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LETTER TO THE EDITOR

Haemostatic changes and outcomes in transcatheter aortic valve implantation

Introduction

The management of aortic stenosis (AS) has undergone a paradigm shift with the advent of transcatheter aortic valve implantation (TAVI). Inintially established as an effective alternative for high-risk patients, TAVI is now a class IA recommendation for patients aged 75 or over, or in those at increased risk or unsuitable for surgery [1]. Despite being less invasive than surgery, TAVI can also lead to complications, including haemorrhagic and thromboembolic events [2–4]. These complications are further compounded by the coagulation abnormalities associated with AS, particularly acquired Von Willebrand syndrome [5]. Additionaly, patients undergoing TAVI often present with hematological disorders, influenced by their advanced age and comorbidities. Pre-procedural anaemia affects 45 to 64% of TAVI candidates, while thrombocytopenia is observed in approximately 40% [6].

Although changes in haemogram and hemostasis profiles following TAVI have been reported, their prognostic significance remains incompletely understood [6–10]. A deeper understanding of these phenomena is essential to enable personalised antiplatelet and anticoagulation strategies before and after TAVI, as well as to improve individual risk stratification. This single-centre retrospective study evaluated the evolution of adenosine diphosphate-induced platelet reactivity (CT ADP) and haemogram parameters before and after TAVI, investigating their potential correlation with mortality and complications.

Methods

Study population

This study concerned 117 consecutive patients with symptomatic severe AS who underwent a TAVI procedure with a self-expandable valve (CoreValve EvolutR®, Medtronic Inc., Minneapolis, Minnesota, USA) at the University of Liège Hospital. Data were collected from the patient hospital records or *via* the local health network (Walloon health network). The decision to perform TAVI was made through a consensus by the institutional heart team, ensuring a multidisciplinary approach tailored to each patient's clinical profile. The study adhered to the principles outlined in the Declaration of Helsinki and received approval from the Ethics Committee of Liege University Hospital (protocol code: 2021/306, date of approval: 12 October 2021).

TAVI procedure

Most TAVI procedures were completed through transfemoral access under locoregional anaesthesia combined with sedation. For selected patients, alternative access routes, including subclavian or carotid arteries, were utilised, with some cases requiring surgical preparation of the vessel. All patients received either Medtronic Corevalve^{MC} Evolut R or Evolut Pro prostheses, with valve sizes 23 to 34 mm determined by the heart team based on detailed preoperative cardiac computed tomography (CT) analysis. Procedural success was assessed by ensuring correct prosthesis positioning and the absence of complications. Comprehensive documentation of potential periprocedural complications including atrial fibrillation (AF), high-grade conduction disorders, ischaemic or hemorragic stroke, vascular access complication or death, was provided in the interventional cardiologist's report. In cases of incomplete prosthesis deployment or significant aortic insufficiency, additional balloon dilatation was performed to optimise outcomes.

A loading dose of aspirin (250 to 500 mg) was administered intravenously to all patients not on chronic aspirin therapy. A loading dose of clopidogrel (300 to 600 mg) was administered in all patients not receiving chronic clopidogrel therapy. Following vascular access preparation, an intravenous dose of unfractionated heparin ranging (5,000 to 7,500 units targeting 100 units/Kg) was administered to achieve an activated clotting time (ACT) between 250 and 300 s. Protamine was used for heparin reversal at the end of the procedure in most patients. Intra-procedural closure time with CT-ADP was measured using the INNOVANCE PFA-200 system (Siemens) at three time points: prior to prosthesis deployment (t1) and 15 min post-deployment (t2).

Statistical analysis

Variables are presented as numbers with percentages or as means with standard deviations. Changes in biomarkers across the follow-up period were analysed using the paired Student's t-test. Survival analysis was performed to evaluate the predictive value of clinical and procedural variables for mortality and complications, using hazard ratios (HR) with corresponding 95% confidence intervals (CI) to quantify the risk. Additional subgroup analyses were performed when appropriate to explore variations in outcomes based on key patient characteristics. All tests were two-sided, and a p-value ≤ 0.05 was considered statistically significant. All statistical analyses were carried out using SAS version 9.4 (SAS Institute, Cary, NC).

