CT-first resuscitation for severe blunt trauma: A propensity score–matched cohort study

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BACKGROUND: In unstable blunt trauma, whole-body computed tomography (WBCT) is often deferred because the bleeding source is uncertain, potentially delaying definitive hemorrhage control. This retrospective cohort study assessed whether CT-first resuscitation (CTFR)—immediate WBCT in a CT-equipped trauma resuscitation room with prespecified triggers for hemorrhage control—is associated with transfusion, time to hemostatic intervention, and mortality after blunt trauma.

METHODS: We conducted a retrospective cohort study (2019–2023) comparing adults managed with CTFR at a single center with patients in the Japan Trauma Data Bank, a national trauma registry. We performed 1:1 propensity score matching (n = 248 per group). The primary outcome was 24-hour red blood cell (RBC) units; secondary outcomes were time to CT initiation, time to first hemostatic intervention (surgical or endovascular), and in-hospital mortality. Sensitivity analyses used multiple imputation.

RESULTS: CTFR shortened the time to CT initiation (median, 0.4 vs. 29.0 min; $p < 0.001$) and time to first hemostatic intervention (median, 53.7 vs. 134.0 min; $p < 0.001$). Any RBC transfusion within 24 hours was similar (29.4% vs. 30.6%; $p = 0.845$). Adjusted 24-hour RBC units were lower with CTFR (mean difference, −0.84 units; 95% CI: −1.65 to −0.03; $p = 0.043$). In-hospital mortality was similar (9.7% vs. 8.9%; $p = 0.877$). In an exploratory subgroup of patients presenting with shock, CTFR was associated with a larger reduction in 24-hour RBC units (adjusted mean difference, −3.76 units; 95% CI: −6.44 to −1.09; $p = 0.006$).

CONCLUSIONS: In a matched comparison with a national registry cohort, CTFR was associated with earlier WBCT, shorter time to hemostatic intervention, and modestly lower adjusted 24-hour RBC transfusion requirements, while mortality was similar. These associations appeared more pronounced among patients presenting with shock in exploratory subgroup analyses. (*J Trauma Acute Care Surg* 2026;00:000–000.

LEVEL OF EVIDENCE: Therapeutic/Care Management; Level III.

KEY WORDS: Blunt trauma, whole-body computed tomography, hybrid ER, hemorrhage control, resuscitation

Delivering definitive hemorrhage control rapidly remains a central challenge in trauma system design.[1,2] Observational studies consistently show that shorter time to definitive hemostasis (surgical or endovascular) is associated with improved survival among severely injured patients.[2–5] Accordingly, some trauma centers have adopted direct-to-operating room (DOR) pathways in which selected patients bypass the emergency department (ED) for immediate surgery, enabling rapid incision times and outcomes in selected populations.[2,4,5] However, DOR is most applicable when the bleeding source is anatomically apparent without cross-sectional imaging, such as penetrating truncal trauma or obvious operative abdominal injury.

In blunt trauma, by contrast, the bleeding source is frequently uncertain based on mechanism, physical examination, and initial bedside tests alone. Concomitant traumatic brain injury is also common in severe blunt trauma, and early recognition of intracranial hemorrhage may influence early resuscitation priorities and the appropriateness of an immediate operative strategy.[6] This diagnostic uncertainty complicates early triage among operative, endovascular, and nonoperative management (NOM) strategies and can prolong time to definitive hemostasis. Early whole-body computed tomography (WBCT) improves detection of thoracoabdominal injuries, refines injury severity assessment, and often changes clinical decision-making.[7,8] Large registry analyses further suggest that WBCT may be associated with improved survival even in hemodynamically compromised patients, provided that scanning does not delay hemorrhage control.[7] Yet, because conventional workflows typically require patient transfers among the resuscitation bay, CT suite, and operating room or

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angiography, rapid imaging can still introduce meaningful delays when timely hemostasis is required.[8,9]

