Platelet recovery delay and survival in patients with myelofibrosis undergoing allogeneic hemopoietic stem cell transplantation

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Received: June 11, 2025.
Accepted: January 7, 2026.
Early view: January 15, 2026.

https://doi.org/10.3324/haematol.2025.288371

Abstract

We studied platelet recovery in 93 patients with myelofibrosis, following an allogeneic hemopoietic stem cell transplant (HSCT). The primary end point of the study was achieving a platelet count of 50x10^9/L within day +100 post transplant, which occurred in 62 patients (67%), predicted 5-year non-relapse mortality (NRM) (5% vs. 55%, *P*=0.0009) and 5-year actuarial survival (85% *vs*. 38%, *P*<0.00001); relapse was unaffected. The cumulative incidence of strong platelet recovery was predicted by a matched sibling donor (MSD) compared to alternative donors (90% *vs*. 60%, *P*=0.001), by the dose of CD34$^+$ cells (cut off 8.68x10^6/kg; 83% *vs*. 61%, *P*=0.01), recipient age (cut off 63 years; 72% *vs*. 48%, *P*=0.01), and splenectomy (86% *vs*. 63%, *P*=0.04). In multivariate Cox analysis, significant predictors of platelet recovery were a MSD (*P*=0.003), a high CD34 cell dose (*P*=0.02), splenectomy (*P*=0.003), and younger patient age (*P*=0.02). Patients with slow platelet recovery have significantly lower platelet counts long term, combined with chronic graft-*versus*-host disease. In conclusion, strong post-HSCT platelet recovery in MF patients is mainly predicted by donor type, together with CD34 cell dose and patient age, and is strongly associated with NRM and survival.

Introduction

Delayed platelet recovery has been described in a significant proportion of patients following an allogeneic hemopoietic stem cell transplant (HSCT)[1] and has been associated with increased non-relapse mortality (NRM).[1-4] Predictors of delayed platelet recovery include low CD34 cell dose,[5] graft-*versus*-host disease (GvHD),[4] age, performance score, and donor type.[1,2] Hematologic recovery is of particular importance in patients with myelofibrosis (MF) undergoing an allogeneic HSCT, since two hallmarks of the disease are bone marrow fibrosis (graded from MF1 to MF3) and splenomegaly.[6] Both these two factors can delay platelet recovery, either by trapping progenitors in the enlarged spleen, and/or by providing a fibrotic marrow environment unsuitable for stem cell homing. In a study comparing MF and leukemia patients after an allogeneic HSCT, the number of circulating CD34 cells post transplant was significantly reduced in MF patients, suggesting spleen pooling.[7] Bone marrow sections exhibited reduced VCAM-1 expression, a key adhesion molecule on endothelial cells, suggesting a microenvironment less suitable for stem cell homing.[7] Prolonged cytopenia results in prolonged transfusion dependence, poor graft function (PGF), and elevated NRM; the 5-year survival of patients with PGF is reported to be 14%.[8]

The aim of this study in MF patients undergoing an allogeneic HSCT was to identify a surrogate marker of strong hematologic recovery, better predicting NRM and survival, and subsequently assess pre-HSCT and HSCT factors predicting hematologic recovery.

Methods

Patients who had undergone a first HSCT for MF at our transplant center between January 2016 and June 2024 who were alive on day +20 post transplant and who had achieved a neutrophil count of 0.5x10^9/L were included in the study. We assessed time to a neutrophil count of 0.5x10^9/L,

time to a platelet count of 20x10^9/L, and time to a platelet count of 50x10^9/L. Variables studied were disease-related (Dynamic International Prognostic Scoring System [DIPSS] score, degree of marrow fibrosis [MF1, MF2, or MF3], driver mutations, spleen size), treatment-related (splenectomy, ruxolitinib, transfusion burden), and transplant-related (conditioning regimen, GvHD prophylaxis, donor type, patient age and gender). Spleen size was recorded as the largest spleen size in the clinical history of the patient.

Transplant procedures

The selection of donors was prioritized as follows: HLA identical sibling donors (MSD), HLA matched (8/8) unrelated donors (MUD), 7/8 HLA matched unrelated donors (UD), and haploidentical family donors (HAPLO). The conditioning regimen was a combination of 10 mg/kg thiotepa, 150 mg/m^2 fludarabine and intravenous busulfan (3.2 mg/kg/day in 4 separate doses) for two consecutive days (N=65) or one day (N=23) (TBF); 5 patients received a non-myeloablative regimen including fludarabine, cyclophosphamide, and 2 Gy total body irradiation. The stem cell source was peripheral blood (N=73) or bone marrow (N=20).

