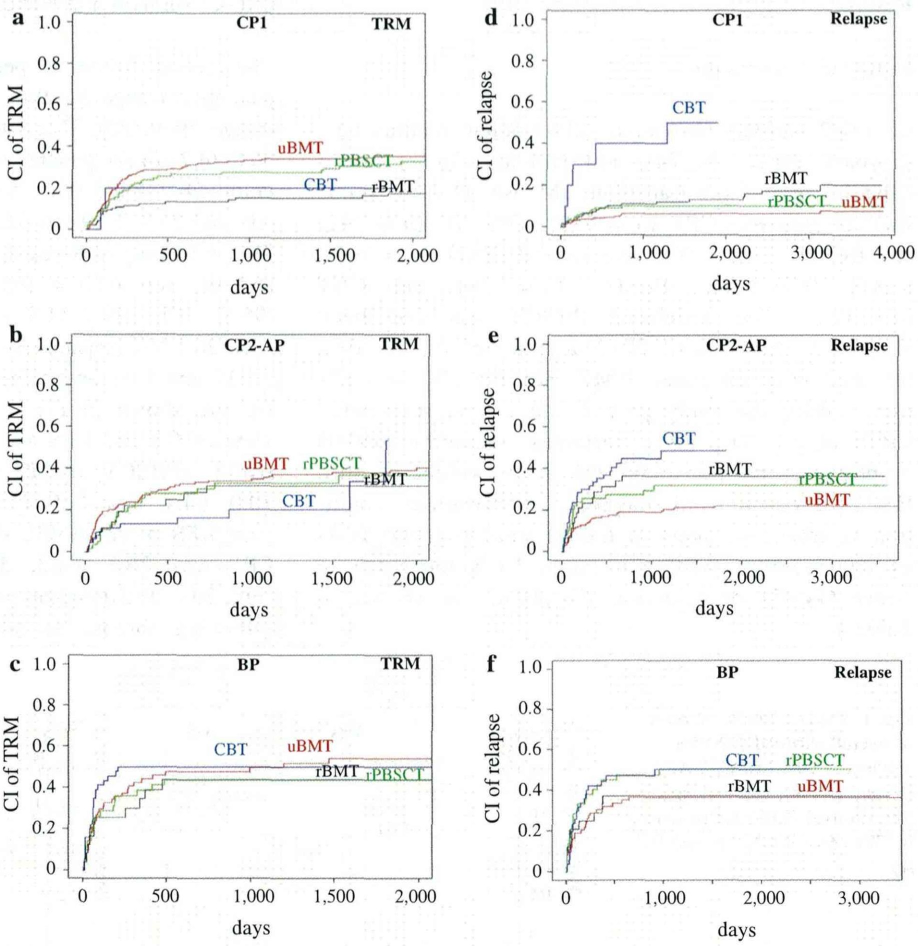
Fig. 2 The cumulative incidence of transplantation-related mortality (TRM) for patients in CP1 (**a**), CP2-AP (**b**) and BC (**c**); and relapse for patients in CP1 (**d**), CP2-AP (**e**) and BC (**f**)



patients in CP1: older age (>median age, 40 years: HR 1.67, 95 % CI, 1.15–2.41, $P = 0.007$), ABO mismatch (HR 1.44, 95 % CI, 1.003–2.06, $P = 0.048$) (Table 2), and uBMT (RR 6.01, 95 % CI, 1.20–29.97, $P = 0.029$) (Table 3). In CP2-AP, older age (> median age, 43 years: HR 1.74, 95 % CI, 1.25–2.43, $P < 0.001$) was the only factor an adverse prognostic factor (Table 2). In BC, pre-transplant IM (HR 0.61, 95 % CI, 0.49–0.89, $P = 0.011$) was the only factor for better OS (Table 2). Concerning LFS, multivariate analysis showed that uBMT (RR 4.26, 95 % CI, 1.24–14.62, $P = 0.021$) and older age (>median age, 40 years: HR 1.43, 95 % CI, 1.02–1.99, $P = 0.038$) were adverse risk factors in CP1 (Table 2, 3). For patients in CP2-AP and BC, no significant factor for OS or LFS was found. Thus, for patients in CP1, GS could have a significant impact on survival outcomes. While, for patients in the advanced phase of CML of beyond CP1, GS could have no significant impact on OS or LFS (Table 3).