This trade-off has prompted the development of all-in-one resuscitation environments. CT-equipped trauma resuscitation rooms (CT-TRRs), also referred to as hybrid emergency rooms (ERs) in Japan, integrate a multidetector CT scanner with angiographic capability and operative infrastructure in a single location.[10,11] By reducing in-hospital transfers, CT-TRRs may shorten the time from diagnosis to definitive hemorrhage control. A key remaining challenge is to make cross-sectional imaging feasible before completion of the primary survey when diagnostic uncertainty is greatest and physiologic status is most unstable, without delaying definitive hemorrhage control. We developed and implemented a CT-first resuscitation (CTFR) strategy in a CT-TRR. Unlike prior trials of early WBCT conducted within conventional multiroom workflows, CTFR embeds rapid WBCT into the resuscitation bay as the first structured step and couples imaging and physiologic findings to prespecified triggers for operative, endovascular, or NOM without in-hospital transfer. In this context, CTFR was developed as a protocolized, decision-triggered system that links early imaging to predefined hemostatic pathways. Using the Japan Trauma Data Bank (JTDB) as an external comparator, we evaluated the association of CTFR with key process measures (time to CT initiation and time to first hemostatic intervention) and clinical outcomes, including 24-hour red blood cell (RBC) transfusion requirements and in-hospital mortality, compared with conventional multiroom workflows.

METHODS

Study Design, Data Source, and Study Population

We conducted a retrospective propensity score–matched cohort study (January 1, 2019, to December 31, 2023) comparing CTFR at Saiseikai Yokohamashi Tobu Hospital, Yokohama, Japan, with a matched cohort from the JTDB, a nationwide trauma registry of 234 facilities in Japan. This study followed the STROBE reporting guideline, Supplemental Digital Content 1, http://links.lww.com/TA/F538. The institutional review board approved this study with a waiver of informed consent due to the retrospective design and use of deidentified registry data.

We defined eligibility criteria a priori and applied them to both cohorts. Eligible patients were adults aged 16 years or older with blunt trauma who were transported directly from the scene to the ED. We excluded patients who were dead on arrival, received physician-staffed prehospital care, had nonblunt mechanisms, or had unsurvivable injury (Abbreviated Injury Scale [AIS] 6 in any region). The CTFR cohort comprised consecutive eligible patients for whom CTFR was activated and immediate WBCT was performed on ED arrival in the CT-TRR. The comparator cohort was derived from the JTDB using the same base eligibility criteria and excluded patients treated at the CTFR center to avoid overlap between cohorts.

CTFR Protocol

CTFR activation criteria were based on institutional prehospital physiologic triage, supported by mechanism and anatomic concerns, and confirmed by the attending trauma surgeon on arrival; safety-based exclusions were applied at the bedside as needed. Patients requiring immediate bedside stabilization, including airway intervention, were not taken through the CTFR pathway. CTFR activation and immediate WBCT in the CT-TRR were ascertained from institutional CT-TRR records. WBCT was performed immediately on arrival using a rapid scoutless protocol (continuous head-to-pelvis noncontrast acquisition) designed to complete in ~1 minute (Supplemental Digital Content, Video S1, Supplemental Digital Content 4, http://links.lww.com/TA/F541). Radiation dose and image quality were monitored per institutional quality-control processes and were within recommended diagnostic reference levels. After initial WBCT, the attending trauma surgeon led definitive management decisions according to Japan Advanced Trauma Evaluation and Care principles. Additional protocol details are provided in Supplemental Digital Content, Table S1, Supplemental Digital Content 2, http://links.lww.com/TA/F539.

Outcomes

We prespecified process and clinical outcomes. Process outcomes included time from ED arrival to CT initiation and time from ED arrival to first hemostatic intervention. Time to first hemostatic intervention was defined as the earliest of surgical hemostasis (skin incision for laparotomy, thoracotomy, or preperitoneal pelvic packing) or endovascular hemostasis (arterial puncture for angioembolization). "Time to incision" and "time to needle" were assessed among patients who underwent surgical and endovascular hemostasis, respectively. Adjunctive procedures such as resuscitative endovascular balloon occlusion of the aorta were not classified as hemostatic interventions for this outcome. Clinical outcomes included receipt of any RBC units within 24 hours, the number of RBC units transfused within 24 hours, in-hospital mortality, and hospital length of stay. Secondary clinical outcomes also included rates of definitive hemostatic interventions. We also summarized additional procedures/interventions (chest tube placement, resuscitative endovascular balloon occlusion of the aorta, and craniotomy).