Only two regimens of GvHD prophylaxis were used. Regimen A consisted of cyclosporine (CSA) (intravenous [i.v.] 3 mg/kg; day -1 onwards), 10 mg/m^2 methotrexate (MTX) (days +1, +3, +6, and +11), 4.5 mg/kg anti-thymocyte globulin (ATG) (Rabbit Thymoglulin; Sanofi, France) (N=13). Regimen B consisted of 50 mg/kg post-transplant cyclophosphamide (PTCY) days +3, +4, combined with CSA day +5 onwards and mycophenolate (MMF) day +5 until day +30 (N=??).

The study was conducted in accordance with the principles of the Declaration of Helsinki and with the approval from the Gemelli Ethical Committee (ID 4751 Prot 6539/22). All patients provided written informed consent for research studies using an institutional form that was approved by the institutional ethics committee.

Chimerism studies and definition of full donor chimerism

Chimerism was assessed by polymerase chain reaction (PCR) analysis of short tandem repeats (STR). The proportion of donor recipient chimerism was calculated using the PowerPlex Fusion System (Promega srl,. Italy) on 24 STR loci. Full donor chimerism (F-DC) was defined as having >95% donor alleles.

Statistical analysis

Descriptive statistics included medians for continuous

Table 1. Clinical characteristics of patients with and without a platelet recovery of 50x10^9/L within day +100 post transplant.

Variable	Plt 50x10^9/L within day +100		*P*
	NO	YES	
N of patients (%)	31 (33)	62 (67)	-
Median donor age, years (range)	30 (19-63)	31 (20-61)	0.5
Donor gender, M/F	24/7	47/15	0.3
Median recipient age, years (range)	59 (42-73)	56 (36-69)	0.05
Recipient gender, M/F	19/12	34/28	0.5
ABO major mismatch (%)	12 (39)	18 (29)	0.5
Donor HLA id SIB (%)	2 (6)	18 (29)	0.01
Spleen > 20 cm, N pts (%)	12 (38)	24 (38)	1.0
Transfusions >20 (%)	9 (29)	12 (19)	0.2
DIPSS high risk (%)	11 (35)	24 (39)	0.7
MTSS high risk (%)	10 (32)	15 (24)	0.4
Fibrosis MF3 (%)	28 (90)	46 (74)	0.06
Pre-transplant ruxolitinib (%)	23 (74)	45 (72)	0.8
Splenectomy (%)	2 (6)	12 (19)	0.1
PTCY (%)	29 (94)	51 (82)	0.1
Comorbidity Index >2 (%)	19 (61)	26 (42)	0.07
Myeloablative conditioning (%)	21 (68)	43 (69)	0.9
Median CD34x10^6/kg (range)	5.68 (1.7-11.9)	6.3 (1.6-15.0)	0.05

DIPSS: Dynamic International Prognostic Scoring System; F: female; HLA id SIB: HLA identical sibling; M: male; MTSS: Myelofibrosis Transplant Scoring System; N: number; Plt: platelets; PTCY: post-transplant cyclophosphamide for graft-*versus*-host disease; pts.: patients.

variables and contingency tables for dichotomous variables. The cumulative incidence of platelet recovery was calculated using death without a platelet recovery as a competing event. The cumulative incidence of acute or chronic GvHD were calculated with death in the absence of acute or chronic GvHD as competing event. Survival curves were generated using Kaplan-Meier analysis. The cumulative incidence of NRM was calculated with relapse as a competing event and viceversa. All statistical analyses were run on the NCSS 2019 Statistical Software (NCSS, LLC, Kaysville, UT, USA; ncss.com/software/ncss).