TRM and relapse

The 1-year cumulative TRM rate by disease stage was 23.1 % (95 % CI, 19.5–26.7 %) in CP1, 24.2 % (95 % CI, 19.5–28.9 %) in CP2-AP, and 43.2 % (95 % CI, 35.9–50.5 %) in BC. TRM by GS is shown in Fig. 2a–c. The TRM rate appeared low in rBMT compared with uBMT or rPBSCT in CP1 (Fig. 2a). Multivariate analysis showed that uBMT (RR 2.49, 95 % CI 1.02–6.10, $P = 0.046$) and older age (>median age, 40 years: HR 1.69, 95 % CI, 1.19–2.39, $P = 0.003$) were factors associated with a significantly increased risk of TRM in CP1 (Table 2, 3).

The 3-year cumulative relapse rate by disease stage was 9.0 % (95 % CI, 3.9–7.9 %) in CP1, 28.2 % (95 % CI, 23.3–33.1 %) in CP2-AP, and 43.6 % (95 % CI, 36.3–50.9 %) in BC. Relapse rate by GS is demonstrated in Fig. 2d–f. For patients in CP1, the relapse rate after CBT appeared to be higher than that after other GS (Fig. 2d). In multivariate analysis by the effect of GS in CP1, CBT (RR

Table 2 Multivariate analysis of risk factors for the main outcomes after allo-HSCT for CML in CP1, CP2-AP, and BP

Main outcomes	Factors	CP1				CP2-AP				BP			
		Factors	HR	(95 % CI)	*P* value	Factors	HR	(95 % CI)	*P* value	Factors	HR	(95 % CI)	*P* value
OS	Age	≤40	1			≤43	1						
		>40	1.67	1.15–2.41	0.007	>43	1.74	1.25–2.43	< 0.001				
	ABO mismatch	No	1										
		Yes	1.44	1.003–2.06	0.048								
	Pre-transplant IM									No	1		
										Yes	0.61	0.41–0.89	0.011
LFS	Age	≤40	1										
		>40	1.43	1.02–1.99	0.038								
TRM	Age	≤40	1										
		>40	1.69	1.19–2.39	0.003								
Relapse	HLA mismatch (rejection)									0, 1	1		
										≥2	1.7	1.04–2.76	0.033
	HLA mismatch (GVHD)					0, 1	1						
						≥2	3.57	1.55–8.21	0.003				
Acute GVHD (all grades[a])	Pre-transplant IM	No	1										
		Yes	0.75	0.57–0.99	0.04								
	BW					≤60 kg	1						
						>60 kg	1.35	1.01–1.82	0.045				
Acute GVHD	BW					≤60 kg	1						
(≥grade 2)						> 60 kg	1.53	1.05–2.24	0.028				
Chronic GVHD (extensive[b])	BW					≤60 kg	1						
						>60 kg	1.75	1.06–2.73	0.028	0			

OS overall survival, *LFS* leukemia-free survival, *TRM* transplantation-related mortality, *ANC* absolute neutrophil count, *GVHD* graft-versus-host disease, *IM* imatinib, *HLA* human leukocyte antigen, *BW* body weight, *HR* hazard ratio, *CI* confidence interval, *CP* chronic phase, *AP* accelerated phase, *BP* blastic phase, *imatinib* imatinib mesylate

[a] Overall grade of acute GVHD assigned according to the Center for International Blood and Marrow Transplant Research (CIBMTR) severity index

[b] Chronic GVHD was graded as limited or extensive based on the Seattle criteria

Table 3 Impact of graft sources on main outcomes after allo-HSCT for CML in CP1, CP2-AP, and BP