Propensity Score Matching

The exposure was management at the CTFR center versus the JTDB cohort. Propensity scores were estimated using complete-case covariates in a multivariable logistic regression model including demographics, prehospital

and ED physiology, Injury Severity Score (ISS), AIS ≥3 indicators by body region, comorbidity burden, preinjury antithrombotic use, time from scene to ED arrival, and mechanism categories. Because the prehospital Glasgow Coma Scale had substantial missingness, we used the Japan Coma Scale (grades 0–3) as a four-level categorical covariate (Supplemental Digital Content, Table S2, Supplemental Digital Content 2, http://links.lww.com/TA/F539). We performed 1:1 nearest-neighbor matching without replacement using a caliper width of 0.2 SDs of the logit of the propensity score and assessed balance using standardized mean differences (absolute value <0.1). Covariates included in the propensity score model are listed in Table 1.

Statistical Analysis

We analyzed the propensity score–matched cohort. Categorical variables were compared using Fisher exact tests and continuous variables using the Mann-Whitney *U* test. Regression models used matched-pair cluster-robust standard errors (pair_id). For binary outcomes, we reported unadjusted odds ratios with 95% CIs. For 24-hour RBC units, we estimated an adjusted mean difference using multivariable linear regression, and assessed robustness to zero inflation and right skewness using prespecified Poisson regression and a two-part model [logistic regression for any RBC units (≥1 unit) and negative binomial regression for RBC units among transfused patients]. Models adjusted for age, sex, shock (systolic blood pressure <90 mm Hg or heart rate >120 beats/min on ED arrival), ISS ≥25, and AIS ≥3 injuries in the head, chest, abdomen, and pelvis. As a sensitivity analysis for missing covariates, we performed multiple imputation by chained equations (m = 20) and conducted overlap-weighted analyses in each imputed data set; estimates were combined using Rubin's rules. Propensity score matching estimates associations in the matched cohort, whereas overlap weighting targets the overlap population.

TABLE 1. Admission Characteristics of CTFR and JTDB Cohorts Before and After Propensity Score Matching

	Before Matching			After Matching		
Covariate	**CTFR (n = 253)**	**JTDB (n = 72,183)**	**SMD**	**CTFR (n = 248)**	**JTDB (n = 248)**	**SMD**
Age (y)	49.7 ± 21.1	67.0 ± 21.4	0.816	49.9 ± 21.1	49.4 ± 21.7	0.024
Sex (male)	187 (73.9)	40,916 (56.7)	0.368	184 (74.2)	193 (77.8)	0.085
Charlson Comorbidity Index	0.3 ± 0.6	0.5 ± 0.8	0.351	0.3 ± 0.6	0.3 ± 0.6	0.006
Anticoagulant therapy	16 (6.3)	7,475 (10.4)	0.146	16 (6.5)	16 (6.5)	0.000
Mechanism of injury						
MVC-vehicle occupant	47 (18.6)	6,091 (8.4)	0.300	47 (19.0)	44 (17.7)	0.031
MVC-motorcyclist	45 (17.8)	6,875 (9.5)	0.242	44 (17.7)	45 (18.1)	0.011
MVC-pedestrian	29 (11.5)	4,069 (5.6)	0.209	29 (11.7)	35 (14.1)	0.072
Fall from height	97 (38.3)	4,853 (6.7)	0.818	93 (37.5)	94 (37.9)	0.008
Other	35 (13.8)	50,295 (69.7)	1.374	35 (14.1)	30 (12.1)	0.060
Prehospital vitals						
SBP (mm Hg)	118.4 ± 33.5	144.4 ± 32.9	0.784	119.0 ± 33.4	120.3 ± 33.4	0.039
HR (/min)	93.5 ± 23.5	85.3 ± 18.4	0.388	92.9 ± 22.7	93.3 ± 23.4	0.017
RR (/min)	25.8 ± 6.0	21.0 ± 4.4	0.899	25.6 ± 5.8	25.3 ± 6.3	0.052
JCS	1.4 ± 1.0	0.9 ± 1.1	0.463	1.4 ± 1.0	1.4 ± 1.3	0.010
Time from prehospital to ED (min)	13.6 ± 7.5	14.8 ± 12.4	0.113	13.7 ± 7.5	14.5 ± 9.7	0.092
ED vitals						
SBP (mm Hg)	124.5 ± 30.1	144.8 ± 31.8	0.657	125.0 ± 29.9	124.3 ± 33.1	0.024
HR (/min)	95.7 ± 23.9	84.4 ± 18.7	0.527	95.1 ± 23.3	93.5 ± 24.6	0.067
Shock on arrival*	57 (22.5)	5,037 (7.0)	0.449	54 (21.8)	57 (23.0)	0.029
RR (/min)	22.0 ± 6.1	20.2 ± 5.2	0.311	21.9 ± 6.0	21.2 ± 6.7	0.105
GCS	12.3 ± 3.6	13.8 ± 2.6	0.503	12.3 ± 3.5	12.3 ± 3.8	0.006
Injury severity						
ISS	20.2 ± 13.6	12.4 ± 7.8	0.710	20.1 ± 13.7	19.3 ± 11.5	0.057
ISS ≥ 25	93 (36.8)	7,571 (10.5)	0.650	90 (36.3)	80 (32.3)	0.085
AIS ≥3 injury						
Head	88 (34.8)	16,183 (22.4)	0.276	85 (34.3)	89 (35.9)	0.034
Chest	93 (36.8)	12,862 (17.8)	0.435	92 (37.1)	87 (35.1)	0.042
Abdomen	39 (15.4)	1,977 (2.7)	0.452	38 (15.3)	35 (14.1)	0.034
Pelvis	81 (32.0)	25,029 (34.7)	0.056	78 (31.5)	73 (29.4)	0.044