Results

Hematologic recovery and outcome

All patients achieved a neutrophil count of 0.5x10^9/L at a median interval of 22 days (range 11-70). For 85 patients, the median time to a platelet count of 20x10^9/L was day 32 (range 11-159). We then identified the strongest marker of hematologic recovery which would affect NRM. The difference in time to a neutrophil count of 0.5x10^9/L was not predictive (*P*=0.3). The difference in time to a platelet count of 20x10^9/L was significantly shorter in patients without NRM (*P*=0.01). The difference in time to a platelet count of 50x10^9/L was highly significantly different in patients with NRM (*P*=0.0001). This was confirmed with a ROC analysis of platelet counts within day +100 and NRM, which identified a platelet count of 50x10^9/L as the best cut off, with a sensitivity of 85% and a specificity of 78%. Therefore, patients were stratified into 2 groups according to whether they achieved a platelet count of 50x10^9/L within day +100 or not. Table 1 outlines the clinical characteristics of these two groups. A platelet count >50x10^9/L within day +100 was achieved overall in 62 patients (67%) at a median interval of 40 days (range 15-98), which was confirmed by cumulative incidence analysis (67%, 95% Confidence Interval [CI]: 58-77%).

Outcome

The overall cumulative incidence of NRM was 20% at five years (95%CI: 13-30%). A platelet count of 50x10^9/L within day +100 predicted 5-year NRM (5% *vs*. 55%, *P*=0.00009) (Figure 1A) and 5-year actuarial survival (Figure 1B) (85% *vs*. 38%, *P*<0.00001). Relapse at five years was unaffected by hematologic recovery (19% and 22%, *P*=0.5). The cumulative incidence of acute GvHD grade II-IV was 19% (range 11-32%) *versus* 42% (range 27-63%) in patients with or without a platelet count of 50x10^9/L within day +100 post transplant (Gray test *P*=0.02). The 3-year cumulative incidence of moderate and severe chronic GvHD was 20% (range 14-35%) *versus* 46% (range 30-67%) (Gray test 0.02); the cumulative incidence of severe chronic GvHD was 2% (95%CI: 0.2-11%) versus 19% (95%CI: 11-39%) in patients with or without a platelet count of 50x10^9/L within day +100 post transplant (Gray test *P*=0.003).

Predictors of early platelet recovery

Table 2 outlines factors predictive of early strong platelet recovery in univariate analysis: donor type (MSD), younger patient age, higher number of CD34 cells infused, and splenectomy. In a multivariate Cox analysis, transplants from an MSD remained the most significant predictor (*P*=0.003), followed by splenectomy (*P*=0.004), a high CD34 cell dose (*P*=0.02), and older age (*P*=0.02) (Table 2). Donor and patient gender and age, primary or secondary disease, DIPSS risk category, Myelofibrosis Transplant Scoring System (MTSS) risk category, driver mutations, transplant year after 2019, conditioning regimen intensity, GvHD prophylaxis, maximum spleen size, transfusion burden pre-transplant, and graft source did not influence strong platelet engraftment (Table 2). We also ran a linear regression analysis between maximum platelet counts and conditioning intensity and found no correlation. When looking in detail at donor type,