Main outcomes	Graft sources	CP1			CP2-AP			BP		
		RR	(95 % CI)	*p* value	RR	(95 % CI)	*p* value	RR	(95 % CI)	*p* value
OS	rBMT	1.00			1.00			1.00		
	uBMT	6.01	(1.20–29.97)	0.029	1.12	(0.33–3.79)	0.851	>99	(0.00–99.99)	0.999
	rPBSCT	1.76	(0.77–4.04)	0.180	0.84	(0.21–3.43)	0.809	1.13	(0.56–2.30)	0.727
	CBT	1.00	(0.00–99.99)	1.000	NA	NA	NA	NA	NA	NA
LFS	rBMT	1.00			1.00			1.00		
	uBMT	4.26	(1.24–14.62)	0.021	1.61	(0.55–4.74)	0.383	0.00	(0–99.99)	0.999
	rPBSCT	1.72	(0.95–3.11)	0.073	0.42	(0.14–1.31)	0.135	0.67	(0.31–1.44)	0.299
	CBT	1.00	(0.00–99.99)	1.000	NA	NA	NA	NA	NA	NA
TRM	rBMT	1.00			1.00			1.00		
	uBMT	2.49	(1.02–6.10)	0.046	1.36	(0.60–3.09)	0.47	2.71	(0.74–9.96)	0.13
	rPBSCT	1.03	(0.52–2.07)	0.93	0.94	(0.52–1.70)	0.83	1.43	(0.64–3.22)	0.39
	CBT	0.33	(0.04–2.63)	0.29	0.98	(0.60–1.60)	0.94	1.26	(0.82–1.92)	0.29
Relapse	rBMT	1.00			1.00			1.00		
	uBMT	0.33	(0.12–0.95)	0.041	0.66	(0.29–1.55)	0.34	2.23	(0.28–17.61)	0.45
	rPBSCT	1.13	(0.62–2.07)	0.68	1.17	(0.64–2.14)	0.6	1.06	(0.44–2.54)	0.9
	CBT	25.16	(1.76–369.10)	0.018	1.15	(0.74–1.80)	0.53	0.77	(0.39–1.60)	0.49
ANC recovery	rBMT	1.00			1.00			1.00		
	uBMT	0.82	(0.55–1.23)	0.35	0.83	(0.53–1.31)	0.43	0.58	(0.27–1.26)	0.17
	rPBSCT	1.31	(1.02–1.69)	0.036	1.2	(0.90–1.59)	0.21	0.91	(0.33–2.52)	0.86
	CBT	2	(0.67-5.98)	0.22	0.53	(0.42–0.67)	<0.001	0.55	(0.37–0.82)	0.003
Platelet recovery	rBMT	1.00			1.00			1.00		
	uBMT	0.75	(0.46–1.21)	0.24	0.89	(0.51–1.56)	0.68	0.21	(0.07–0.61)	0.0039
	rPBSCT	0.93	(0.69–1.26)	0.65	0.91	(0.61–1.35)	0.63	0.67	(0.28–1.57)	0.35
	CBT	1.07	(0.35–3.28)	0.9	0.78	(0.62–0.99)	0.049	0.44	(0.26–0.74)	0.0018
Acute GVHD (all grades[a])	rBMT	1.00			1.00			1.00		
	uBMT	3.35	(1.50–6.22)	<0.001	1.67	(0.92–3.02)	0.09	1.22	(0.46–3.25)	0.69
	rPBSCT	1.49	(0.94–2.37)	0.091	0.86	(0.51–1.44)	0.56	0.94	(0.32–2.73)	0.91
	CBT	1.67	(0.68–4.11)	0.26	0.76	(0.58–1.01)	0.054	1.05	(0.56–1.96)	0.87
Acute GVHD (≥grade 2)	rBMT	1.00			1.00			1.00		
	uBMT	4.28	(1.92–9.53)	<0.001	2.14	(0.93–4.94)	0.075	1.34	(0.39–4.61)	0.65
	rPBSCT	1.5	(0.82–2.72)	0.19	1.53	(0.82–2.86)	0.18	2.23	(0.36–1.39)	0.39
	CBT	1.00	(0.00–99.99)	1.000	0.84	(0.58–1.22)	0.36	1.45	(0.55–3.81)	0.45
Chronic GVHD	rBMT	1.00			1.00			1.00		
	uBMT	0.95	(0.53–1.70)	0.86	1.1	(0.45–2.68)	0.84	0.27	(0.06–1.33)	0.11
	rPBSCT	1.37	(0.97–1.92)	0.075	1.24	(0.70–2.19)	0.47	0.84	(0.22–3.20)	0.8
	CBT	8.52	(0.64–11.43)	0.11	0.8	(0.52–1.25)	0.33	0.73	(0.32–1.66)	0.46
Chronic GVHD (extensive[b])	rBMT	1.00			1.00			1.00		
	uBMT	1	(0.49–2.04)	1	0.84	(0.33–2.15)	0.72	0.69	(0.14–3.46)	0.65
	rPBSCT	1.31	(0.87–1.96)	0.19	1.19	(0.60–2.34	0.62	1.08	(0.27–4.24)	0.92
	CBT	6.61	(0.22–200.8)	0.28	0.63	(0.36–1.09)	0.097	0.77	(0.31–1.88)	0.56

