01), and blasts ≥3% (HR[95% CI]: 2.06 [1.02-1.14], p=0.04). Analysing PMF separately, univariate analysis failed to detect parameters associated with BP evolution. Conversely, in PPV/PET-MF, predictors for BP were PLT <150x10^9/l (HR[95% CI]: 3.72[1.35-10.24], p=0.01) and blasts ≥3% (HR[95% CI]: 3.05[1.12-8.29], p=0.03); previous interferon (IFN) significantly reduced the risk of BP (HR[95% CI]: 0.72 [0.61-0.83], p<0.001). High DIPSS risk significantly predicted BP evolution in PMF (p=0.04, HR [95% CI]: 2.6 [1.1-6.5]) but not in PPV/PET-MF (p=0.35). In this latter cohort, only the MYSEC-PM score was associated with BP (p=0.01) (Fig.1). Estimated HRs, in reference to the lower score category, were: 1.18 (CI 95%: 1.10-1.29) for intermediate-1, 2.80 (CI 95%: 1.51-20.34) for intermediate-2, and 5.52 (CI 95%: 2.04-19.63) for high risk. HR for high risk pts, comparing to all lower risk groups, was 2.86 (CI 95%: 1.23-6.61). Overall, 11% of RUX-treated pts developed BP. The risk of BP was increased by thrombocytopenia/peripheral blasts at RUX start, and decreased by IFN use. DIPSS and MYSEC-PM predicted BP in PMF and PPV/PET-MF, respectively.



Cumulative Incidence of blast phase transformation (BP) according to DIPSS risk score in PMF (a), DIPSS risk score in PPV/PET-MF (b) and MYSEC-PM risk score in PPV/PET-MF (c).

Figure 1.