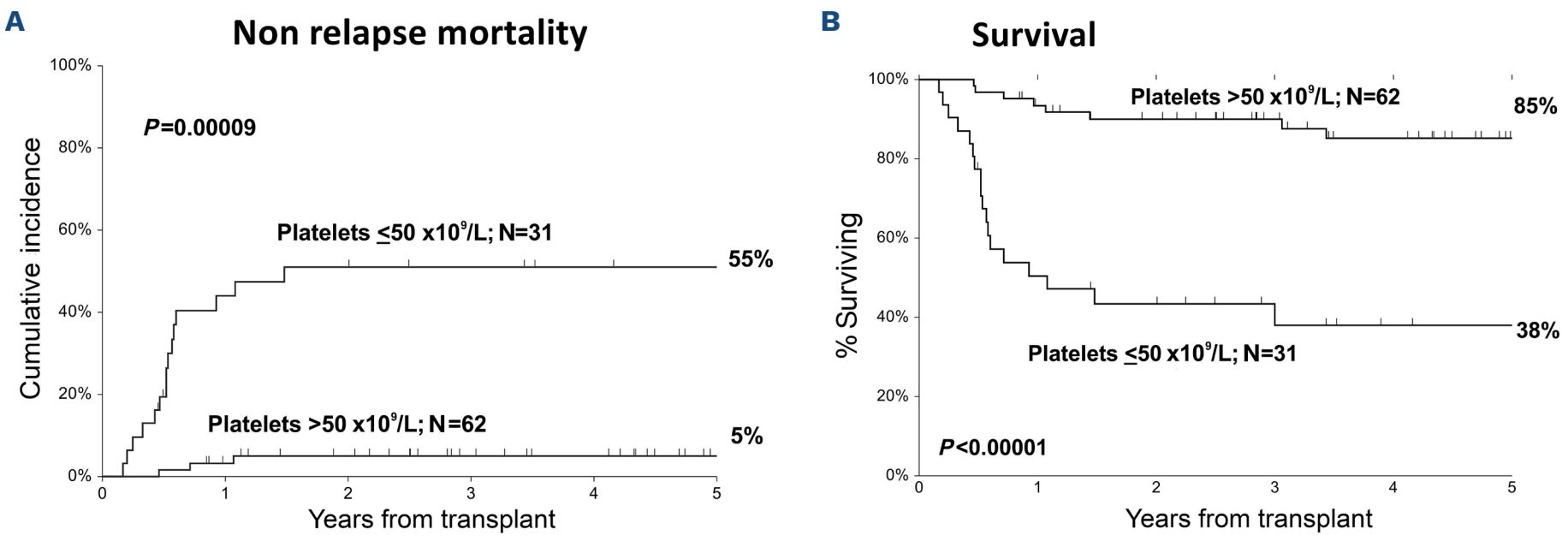

Figure 1. Overall cumulative incidence of non-relapse mortality (5% *vs*. 55%) (A) and actuarial survival (85% *vs*. 38%) (B) in patients stratified for achieving or not a platelet count of 50x10^9/L within day +100.

we found strong platelet recovery in 90% MSD (N=20), 55% in HAPLO (N=20), 62% in MUD (N=40), and 62% in mismatched UD (N=13). Driver mutations did not seem to affect platelet recovery (JAK2, 45/69, CALR, 8/14, MPL, 4/5 and triple negative 5/5, *P*=0.3). ABO mismatch also had no significant impact on strong platelet recovery: ABO match (65%), ABO major mismatch (60%), ABO minor mismatch (75%); only 2 patients had a double ABO mismatch donor (*P*=0.5).

Because today splenectomy is less likely to be performed, we wanted to combine donor type, CD34 cell dose, and age; we used ROC cut off values for age (62 years) and CD34 dose (8.41x10^6/kg). We identified 3 separate groups of patients with 0-1 (N=33), 2 (N=42), and 3 (N=18) negative predictors. The cumulative incidence of achieving a platelet count of 50x10^9/L was, respectively, 91%, 57%, and 44% (*P*=0.00007) (Figure 2A); 5-year actuarial survival in these 3 groups of patients was 89%, 65%, and 43%, respectively (*P*=0.003) (Figure 2B). NRM in the three groups was 3% (95%CI: 0.4-20%), 25% (95%CI: 14-42%), and 42% (95%CI: 23-73%) (Gray test *P*=0.003)

Long-term follow up

Platelet counts after HSCT are shown in Figure 3. Patients who failed to achieve a platelet count of 50x10^9/L within