OS overall survival, *LFS* leukemia-free survival, *TRM* transplantation-related mortality, *ANC* absolute neutrophil count, *GVHD* graft-versus-host disease, *RR* relative risk, *CI* confidence interval, *CP* chronic phase, *AP* accelerated phase, *BP* blastic phase, *rBMT* related bone marrow transplantation, *rPBSCT* related peripheral blood stem cell transplantation, *uBMT* unrelated bone marrow transplantation, *CBT* unrelated cord blood transplantation, *NA* not available

[a] Overall grade of acute GVHD assigned according to the Center for International Blood and Marrow Transplant Research (CIBMTR) severity index

[b] Chronic GVHD was graded as limited or extensive based on the Seattle criteria

25.16, 95 % CI 1.76–369.10, $P = 0.018$) showed higher relapse, while uBMT (RR 0.33, 95 % CI 0.12–0.95, $P = 0.041$) was lower relapse compared with those in rBMT (Table 3).

Engraftment

The cumulative neutrophil recovery rate on day 90 was 97.5 % (95 % CI, 96.1–98.9 %) in CP1, 93.2 % (95 % CI, 90.5–95.9 %) in CP2-AP, and 82.3 % (95 % CI, 76.8–87.8 %) in BC. On day 180, the cumulative platelet recovery rate, as indicated by more than 2×10^{10}/L of platelets in blood, was 91.9 % (95 % CI, 89.5–94.3 %) in CP1, 85.1 % (95 % CI, 81.2–89.0 %) in CP2-AP, and 67.2 % (95 % CI, 60.3–74.1 %) in BC. Note that the neutrophil recovery and platelet recovery rates were lower after CBT, especially in patients in the advanced phase; i.e., neutrophil recovery in CBT: 90 % in CP1, 79.4 % in CP2-AP, and 64.0 % in BC; platelet recovery after CBT: 90.0 % in CP1, 72.5 % in CP2-AP, and 52.0 % in BC (Fig. 3a–f). Multivariate analysis showed that rPBSCT (RR 1.31, 95 % CI 1.02–1.69, $P = 0.0396$ was a significant factor for early neutrophil recovery in CP1. While, CBT (RR 0.53, 95 % CI 0.42–0.67, $P < 0.001$) was a significant factor for delayed neutrophil recovery in CP2-AP (Table 3). The factor statistically associated with delayed platelet recovery was CBT in CP2-AP (RR 0.78, 95 % CI 0.62–0.99, $P = 0.0049$) and in BC (RR 0.44, 95 % CI 0.26–0.74, $P = 0.0018$). Unrelated BMT (RR 0.21, 95 % CI 0.07–0.61, $P = 0.0039$) was also a significant factor for delayed platelet recovery in BC (Table 3).