Continuous variables are presented as mean ± SD; categorical variables as n (%).
*Shock was defined as SBP <90 mm Hg or HR >120 beats/min.
GCS, Glasgow Coma Scale; HR, heart rate; JCS, Japan Coma Scale; MVC, motor vehicle collision; RR, respiratory rate; SBP, systolic blood pressure; SMD, standardized mean difference.

Additional sensitivity analyses excluded patients who died within 24 hours of ED arrival. To assess robustness to facility-level differences, we conducted separate overlap-weighted analyses restricting the JTDB cohort to high-volume facilities and clustering SEs by facility. We also performed an exploratory descriptive and adjusted subgroup analysis among patients presenting with shock on ED arrival, defined as systolic blood pressure < 90 mm Hg or heart rate > 120 beats/min. This analysis was conducted within the matched cohort using a parsimonious adjustment model, including age, sex, and ISS ≥ 25 to estimate the subgroup-specific association between CTFR and 24-hour RBC units.

All tests were two-sided, and $p < 0.05$ was considered statistically significant.

RESULTS

Study Population and Matching

Of 296 CTFR activations, 14 were excluded because of bedside safety concerns, CT-TRR unavailability, or technical issues; 282 patients underwent immediate WBCT, of whom 253 met analytic eligibility criteria. In the JTDB, 72,183 of 113,985 had complete covariate data for matching. After propensity score matching, 248 CTFR patients were matched 1:1 with JTDB patients (Fig. 1). Before matching, CTFR patients were younger than JTDB patients (mean age, 49.7 vs. 67.0 y) and had more severe physiologic abnormalities and higher injury severity, with different injury mechanisms. After matching, these differences were largely attenuated (Table 1). Missingness of key covariates in the eligible JTDB cohort was most prominent for prehospital physiology and transportation time, and complete-case and missing-case patients were broadly similar (Supplemental Digital Content, Table S3, Supplemental Digital Content 2, http://links.lww.com/TA/F539).

Primary and Secondary Outcomes

The distribution of 24-hour RBC units was highly zero-inflated (Supplemental Digital Content, Figure S1, Supplemental Digital Content 3, http://links.lww.com/TA/F540). Unadjusted 24-hour RBC units did not differ between groups [median (interquartile range, IQR), 0.0 (0.0–2.0) in CTFR vs 0.0 (0.0–3.6) in JTDB; $p = 0.520$]. The proportion of patients receiving any RBC transfusion within 24 hours was similar between groups (29.4% vs. 30.6%; $p = 0.845$) (Table 2). In multivariable linear regression with matched-pair cluster-robust SEs, CTFR was associated with lower 24-hour RBC transfusion requirements compared with the matched JTDB cohort (adjusted mean difference, −0.84 units; 95% CI: −1.65 to −0.03; $p = 0.043$) (Table 2). As shown in Table 2, CTFR was associated with a shorter time to CT initiation [median (IQR), 0.4 (0.0–1.0) vs. 29.0 (19.0–48.0) min; $p < 0.001$] and more frequent use of WBCT (100.0% vs. 89.1%; $p < 0.001$).