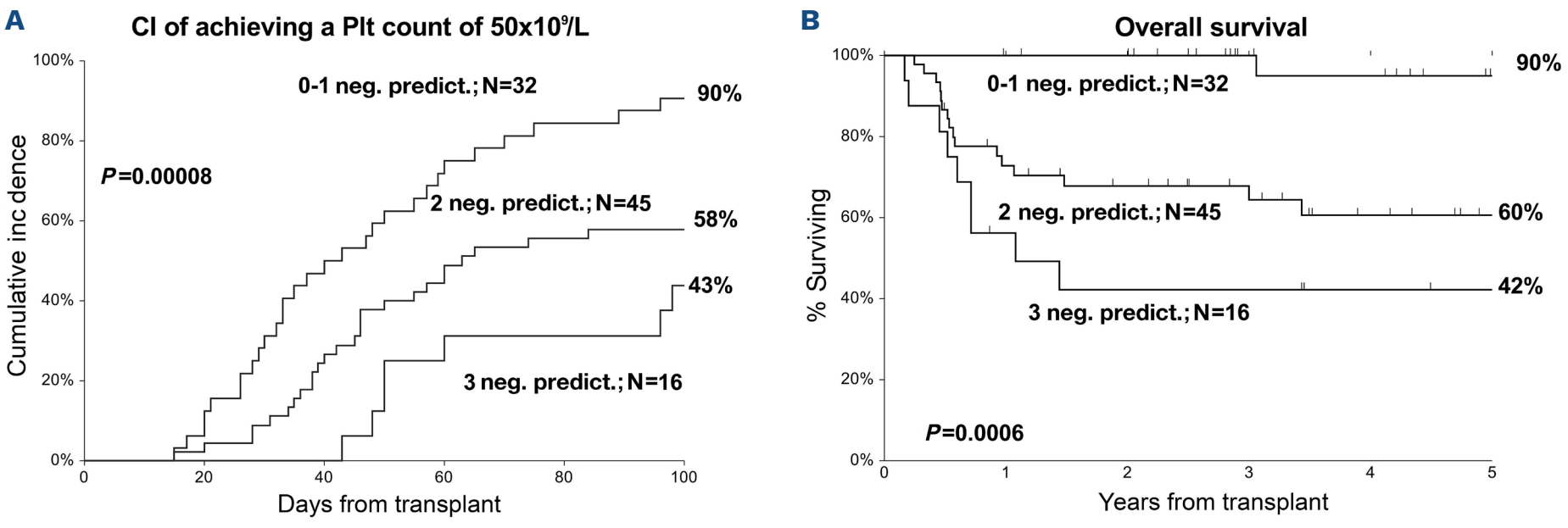

Figure 2. Platelet recovery and survival for the 3 groups of patients. (A) Platelet (Plt) recovery (50x10^9/L) within 100 days after transplant and (B) survival at five years stratified for 3 groups of patients according to 3 negative predictors (neg. predict.): CD34 cells infused x10^6/kg <8.41; age ≥63 years, and donor other than matched sibling donor (MSD).

Table 2. Univariate and multivariate Cox regression model on achieving a platelet count of 50x10^9/L: disease and transplant variables.

Variable	Value	Comp	Univariate		Multivariate		
			RR	*P*	RR	95% CI	*P*
Splenectomy	No	Yes	1.87	0.05	3.11	1.54- 6.26	0.004
Transfusions, N	<20	>20	0.69	0.21	-	-	-
Spleen, cm	<20	>20	0.98	0.94	-	-	-
DIPSS	<High	High	0.96	0.90	-	-	-
MTSS	Low	>Low	0.72	0.24	-	-	-
Fibrosis MF3	No	Yes	0.65	0.14	-	-	-
Comorbidity Index	<2	>2	0.68	0.14	-	-	-
Donor type	MSD	Alt.	0.44	0.002	0.42	0.23-0.74	0.003
Recipient age, years	Continuous		0.47	0.02	0.96	0.93-0.99	0.03
Conditioning	MA	RIC	0.96	0.90	-	-	-
CD34x10^6/kg	Continuous		1.85	0.02	1.11	1.01-1.22	0.02
PTCY	No	Yes	0.57	0.09	-	-	-

CD34: number of CD34 positive infused cells; Comp: compared value; DIPSS: Dynamic International Prognostic Scoring System; MF3: grading of marrow fibrosis in biopsy; MTSS: molecular transplant scoring system; PTCY: post-transplant cyclophosphamide; RR: relative risk; Donor type - Alt: alternative donor; MSD: matched sibling donor. Conditioning - MA: myeloablative; RIC: reduced intensity conditioning.

day +100 post transplant (Group B) show significantly lower platelet counts up to four years after HSCT than patients with robust early platelet recovery (Group A).

Chimerism and platelet recovery

A strong platelet recovery was achieved in 66% of patients with full donor chimerism (N=75) and in 66% of patients with mixed donor chimerism (N=18).

Causes of death

The primary cause of death for patients achieving a platelet count of 50x10^9/L *versus* patients not achieving this are as follows: relapse (N=6 *vs*. 3), GvHD (N=1 *vs*. 6), graft failure (N=0 *vs*. 1), cardiac toxicity (N=0 *vs*. 1), infection (N=2 *vs*. 4), multiorgan toxicity (N=1 *vs*. 5) (*P*=0.004).

Discussion

We have shown in this study that patients with MF achieving a platelet count of 50x10^9/L within day +100 following an allogeneic transplant have a significantly lower risk of NRM (*P*=0.00009) and significantly improved survival (*P*<0.00001), whereas relapse was unaffected. A more conventional end point of 20x10^9/L platelets within day +30 or within day +50 was less predictive of NRM. The same platelet count (50x10^9/L) was identified in a study by the Minnesota group that looked at predictors of delayed recovery and NRM.[1] In a group of 850 patients with malignant and non-malignant disease, failure to achieve a platelet count of 50x10^9/L within day 60 after HSCT was identified as delayed platelet recovery (DPR).[1] Platelet recovery was seen in 40% cord blood grafts, 57% in unrelated donor (URD), and 74% in MSD transplants. In this study, we have selected only patients with MF and chose 50x10^9/L platelets as a strong marker of robust hematologic recovery.