Acute and chronic GVHD

The cumulative incidence of acute GVHD at all grades before day 100 was 62.8 % (95 % CI, 58.6–67.0 %) in CP1, 63.5 % (95 % CI, 58.2–58.8 %) in CP2-AP, and 68.6 % (95 % CI, 61.3–74.9 %) in BC. Patients who underwent uBMT showed a higher incidence of acute GVHD (all grades) in CP1 and CP2-AP (Fig. 4a, b). This association was confirmed by multivariate analysis; uBMT (RR 3.35, 95 % CI 1.50–6.22, $P < 0.001$) was a significant factor in CP1 (Table 3). Pre-transplant IM (HR 0.75, 95 % CI 0.57–0.99, $P = 0.04$) was a significant risk factor for acute GVHD (all grades) in CP1 (Table 2). Focusing exclusively on grade II or higher acute GVHD, uBMT (RR 4.28, 95 % CI 1.92–9.53, $P < 0.001$) (Table 3) was a significant risk factor in CP1 (Table 2). For patients in CP2-AP, body weight (>60 kg) was a factor significantly associated with increased risk of aGVHD (all grade; RR 1.35, 95 % CI, 1.01–1.82, $P = 0.045$, grade II or higher grade; RR 1.53, 95 % CI, 1.05–2.24, $P = 0.028$) (Table 2).

The cumulative incidence of chronic GVHD among evaluable patients who survived at least 100 days after allo-HSCT was 49.4 % (95 % CI, 44.7–54.1 %) in CP1, 42.2 % (95 % CI, 36.4–48.0 %) in CP2-AP, and 37.8 % (95 %CI, 30.0–45.6 %) in BC. For patients in CP1, rPBSCT showed a higher incidence of chronic GVHD (71.4 %), which was compared to other GS (Fig. 4d); however, this significant association was not confirmed in multivariate analysis (rPBSCT: RR 1.37 95 % CI 0.97–1.92, $P = 0.075$). For patients in CP2-AP and BC, chronic GVHD after CBT occurred at rates of 23.1 and 23.8 %, respectively, which were apparently lower than that of other GS (Fig. 4e, f), but these statistical associations were not also confirmed by multivariate analysis in CP2-AP or BC (Table 3). Concerning extensive chronic GVHD, multivariate analysis showed the significant association between body weight (>60 kg; RR 1.75, 95 % CI, 1.06–2.73, $P = 0.028$) and chronic GVHD in CP2-AP (Table 2).

Discussion

Our study reviewed 1,062 Japanese adult patients who underwent allo-HSCT during the past decade (2000–2009); thus, our cohort reflects the current use and results of allo-HSCT for CML in Japan. Moreover, the TRUMP database offers the advantage of a large number of patients with extensive data, which permits multivariate analysis. The 3-year OS was 70.6 % for patients in CP1, and the probability of 3-year LFS for patients in CP1 was 64.6 %. These survival data for patients in CP1 were comparable to those reported by others [12]. Based on the report from the EBMT, which included 13,416 CML patients and was apparently the largest CML transplant database including the 3 times cohorts (i.e., 1980–1990, 1991–1999, 2000–2003), the probability of OS at 2 years for patients transplanted in CP1 from an HLA-identical sibling was 74 %, with a cumulative incidence of TRM at 2 years of 22 % and of relapse of 18 % among the most recent cohort transplanted between 2000 and 2003 ($n = 3{,}018$) [13]. The Center for International Blood and Marrow Transplant Research (CIBMTR) recently reported the transplant outcomes of 449 patients with advanced phase CML; the disease-free survival rates remained as low as 35–40 % for CP2, 26–7 % for AP, and 8–11 % for BC [14]. Our series including 432 cases of CP2-AP and 189 cases of BC showed similar survival rates, as the probabilities of 3-year LFS in CP2-AP and BC were 46.1 and 19.2 %, respectively.