Among patients receiving any hemostatic intervention, time to first hemostatic intervention was shorter in the CTFR group [median (IQR), 53.7 (41.4–87.0) vs. 134.0 (77.0–176.6) min; $p < 0.001$] (Table 2; Supplemental Digital Content, Figure S2, Supplemental Digital Content 3, http://links.lww.com/TA/F540). When stratified by modality, time to needle was shorter with CTFR, whereas time to incision did not differ (Table 2). In component analyses (Supplemental Digital Content, Table S4, Supplemental Digital Content 2, http://links.lww.com/TA/F539), rates of laparotomy (5.6% vs. 11.7%) and damage-control surgery (4.0% vs. 9.3%) were lower in the CTFR group, whereas pelvic angioembolization was higher (9.7% vs. 4.4%). In-hospital mortality was similar between groups [24/248 (9.7%) vs. 22/248 (8.9%); $p = 0.877$] (Table 2).

Adjusted and Sensitivity Analyses

Sensitivity analyses using Poisson regression yielded consistent results (incidence rate ratio, 0.65; 95% CI: 0.46–0.92; $p = 0.016$). In a prespecified 2-part model, CTFR was not associated with transfusion incidence, and transfusion volume was attenuated among transfused patients. In the logistic component, CTFR was not associated with the odds of any RBC transfusion within 24 hours (adjusted OR, 0.84; 95% CI: 0.53–1.31; $p = 0.436$). In the negative binomial component among transfused patients (n = 149), CTFR was associated with lower 24-hour RBC transfusion requirements, although the association did not reach statistical significance (incidence rate ratio, 0.79; 95% CI: 0.62–1.02; $p = 0.076$) (Supplemental Digital Content, Table S5, Supplemental Digital Content 2, http://links.lww.com/TA/F539). After excluding patients who died within 24 hours of ED arrival, the association remained consistent and statistically significant (adjusted mean difference, −0.92 units; 95% CI: −1.75 to −0.07; $p = 0.034$). Results were directionally consistent in overlap-weighted analyses restricting the JTDB cohort to high-volume facilities and in multiple-imputation analyses addressing missing covariates (Supplemental Digital Content, Table S6, Supplemental Digital Content 2, http://links.lww.com/TA/F539).

Subgroup Analyses

Subgroup analyses suggested potentially larger transfusion reductions with CTFR among patients with shock, higher injury severity, severe abdominal injury, and among those with any hemostatic intervention (Fig. 2). The mean difference favored CTFR among patients with shock on ED arrival but not among those without shock (p for interaction = 0.001). Spline-based interaction models treating ISS and ED arrival systolic blood pressure as continuous modifiers also supported effect modification (p for interaction = 0.004 and 0.021, respectively) (Figs. 3A, B). In spline models for abdominal AIS, evidence of interaction was not observed ($p = 0.123$) (Fig. 3C). Among patients presenting with shock on ED arrival (n = 54 in the CTFR group and n = 57 in