The primary end point of this study was to identify predictors of platelet recovery. In univariate and multivariate analysis, a transplant from an HLA identical sibling was the most significant positive predictor, followed by a higher number of CD34 cells infused, splenectomy, and younger age. There was a trend for better recovery in patients with fibrosis less than MF3. There was no effect of driver mutations, DIPSS, MTSS, conditioning regimen, pre-transplant ruxolitinib, or GvHD prophylaxis with PTCY. As to donor type, our finding confirms that MSD is the best donor for MF patients. In a prospective study, there was a very strong impact of donor type on survival:[9] NRM was 22% for matched siblings and 59% for alternative donors, and engraftment was 97% *versus* 76%, respectively. The actuarial 5-year survival was 35% for alternative donors.[9] Similar outcome was shown in another study, with 31% 5-year survival for alternative donor grafts.[10] On the other hand, in patients with acute leukemia, the outcome of transplants from MSD and alternative donors appears to be quite comparable.[11,12] This difference between acute leukemia and MF may be due to the inflammatory nature of MF which is aggravated after an allogeneic transplant, and calls, indeed, for improved GvHD control. A recent study with peri-transplant ruxolitinib shows that this appears to have reduced GvHD and improved outcome.[13] The use of a CD34 selected stem cell graft also seems to achieve this and reduce inflammation, and excellent results have been reported, with 3-year survival of 88%.[14]

The second strong predictor of early platelet engraftment is infusion of a high number of CD34 cells. This has already been identified as a positive indicator of outcome in an EBMT study,[15] with a significant impact on survival, relapse-free survival, and NRM. A word of caution about using high-dose CD34 cells: this may result in a high incidence of GvHD and may call for increased GvHD prophylaxis.[16]

The third predictor of a fast and strong platelet engraftment in our study was splenectomy, again in keeping with registry-based studies.[17] Patients grafted after splenectomy had significantly reduced NRM, athough there was an increased