Results

Baseline characteristics of the study population

The baseline characteristics of the study population are summarised in Table 1. A total of 117 patients were included, with a mean age of 81.9 ± 6.58 years and 57% were male. The mean indexed aortic valvular area was 0.44 ± 0.11 cm^2/m^2 and the mean aortic peak velocity (Vmax) was 4.04 ± 0.73 m/s. Regarding antithrombotic therapies, 46.55% of patients were on single antiplatelet therapy (SAPT), 17% on dual antiplatelet therapy (DAPT), 11.11% on antivitamin K (AVK) and 27.35% on non-vitamin K oral anticoagulants (NOAC). The predominant procedural approach was transfemoral, performed in 83.76% of cases.

Outcomes after TAVI

Outcomes after TAVI are summarised in Table 2. During the hospital stay after TAVI (mean duration 10.8 ± 7.2 days), 8 (7.02%) patients died, including 6 (5.22%) from cardiovascular causes. Cardiac decompensation was observed in 13 (11.6%) patients, most of which pre-existed the procedure. Seven (6.25%) patients experienced an ischaemic stroke and 7 (5.98%) suffered from vascular dissections related to the procedure. Bleeding events occurred in 20 patients (17.09%) and were procedure related in most cases ($n = 17$). Two bleeding were classified as BARC 3a (hemorrhages with haemoglobin loss >3 g/dL requiring transfusion, vasoactive treatment, or surgery) with remaining events considered as minor bleedings (BARC 1 or 2) [11]. Furthermore, 15 (13.27%) patients required implantation of a permanent pacemaker and 18 (15.79%) developed acute renal failure, the majority of which were classified as stage 1 according to the AKIN classification.

Mean follow-up was 2.16 ± 0.64 years. At 3 months, 2 (1.87%) patients died of non-cardiovascular causes, 5 (4.27%) experienced cardiac decompensation requiring hospitalisation, 1 (0.85%) had an ischaemic stroke, and 5 (4.72%) had significant bleeding. At 1 year, 9 (8.33%) patients died, 12 (10.25%) experienced cardiac decompensation requiring hospitalisation, 6 (5.61%) suffered ischaemic strokes, and 9 (8.41%) had significant bleeding.

Changes in blood count and CT-ADP

The changes in blood count parameters and CT-ADP are illustrated in Figures 1–5. There was a significant increase in the white blood cell count (WBC) between T1 and T2 ($p = 0.0001$), between T2 and T3 ($p < 0.0001$) and between T1 and T3 ($p < 0.0001$). Haemoglobin levels showed a significant decrease between T1 and T2 ($p < 0.0001$). This was

Table 1. Baseline characteristics of the study population.