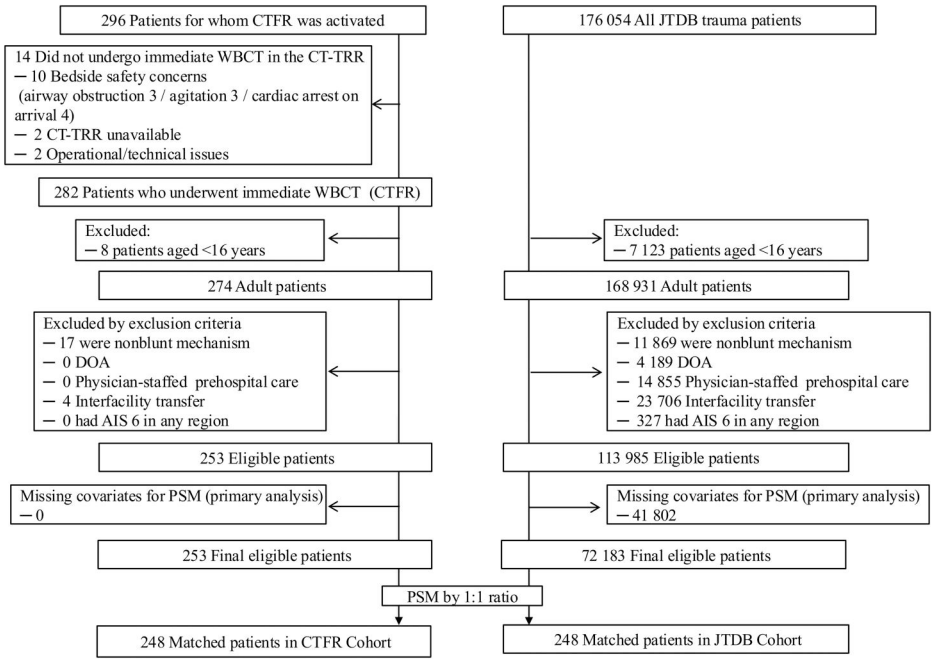

Figure 1. Study the flow diagram of patient selection and matching. Flow diagram summarizing patient inclusion, exclusion, and 1:1 propensity score matching for the CT-First Resuscitation (CTFR) and Japan Trauma Data Bank cohorts. Sensitivity analyses addressed missing covariates in the registry cohort (Supplemental Digital Content, Table S6, Supplemental Digital Content 2, http://links.lww.com/TA/F539). DOA, dead on arrival; PSM, propensity score matching.

the JTDB group), CTFR was associated with a larger reduction in 24-hour RBC units than observed in the overall matched cohort (adjusted mean difference, −3.76 units; 95% CI: −6.44 to −1.09; $p = 0.006$). In this subgroup, median 24-hour RBC units were 0.0 (IQR, 0.0–3.7) in the CTFR group versus 4.0 (IQR: 0.0–10.0) in the JTDB group ($p = 0.009$), and any RBC transfusion within 24 hours was less frequent (42.6% vs. 63.2%; $p = 0.037$). Among shock patients who underwent any hemostatic intervention, time to first hemostatic intervention was also shorter in the CTFR group [median (IQR), 62.3 (51.3–89.1) vs. 147.5 (89.8–178.6) min; $p = 0.001$]. In the overall shock subgroup, in-hospital mortality was similar between groups (14.8% vs. 21.1%; $p = 0.463$). These subgroup and interaction analyses were exploratory and should be interpreted cautiously because of the limited sample size. Detailed results of the shock subgroup analysis are provided in Supplemental Digital Content, Table S7, Supplemental Digital Content 2, http://links.lww.com/TA/F539.

DISCUSSION

In this propensity score–matched analysis comparing a single-center CTFR strategy with a nationwide trauma registry, CTFR operationalized a CT-first approach for severe blunt trauma by incorporating immediate WBCT into early resuscitation and linking imaging and physiologic findings to prespecified pathways for definitive hemorrhage control. This strategy was associated with lower 24-hour RBC transfusion requirements and shorter time to first hemostatic intervention, with associations that appeared more pronounced in exploratory subgroup analyses among patients with shock or higher ISS. In a direct adjusted subgroup analysis, the reduction in 24-hour RBC transfusion among patients presenting with shock was larger than in the overall matched cohort.

Previous DOR programs reported short arrival-to-incision times (13–25.5 min), but primarily in penetrating trauma, where the bleeding source is often anatomically apparent.[2,5] In blunt trauma, diagnostic uncertainty makes WBCT indispensable yet time-consuming, limiting the applicability of traditional DOR pathways. CTFR integrated WBCT into the resuscitation bay and used CT findings to triage patients to operative, endovascular, or NOM, and was associated with a shorter time to first hemostatic intervention and less frequent laparotomy.

Our primary finding should be interpreted in context. The overall adjusted mean difference of −0.84 units was clinically modest at the population level and should not be interpreted as evidence that CTFR reduced transfusion incidence or improved survival in the matched cohort. This modest overall difference was likely attenuated by the high proportion of non-transfused or minimally transfused patients, creating a substantial floor effect. This interpretation is consistent with the shock

TABLE 2. Primary and Secondary Outcomes in the CTFR and JTDB Cohorts After Propensity Score Matching