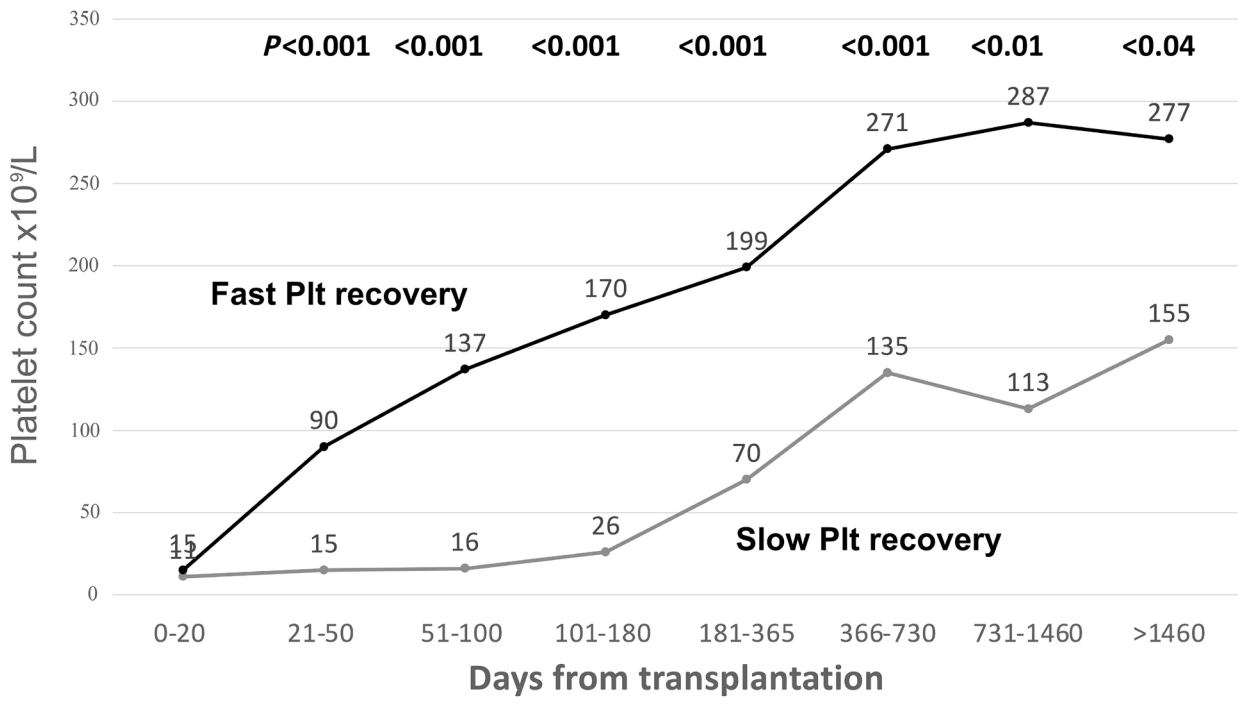

Figure 3. Median platelet counts of the two groups. Median platelet (Plt) counts of the two groups with Fast or Slow Plt recovery within day +100 post transplant. The latter group exhibits significantly lower Plt counts long term, beyond five years after transplantation.

incidence of relapse, resulting in comparable survival with non-splenectomized patients.[18] Other measures to reduce spleen size, and, therefore, spleen pooling, would be the new JAK inhibitors[19] and splenic irradiation.[20]

In addition, in our series, older age was associated with a significantly lower cumulative incidence of strong platelet recovery, both in univariate and multivariate analysis. Again, older age is a well-known predictor of outcome in patients undergoing allogeneic HSCT, so this finding in MF comes as no surprise.

We then wanted to further predict the incidence of strong platelet recovery, and decided to exclude splenectomy, since today it is rarely performed; in fact, it has not been used in our unit for the past four years. We restricted this model to donor type, CD34 cell dose (cut off $8.41x10^6$/kg), and patient age (cut off 62 years). Patients with 0-1, 2, or 3 negative predictors had a cumulative incidence of strong platelet recovery, respectively, of 90%, 58%, and 43% (P=0.00008), and an actuarial 5-year survival of 90%, 60%, and 42% (P=0.003). Therefore, it is possible to identify patients who will be at high risk of poor graft function and NRM at the time of transplant. We have also tested the MTSS score,[21] which predicted survival but, significantly, not platelet recovery.

Finally, patients who fail to achieve a platelet count of $50x10^9$/L within day 100 post transplant continue to have significantly lower counts up to four years post transplant and beyond. A search of the literature failed to find any reports on long-term hematologic recovery in MF patients following an allogeneic HSCT. We show in this study that moderate/severe chronic GvHD was seen in 46% of patients with long-lasting low platelet counts, despite the fact that the majority of them (over 90%) had received post-transplant cyclophosphamide for GvHD prophylaxis. Indeed, GvHD is associated with a reduced stem cell pool and poor peripheral blood cell counts. In a detailed study on 126 allografts, patients with GvHD had significantly lower peripheral blood counts, granulocyte macrophage colony forming units (CFU-GM), and erythroid burst forming units (BFU-E) compared to patients without GvHD,[22] all possibly due to inflammatory cytokines. These results would support transplant platforms aimed at reducing GvHD, such as the use of CD34 selected cells[14] or peri-transplant rituximab.[13]

The limitations of this study are its retrospective nature and the relatively small number of patients. Nevertheless, the identification of predictors for strong platelet recovery in MF, and therefore of NRM, may be useful when selecting patients and CD34 cell dose. A patient over the age of 62 with an alternative donor will have a risk of poor graft function of 43% at day 100, and an NRM of 25%; a high dose of CD34 cells may well be required in these patients. On the other hand, a young patient (<62 years) with an MSD will have a very low NRM and a high probability of fast platelet recovery; in these patients, the CD34 cell dose may not be crucially important. We have also shown that, in the long term, low platelet counts combined with significant GvHD suggest that PTCY may not be sufficient GvHD prophylaxis for MF patients undergoing an alternative donor graft. These data would support trials looking at a high CD34 cell dose[14] combined with peri-transplant rituximab[13] in patients undergoing alternative donor grafts.

Disclosure

No conflicts of interest.

Contributions

FS and AB wrote the paper; SG, EM and MAL contributed to clinical data collection; FF and EG performed data analysis; SS and PC supervised the study.

Funding

This study was supported by the Associazione Italiana Ricerca contro il Cancro (AIRC) Milano, grant 2017 (to AB).

Data-sharing statement

The data are available upon request to the corresponding author.

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