Demographics		Transthoracic echocardiography	
Age (years)	81.9 ± 6.58	Left ventricular ejection fraction (%)	55.95 ± 11.72
Male sex (%)	57 (48.72)	Aortic peak velocity (m/s)	4.04 ± 0.73
Body Mass Index (kg/m^2)	2719 ± 5.6	Transaortic mean gradient (mmHg)	41.77 ± 14.94
Euroscore II (%)	5.96 ± 5.29	Aortic valvular area (cm^2)	0.78 ± 0.18
STS-score (%)	4.83 ± 2.74	Indexed aortic valvular area (cm^2/m^2)	0.44 ± 0.11
Cariovascular risk factors			
Active smoker (%)	35 (30.17)	Permeability index	0.21 ± 0.05
Diabetes (%)	40 (34.19)	Aortic regurgitation (%)	47 (43.12)
Dyslipidemia (%)	90 (76.92)	Estimated systolic PAP (mmHg)	45.95 ± 16.13
Hypertension (%)	105 (89.74)	**Procedure**	
Comorbidities and medical history		Aspirin load (%)	29 (24.79)
Chronic obstructive pulmonary disease (%)	20 (17.09)	Clopidogrel load (%)	27 (23.08)
Atrial fibrillation (%)	48 (41.43)	HNF (%)	108 (94.74)
Pacemaker (%)	24 (20.51)	Protamine (%)	89 (78.07)
Peripheral arteriopathy (%)	47 (40.17)	General anaesthesia (%)	71 (60.68)
Coronary artery disease (at least one stenosis of >50%) (%)	45 (38.46)	Loco-regional anaesthesia + sedation (%)	46 (39.32)
Reduced mobility (%)	23 (20)	Approach (%)	
Stroke (%)	15 (12.82)	Transfemoral	98 (83.76)
Cardiac surgery (%)	39 (33.33)	Subclavian	16 (13.68)
Balloon aortic valvuloplasty (%)	10 (8.55)	Carotid	3 (2.56)
Medical treatment		Left	39 (33.33)
Beta-blocker (%)	86 (73.50)	Right	78 (66.67)
Angiotensin-Converting Enzyme Inhibitors (%)	39 (33.33)	Corevalve Evolut-R (%)	27 (23.08)
Angiotensin II Receptor Blockers (%)	31 (26.50)	Corevalve Evolut-Pro (%)	90 (76.92)
Mineralocorticoid Receptor Antagonists (%)	19 (16.24)	Valve size (mm)	
Single Antiplatelet Therapy (%)	54 (46.55)	23–26 (%)	33 (28.21)
Dual Antiplatelet Therapy (%)	20 (17.09)	28–29 (%)	46 (39.32)
Antivitamin K Anticoagulants (%)	13 (11.11)	34 (%)	38 (32.48)
Non-vitamin K Oral Anticoagulants (%)	32 (27.35)	Post-ballon dilatation (%)	11 (9.65)
Statine (%)	79 (67.52)	**Computed Tomography**	
Blood tests		Calcium score	2753.24 ± 1544.28
eGFR (Cockroft) (ml/min/1.73 m^2)	50.11 ± 23.38	Aortic annulus (mm)	24.96 ± 2.56
B-type Natriuretic Peptide (pg-mL)	405.12 ± 434.43	Aortic valvular area (mm^2)	482.15 ± 113.86
HbA1c (%)	5.89 ± 0.94		
Hemoglobine (g/dL)	11.19 ± 1.61		
Haematocrit (%)	35.53 ± 5.05		
Platelets (1000/µl)	219.36 ± 69.6		
White Blood Cells (1000/µl)	6.29 ± 2.52		

Table 2. Outcomes in hospital, at 3 months and at 1 year after TAVI.

	In hospital	3 months	1 year
Death	8 (7.02%)	2 (1.87%)	9 (8.33%)
Cardiovascular death	6 (5.22%)	0 (0.00%)	0 (0.00%)
Cardiac decompensation	13 (11.61%)	5 (4.27%)	12 (10.25%)
Ischaemic Stroke	7 (6.25%)	1 (0.08%)	6 (5.61%)
Bleeding	20 (17.09%)	5 (4.72%)	9 (8.41%)
Artery dissection	7 (5.98%)	0 (0.00%)	0 (0.00%)
Permanent pacemaker implantation	15 (13.27%)	0 (0.00%)	0 (0.00%)
Acute kidney failure	18 (15.79%)	0 (0.00%)	0 (0.00%)

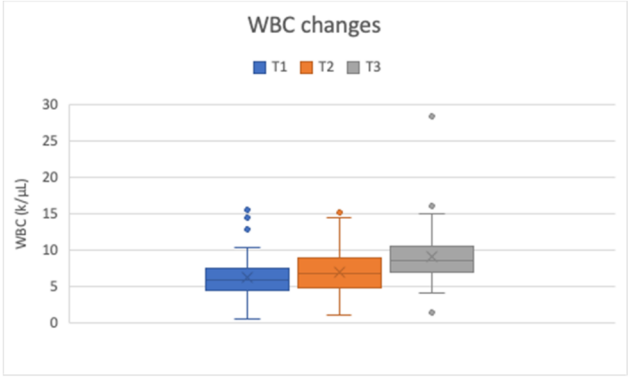

Figure 1. WBC (=white blood cells) at T1, T2 and T3.