Our primary object in this study was to assess the clinical impact of GS according to each disease status. Our study results revealed that the patients in CP1 who were

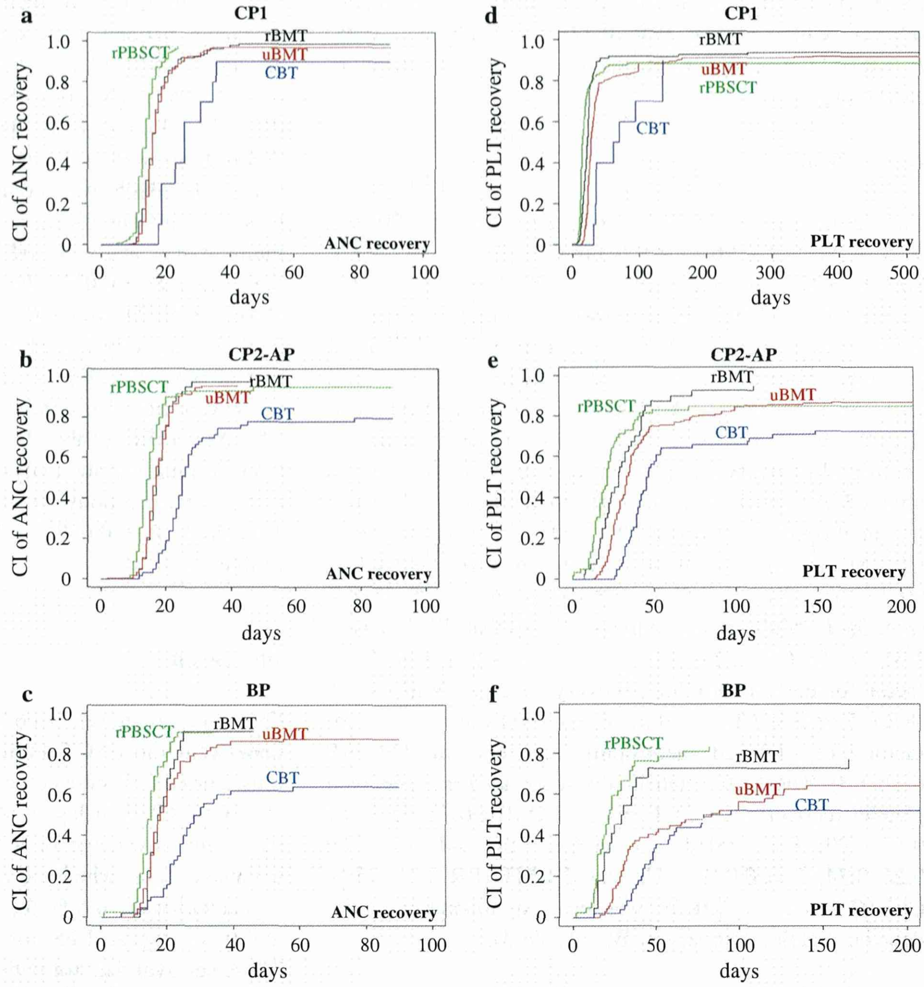


Fig. 3 The cumulative incidence of absolute neutrophil count (ANC) recovery for patients in CP1 (**a**), CP2-AP (**b**) and BC (**c**); and platelet (PLT) recovery for patients in CP1 (**d**), CP2-AP (**e**) and BC (**f**)

treated by rBMT showed a better 3-year OS (84.4 %) with a lower 1-year cumulative incidence of TRM, but the 3-year LFS and relapse rates were similar between patients receiving rBMT and patients receiving rPBSCT. These data were essentially in line with previous reports in which the CIBMTR reported the data of CML patients undergoing rPBSCT or rBMT in CP1; the 1-year LFS and relapse rates were similar for patients receiving rBMT or rPBSCT [14]. We also assessed the clinical impact of GS in CP2-AP; our results showed that there were no significant differences in OS or LFS between GS, despite lower probabilities of relapse after uBMT and lower probabilities of TRM after CBT. These results differ from the IBMTR reports in that for patients in CP2 or AP, rPBSCT was associated with a lower incidence of treatment failure and a higher probability of LFS at 1 year [15]. Regarding GVHD, a recent prospective randomized trial showed a trend toward a higher incidence of chronic GVHD after rPBSCT (59 % after rPBSCT vs. 40 % after rBMT, $P = 0.11$) for patients in CP1 [16]. Our results may confirm this report; although multivariate analysis in our study showed that rPBSCT (RR 1.37 95 % CI 0.97–1.92, $P = 0.075$) was not a significant risk factor for developing chronic GVHD (Table 3), rPBSCT showed a higher incidence of chronic GVHD (71.4 %), which was compared to other GS in CP1 (Fig. 4d).