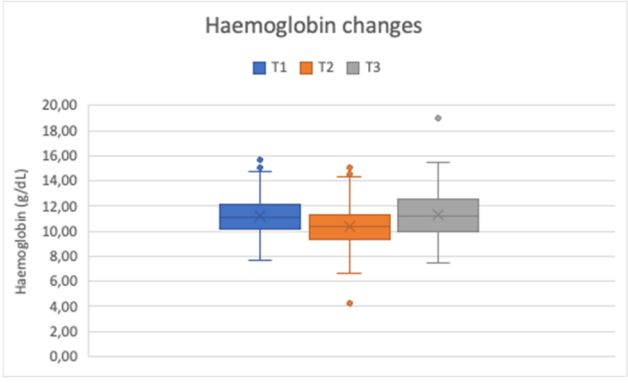

Figure 2. Hemoglobin at T1, T2 and T3.

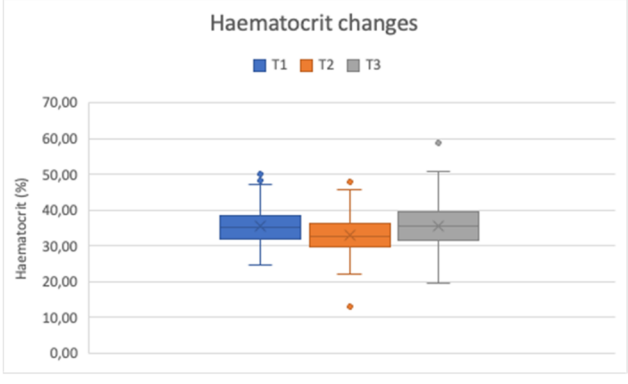

Figure 3. Hematocrit at T1, T2 and T3.

followed by a significant increase between T2 and T3 ($p < 0.0001$). However, the overall change between T1 and T3 was not statistically significant. Haematocrit demonstrated

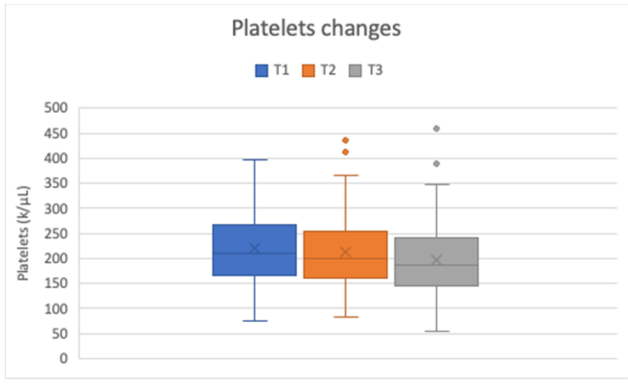

Figure 4. Platelets at T1, T2 and T3.

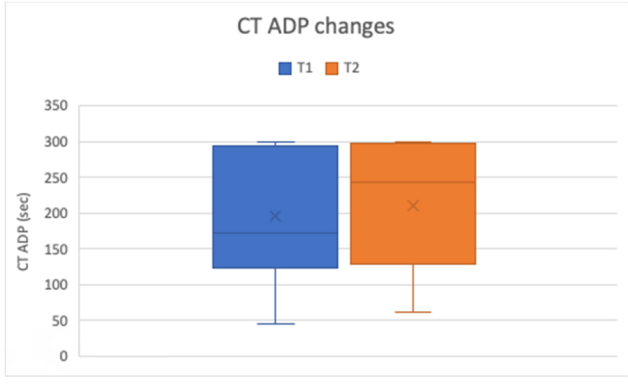

Figure 5. CT-ADP at T1 and T2.

a significant decrease between T1 and T2 ($p < 0.0001$), with a subsequent modest increases observed between T2 and T3 ($p = 0.082$) and T1 and T3 ($p = 0.29$). Platelet counts significantly decreased between T1 and T2 ($p = 0.016$), T2 and T3 ($p < 0.0001$) and T1 and T3 ($p < 0.0001$). No significant overall variation was observed in CT-ADP between T1 and T2. However, inter-individual variability was noted. At T2, 20.53% of patients had a CT-ADP <120 s in 20.53% (mean: 210.36 ± 81.10 s, $p < 0.001$), compared to 22.80% at T1 (mean: 197.32 ± 79.94 s, $p < 0.001$).

Blood count, CT-ADP and outcomes

Survival analysis revealed a statistically significant association between elevated WBC levels and mortality during the follow-up period. Specifically, an increase in WBC levels from T1 to T2 was linked to a higher risk of death with a Hazard Ratio (HR) of 1.51 (95% Confidence Interval [CI]: 1.13–2.01; $p = 0.05$). No other significant association was observed between blood count parameters (haemoglobin, haematocrit, platelet count) or CT-ADP changes and the rates of death, stroke, or bleeding. All the results are summarised in Table A2 appendix.

Discussion

The main findings of the study are as follows: post-TAVI, significant changes in hematological parameters, including WBC, haemoglobin, and platelet levels, were observed,

reflecting the physiological response to the intervention. An elevation in WBC levels from T1 to T2 was significantly associated with increased mortality, while changes in haemoglobin and platelet counts were not linked to adverse outcomes. No significant variations in CT-ADP were observed post-TAVI, despite its established role in reflecting von Willebrand factor (vWF) activity. These findings provide important insights into the inflammatory and haemostatic changes following TAVI and their potential clinical implications.

Haemogram changes

After TAVI, significant changes in hematological parameters were observed, reflecting the physiological response to the intervention and its impact on both the cardiovascular and hematological systems. Previous TAVI studies consistently report transient alterations in blood count parameters such as WBC, haemoglobin, and platelet levels, often driven by procedural factors, inflammation, and hemodynamic changes. Haemoglobin levels typically decrease temporarily due to blood loss during the procedure or exacerbation of pre-existing anaemia. Post-TAVI inflammation, potentially triggered by the mechanical deployment of the valve and associated vascular manipulation, frequently results in an initial rise in WBC counts. Platelet counts may decline due to the inflammatory response or exposure to bioprosthetic valve materials, which can induce mild thrombocytopenia [6].

In our study, 75.42% of patients had anaemia at baseline, defined as haemoglobin levels <12 g/dL in females and <13 g/dL in males, consistent with prior reports among TAVI candidates (45%-64%) [6]. We observed a significant decrease in haemoglobin levels between T1 and T2, followed by a significant increase between T2 and T3, though the overall change from T1 to T3 was not statistically significant. Importantly, no significant association was found between haemoglobin levels and risk of bleeding, stroke, or death. This contrasts with existing literature, which links anaemia with adverse outcomes following TAVR, including a higher rates of blood transfusions, an increased risk of acute kidney injury, and elevated short and long-term mortality rates [6,12,13].

Conversely, WBC levels significantly increased across all time points (T1 to T3), and platelet counts showed a significant decline over the same period. Importantly, an elevation in WBC levels from T1 to T2 was independently associated with an increased risk of mortality. Despite the observed decline in platelet counts, no significant association was found between platelet changes and adverse outcomes. These findings highlight the distinct roles of WBC and platelet dynamics in post-TAVI prognosis. Several studies have linked the thromboinflammatory response to adverse outcomes following TAVR [14–16]. Kalińczuk et al. observed a transient increase in WBC counts and a consistent decline in platelet levels, both of which independently predicted higher long-term mortality over a 4-year period. A combined assessment of these markers enhanced their individual predictive performance [14]. The severity of thrombocytopenia appears to depend on factors such as the type of valve prosthesis and the vascular access site, with new-generation self-expanding prostheses and femoral access being associated with less severe thrombocytopenia [14,17,18]. Furthermore, the effect of thrombocytopenia on mortality depends on its severity [6,17,19]. In our study, no cases of severe thrombocytopenia were observed, and the incidence of moderate thrombocythemia was low. At T2, one patient exhibited moderate thrombocytopenia, while 20 patients experienced mild thrombocytopenia. By T3, the number of moderate thrombocytopenia cases increased to five, and mild thrombocytopenia rose to 24. These findings may be attributed to the predominant use of self-expandable prostheses and the high prevalence of transfemoral approaches in our cohort, both of which are associated with a reduced risk of significant thrombocytopenia. The low incidence of severe and moderate thrombocytopenia likely explains the lack of observed correlation between platelet count changes and survival in our study.