Several investigators have addressed the clinical impact of pre-transplant IM on post-transplant outcomes for CML [14, 17–20]. The CIBMTR data demonstrated that pre-transplant IM was associated with better survival, but revealed no statistically significant differences in TRM, relapse, and LFS for patients in CP1 [17]. Among patients transplanted in the more advanced phases beyond CP1, pre-transplant IM was not associated with TRM, relapse, LFS, OS, or acute GVHD [17]. In contrast to these studies, our analysis showed that pre-transplant IM was significantly associated with better OS for patients in BC. In addition, multivariate analysis found pre-transplant IM was a

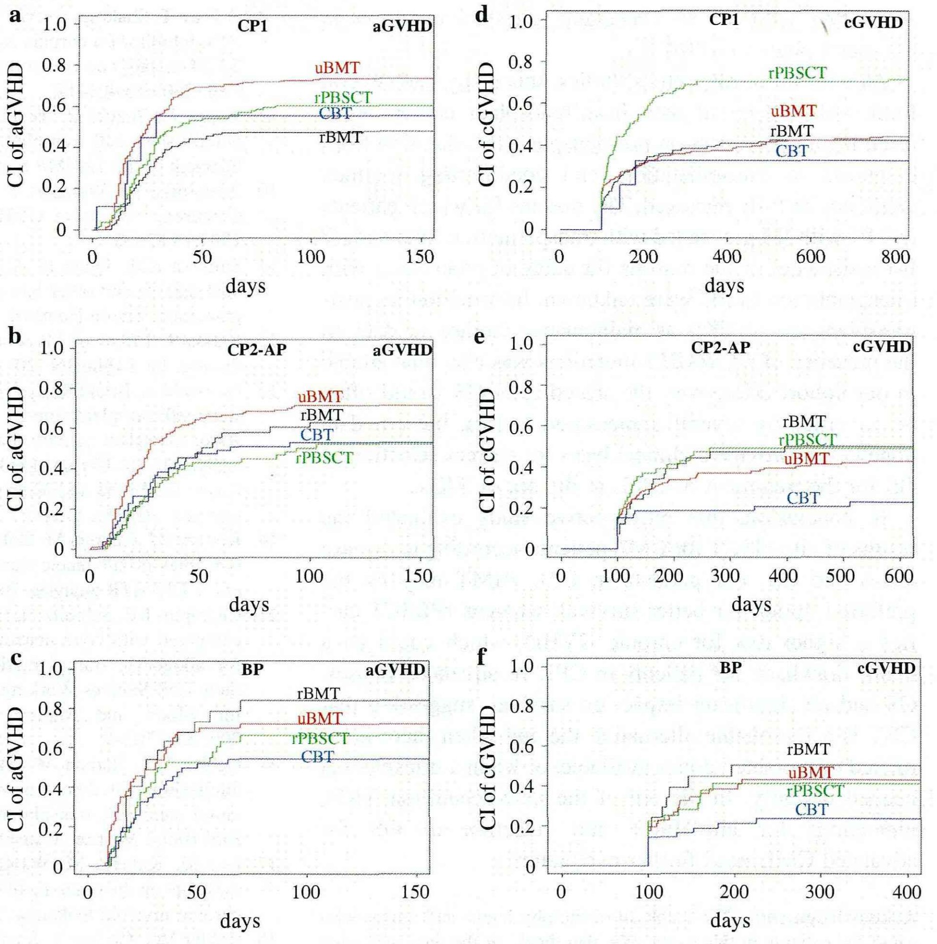


Fig. 4 The cumulative incidence of acute GVHD at all grades for patients in CP1 (**a**), CP2-AP (**b**) and BC (**c**); and chronic GVHD at all grades for patients in CP1 (**d**), CP2-AP (**e**) and BC (**f**)