CT-ADP and von Willebrand factor

Acquired type 2 A von Willebrand syndrome (vWS) is a well-recognized condition frequently associated with severe AS. Its severity seems to correlate directly to the degree of aortic valve narrowing [5]. This condition results from the loss of high molecular weight (HMW) multimers of von Willebrand factor (vWf), which are critical for primary hemostasis. vWf enables platelet adhesion to damaged endothelium *via* platelet glycoprotein GPIbα and endothelial collagen, and it supports secondary hemostasis by stabilising and transporting factor VIII, thereby preventing its degradation. The primary mechanism behind vWS in AS is the proteolysis of HMW vWf multimers as they pass through the narrowed aortic valve. The high shear forces exerted across the stenotic valve induce structural changes in vWf, exposing amino acids 842 and 843, which render the molecule more susceptible to cleavage by the protease ADAMTS13. This enzymatic action reduces HMW multimers to low molecular weight forms, impairing vWf's adhesive function, as its activity is directly proportional to its multimeric state [20]. CT-ADP, a rapid point-of-care test, assesses haemostatic function and is highly dependent on vWF activity. Previous studies have reported that TAVI restores vWf abnormalities within minutes after procedure [8–10,21,22]. Some evidence suggests that persistent defects in HMW-multimer post-TAVI may predict complications such as paravalvular leaks, though this remains a topic of debate and was not observed in the present study [8,23]. Surprisingly, in our study, we did not observe any significant variation in CT-ADP values 15 min after the deployment of the prosthesis. While the underlying reason remains unclear, this may be due to differences in the population studied or variations in the measurement equipment and protocols used. Further investigation is warranted to elucidate these discrepancies and the clinical implications of CT-ADP dynamics following TAVI.

Study limitations

This study's single-centre, retrospective study presents several limitations that may affect the generalisability of

our findings. First, the sample size of 117 patients, while yielding valuable insights, may not sufficiently capture the variability in clinical outcomes, particularly for less common events. Second, the single-centre nature of the study constraints external validity, as the results may reflect specific local practices, patient demographics, and procedural techniques. Third, retrospective data collection may be prone to selection bias and inconsistencies in documentation, which could influence the robustness of the analysis. Additionally, the lack of a control group further restricts the ability to draw causal inferences between observed changes and clinical outcomes. Despite these limitations, the study offers meaningful preliminary insights into the hematological and haemostatic dynamics following TAVI and their potential prognostic significance. These findings serve as a foundation for future research and emphasise the necessity of larger, multicentre, prospective studies to validate these observations and refine our understanding of their implications for patient care.

Conclusion

Our findings emphasise the importance of WBC monitoring as a predictor of mortality in TAVI patients, reflecting the role of systemic inflammation in adverse outcomes. While platelet changes were not predictive in our cohort, their assessment remains relevant, particularly in populations with higher rates of severe thrombocytopenia. Additionally, the lack of significant changes in CT-ADP suggests that more research is needed to establish its role in TAVI-related haemostatic recovery and complication prediction. These insights are crucial for refining post-TAVI management and improving long-term patient outcomes.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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Appendix

Table A1. Changes in blood count and CT ADP at T1, T2 and T3.

	N	Missing values	Mean	p value	Variation
WBC T2-T1	111	6	0.69 ± 1.84	0.0001	Increase
WBC T3-T2	104	13	1.94 ± 2.63	<0.0001	Increase
WBC T3-T1	107	10	2.78 ± 2.78	<0.0001	Increase
Haemoglobin T2-T1	112	5	−0.79 ± 0.78	<0.0001	Decrease
Haemoglobin T3-T2	105	12	0.87 ± 1.44	<0.0001	Increase
Haemoglobin T3-T1	107	10	0.09 ± 1.47	0.50	Increase
Haematocrit T2-T1	112	5	−2.39 ± 2.53	<0.0001	Decrease
Haematocrit T3-T2	105	12	6.06 ± 35.35	0.0817	Increase
Haematocrit T3-T1	107	10	3.64 ± 35.45	0.2906	Increase
Platelets T2-T1	112	5	−7.02 ± 30.31	0.0158	Decrease
Platelets T3-T2	105	12	−16.84 ± 39.83	<0.0001	Decrease
Platelets T3-T1	107	10	−23.41 ± 40.78	<0.0001	Decrease
CT-ADP T2-T1	110	7	12.75 ± 78.84	0.093	Increase

Table A2. Changes in blood count or CT-ADP and rates of death, stroke, or bleeding.