significant factor associated with acute GVHD (>grade II) in CP1 (data not shown). Despite the study in the era of TKI, half of patients were in CP1, and 61 % of patients underwent allo-HSCT without use of pre-transplant TKI in this study. We should interpret these findings with utmost caution. We assume that most patients had already initiated the conventional treatment but could not reach a new, but expensive IM treatment before allo-HSCT, as a reason for these findings. Moreover, the findings that the number of patients in CP1 underwent allo-HSCT was 447 in the early period of IM from 2000 to 2004 and only 84 from 2005 to 2009 might support our assumption. Deininger et al. reported an effect of pre-transplant IM in their study that included 70 cases of CML and 21 cases of Ph (+) acute lymphoid leukemia. These investigators compared the outcomes with historical controls identified in the EBMT database [21], and observed a trend towards higher relapse mortality and significantly less chronic GVHD in patients with pre-transplant IM (OR = 0.44, $P = 0.027$). Thus, the clinical impact of pre-transplant IM is still a contentious issue; additional studies evaluating the long-term use of IM with a larger number of patients might permit a more refined analysis of the effect of pre-transplant IM.

Although data on clinical outcomes after CBT are conflicting, CBT has apparent advantages over uBMT, including no risk to the donor and ease of availability. Previous reports, mostly from pediatric studies, have shown that, despite higher HLA mismatch, CBT carries a lower risk of acute GVHD and chronic GVHD in comparison with uBMT [22–24]. A recent Japanese retrospective analysis assessing 86 patients, including pediatric patients, disclosed the transplant outcomes of CBT: 2-year OS was 53 %; for patients in CP, AP and BC, the OS rates were 71, 59 and 32 %, respectively [25]. Although our small population with only 10 cases of CBT in CP1 may prohibit drawing meaningful conclusions, a trend of higher relapse and lower TRM, OS and LFS in CP1 was similar to results obtained by previous study groups. Nevertheless, in CP2-AP and BC, transplant outcomes after CBT were comparable to those of other GS, suggesting CBT as an acceptable alternative option in advanced phases of CML.

As with all retrospective studies, this study had several limitations. Reported data from transplant centers were often incomplete: data on pre-transplant IM, duration from diagnosis to transplantation, and conditioning regimen could not be fully retrieved. The reasons for which patients in CP1 with IM proceeded with transplantation (planned, or IM resistance) or the reasons for delay in proceeding with transplantation in BC were unknown. Information on post-transplant use of TKIs as maintenance therapy or data on the presence of *BCR/ABL1* mutations was also unavailable in our cohort. Moreover, the selection of GS would often be governed by several unmeasured factors, but our data nonetheless provide a clinical basis for current selection of GS for the treatment of CML in the era of TKIs.

In conclusion, this retrospective study evaluated the results of allo-HSCT for CML patients according to disease status and GS. For patients in CP1, rBMT may be the preferred option for better survival, whereas rPBSCT carries a higher risk for chronic GVHD, which could be a major drawback for patients in CP1. In advanced phases, GS had no significant impact on survival, suggesting that CBT is a reasonable alternative therapy when there is no related or unrelated donor available, or when a transplant is needed urgently. In the era of the new-generation TKIs, indications for allo-HSCT and selection of GS for advanced CML need further evaluation.

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Conflict of interest The authors declare no conflict of interest.

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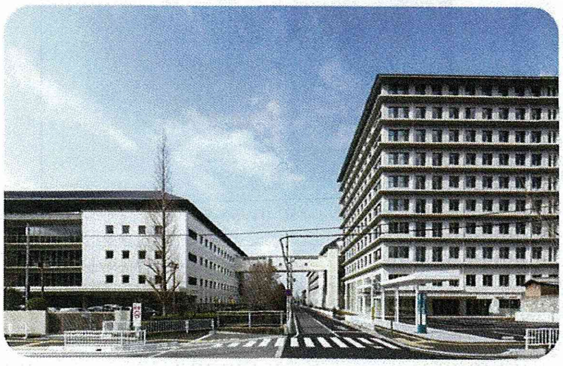

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