Description	Hazard ratio	LCL	UCL	P val	n
DEATH					
ctadp_bmt1 Unit = 1	1.00	0.99	1.01	0.772	105
ctadp_bmt2 Unit = 1	1.00	0.99	1.00	0.398	105
ctadp_bmt3 Unit = 1	1.00	0.99	1.01	0.958	100
wbc_bmt1 Unit = 1	0.89	0.65	1.20	0.440	107
wbc_bmt2 Unit = 1	1.09	0.87	1.36	0.465	104
wbc_bmt3 Unit = 1	0.98	0.79	1.22	0.877	101
hgb_bmt1 Unit = 1	1.12	0.77	1.64	0.552	107
hgb_bmt2 Unit = 1	1.19	0.82	1.73	0.349	105
hgb_bmt3 Unit = 1	0.92	0.64	1.33	0.665	101
hct_bmt1 Unit = 1	1.05	0.93	1.19	0.436	107
hct_bmt2 Unit = 1	1.06	0.94	1.19	0.344	105
hct_bmt3 Unit = 1	0.98	0.88	1.10	0.746	101
plat_cd_bmt1 Unit = 1	1.00	0.99	1.01	0.685	107
plat_cd_bmt2 Unit = 1	1.00	0.99	1.01	0.793	105
plat_cd_bmt3 Unit = 1	1.00	0.99	1.01	0.838	101
ctadp_bmdt21 Unit = 1	0.99	0.98	1.00	0.163	103
wbc_bmdt21 Unit = 1	1.51	1.13	2.01	0.005	104
hgb_bmdt21 Unit = 1	1.51	0.57	3.99	0.409	105
hct_bmdt21 Unit = 1	1.06	0.80	1.40	0.693	105
plat_cd_bmdt21 Unit = 1	1.02	1.00	1.03	0.072	105
ctadp_bmdt31 Unit = 1	1.00	0.99	1.01	0.922	98
wbc_bmdt31 Unit = 1	1.02	0.85	1.22	0.824	101
hgb_bmdt31 Unit = 1	0.74	0.40	1.38	0.339	101
hct_bmdt31 Unit = 1	0.92	0.76	1.11	0.368	101
plat_cd_bmdt31 Unit = 1	1.00	0.98	1.01	0.594	101
ctadp_bmdt32 Unit = 1	1.00	1.00	1.01	0.402	98
wbc_bmdt32 Unit = 1	0.99	0.74	1.31	0.934	98
hgb_bmdt32 Unit = 1	0.62	0.35	1.12	0.114	99
hct_bmdt32 Unit = 1	0.88	0.74	1.06	0.174	99
plat_cd_bmdt32 Unit = 1	0.99	0.97	1.01	0.141	99
STROKE					
ctadp_bmt1 Unit = 1	1.00	0.99	1.01	0.577	104
ctadp_bmt2 Unit = 1	1.00	0.99	1.01	0.549	104
ctadp_bmt3 Unit = 1	1.00	0.99	1.01	0.673	99
wbc_bmt1 Unit = 1	0.90	0.61	1.31	0.569	106
wbc_bmt2 Unit = 1	0.93	0.67	1.28	0.642	103
wbc_bmt3 Unit = 1	1.06	0.86	1.30	0.609	100
hgb_bmt1 Unit = 1	0.86	0.53	1.42	0.559	106
hgb_bmt2 Unit = 1	0.92	0.56	1.50	0.734	104
hgb_bmt3 Unit = 1	0.95	0.60	1.51	0.829	100
hct_bmt1 Unit = 1	0.92	0.78	1.09	0.346	106
hct_bmt2 Unit = 1	0.95	0.82	1.11	0.550	104
hct_bmt3 Unit = 1	0.97	0.84	1.13	0.699	100
plat_cd_bmt1 Unit = 1	1.00	0.99	1.01	0.702	106
plat_cd_bmt2 Unit = 1	1.00	0.99	1.01	0.758	104
plat_cd_bmt3 Unit = 1	1.00	0.99	1.01	0.575	100
ctadp_bmdt21 Unit = 1	1.00	0.99	1.01	0.985	102
wbc_bmdt21 Unit = 1	1.04	0.64	1.69	0.873	103
hgb_bmdt21 Unit = 1	1.41	0.43	4.65	0.571	104
hct_bmdt21 Unit = 1	1.16	0.80	1.69	0.433	104
plat_cd_bmdt21 Unit = 1	1.00	0.96	1.03	0.844	104
ctadp_bmdt31 Unit = 1	0.99	0.99	1.00	0.251	97
wbc_bmdt31 Unit = 1	1.08	0.91	1.27	0.395	100
hgb_bmdt31 Unit = 1	1.09	0.64	1.87	0.754	100
hct_bmdt31 Unit = 1	1.00	0.95	1.05	0.870	100
plat_cd_bmdt31 Unit = 1	1.01	0.99	1.03	0.290	100
ctadp_bmdt32 Unit = 1	1.00	0.99	1.01	0.462	97
wbc_bmdt32 Unit = 1	1.24	0.87	1.78	0.240	97
hgb_bmdt32 Unit = 1	0.95	0.47	1.89	0.877	98
hct_bmdt32 Unit = 1	0.98	0.79	1.21	0.854	98
plat_cd_bmdt32 Unit = 1	1.00	0.98	1.03	0.728	98
BLEEDING					
ctadp_bmt1 Unit = 1	1.00	0.99	1.01	0.515	104
ctadp_bmt2 Unit = 1	1.00	0.99	1.01	0.555	104
ctadp_bmt3 Unit = 1	1.00	1.00	1.01	0.322	99
wbc_bmt1 Unit = 1	1.05	0.84	1.32	0.643	106
wbc_bmt2 Unit = 1	1.06	0.84	1.33	0.614	103
wbc_bmt3 Unit = 1	1.02	0.84	1.24	0.844	100
hgb_bmt1 Unit = 1	0.83	0.55	1.26	0.394	106
hgb_bmt2 Unit = 1	0.98	0.65	1.47	0.921	104
hgb_bmt3 Unit = 1	0.93	0.65	1.34	0.698	100
hct_bmt1 Unit = 1	0.93	0.81	1.07	0.300	106

(*Continued*)

Table A2. Continued.

Description	Hazard ratio	LCL	UCL	P val	n
hct_bmt2 Unit = 1	0.98	0.86	1.12	0.759	104
hct_bmt3 Unit = 1	1.01	1.00	1.01	0.020	100
plat_cd_bmt1 Unit = 1	1.00	0.99	1.01	0.946	106
plat_cd_bmt2 Unit = 1	1.00	0.99	1.01	0.982	104
plat_cd_bmt3 Unit = 1	1.00	0.99	1.01	0.993	100
ctadp_bmdt21 Unit = 1	1.00	0.99	1.01	0.986	102
wbc_bmdt21 Unit = 1	1.04	0.68	1.59	0.868	103
hgb_bmdt21 Unit = 1	3.13	0.99	9.90	0.052	104
hct_bmdt21 Unit = 1	1.38	0.97	1.95	0.074	104
plat_cd_bmdt21 Unit = 1	1.00	0.97	1.03	0.767	104
ctadp_bmdt31 Unit = 1	1.01	1.00	1.01	0.124	97
wbc_bmdt31 Unit = 1	1.02	0.84	1.23	0.866	100
hgb_bmdt31 Unit = 1	1.08	0.71	1.63	0.727	100
hct_bmdt31 Unit = 1	1.01	1.00	1.01	0.015	100
plat_cd_bmdt31 Unit = 1	1.00	0.98	1.02	0.962	100
ctadp_bmdt32 Unit = 1	1.00	1.00	1.01	0.192	97
wbc_bmdt32 Unit = 1	1.02	0.78	1.34	0.868	97
hgb_bmdt32 Unit = 1	0.81	0.45	1.43	0.460	98
hct_bmdt32 Unit = 1	1.01	1.00	1.01	0.017	98
plat_cd_bmdt32 Unit = 1	1.00	0.98	1.02	0.798	98