Measure	CTFR (n = 248)	JTDB (n = 248)	*p*	OR or Difference (95% CI), When Applicable
Outcomes				
Any RBC transfusion within 24 h	73 (29.4)	76 (30.6)	0.845	0.94 (0.64–1.39)
RBC units transfused within 24 h	0.0 [0.0–2.0]	0.0 [0.0–3.6]	0.520	—
Adjusted mean difference in 24-h RBC units*	—	—	0.043	−0.84 (−1.65 to −0.03)
In-hospital mortality	24 (9.7)	22 (8.9)	0.877	1.10 (0.60–2.00)
Hospital length of stay (d)	16.0 [5.0–41.0]	18.0 [7.0–39.2]	0.882	—
Imaging and process				
WBCT	248 (100.0)	221 (89.1)	< 0.001	Not estimable
Time to CT initiation (min)	0.4 [0.0–1.0]	29.0 [19.0–48.0]	< 0.001	—
Hemostasis process				
Any hemostatic intervention †	54 (21.8)	55 (22.2)	1.000	0.98 (0.64–1.49)
Surgical hemostasis	21 (8.5)	30 (12.1)	0.237	0.68 (0.38–1.21)
Endovascular hemostasis	46 (18.5)	37 (14.9)	0.336	1.30 (0.81–2.08)
Surgical + endovascular hemostasis	13 (5.2)	12 (4.8)	1.000	1.08 (0.49–2.39)
Time to first hemostatic intervention (min)	53.7 [41.4–87.0]	134.0 [77.0–176.6]	< 0.001	—
Time to needle (min)	57.6 [42.5–86.0]	96.0 [71.0–161.0]	0.002	—
Time to incision (min)	125.4 [83.8–227.2]	170.0 [109.2–186.3]	0.322	—
Procedures and interventions				
Chest tube placement	36 (14.5)	25 (10.1)	0.171	1.51 (0.88–2.58)
REBOA	7 (2.8)	6 (2.4)	1.000	1.16 (0.40–3.36)
Craniotomy	20 (8.1)	15 (6.0)	0.484	1.35 (0.68–2.68)
Time to craniotomy incision (min)	164.9 [102.6–232.2]	129.0 [85.0–164.1]	0.075	—

Continuous variables are presented as median (IQR) and categorical variables as n (%).

*From multivariable linear regression in the propensity score–matched cohort adjusted for age, sex, shock on ED arrival, ISS ≥ 25, and AIS ≥ 3 injuries in the head, chest, abdomen, and pelvis (Supplementary Material, Table S5, Supplemental Digital Content 2, http://links.lww.com/TA/F539). Negative values indicate fewer transfused units with CTFR than with JTDB.

†Time to first hemostatic intervention was defined as the earliest of surgical hemostasis (skin incision for laparotomy, thoracotomy, or preperitoneal pelvic packing) or endovascular hemostasis (arterial puncture for angioembolization). REBOA was not considered a hemostatic intervention. All-time outcomes were measured from ED arrival and assessed among patients undergoing the corresponding procedures.

CT, computed tomography; OR, odds ratio; REBOA, resuscitative endovascular balloon occlusion of the aorta.

subgroup analysis, in which CTFR was associated with an adjusted reduction of 3.76 units of 24-hour RBC transfusion. Together, these findings suggest that the clinical relevance of CTFR may be concentrated in higher-risk patients, particularly those presenting with physiologic shock, in whom delays in diagnosis and hemorrhage control are most consequential. These subgroup findings should be considered exploratory.

For clinicians, the potential benefit of CTFR may be better reflected by complementary process and system-level outcomes, including earlier CT initiation, shorter time to first hemostatic intervention, and earlier selection of definitive hemorrhage-control modalities, rather than by the modest population-level reduction in RBC units alone. Thus, the reductions in time to CT initiation and hemostatic intervention should be considered in the context of physiologic risk: they are most likely to be clinically meaningful in patients presenting with shock or severe injury, whereas similar time differences in patients without shock may have less immediate clinical impact.

This study was underpowered to detect clinically important mortality differences; therefore, similar in-hospital mortality should not be interpreted as equivalence. In mature trauma systems with high baseline survival, process improvements may be more readily reflected in blood product use or invasive procedures than in mortality, but larger multi-institutional studies are needed to evaluate survival effects.[12]

Evidence from systematic reviews and large registry analyses suggests that early WBCT may be associated with improved survival compared with selective imaging among severely injured patients, particularly those with multiregion blunt injuries.[13] Huber-Wagner et al.[7] similarly reported, in a large German registry of severe blunt trauma (ISS ≥ 16), that integrating WBCT into initial trauma room care was associated with a lower standardized mortality ratio than expected. Trauma care in Japan often follows a CT-intensive workflow, with registry comparisons showing that CT is used in > 80% of JTDB patients—over 1.5-fold higher than in US Level I trauma centers.[14] However, prior "immediate CT" studies largely accelerated CT within conventional multiroom workflows and typically positioned WBCT after completion of the Advanced Trauma Life Support primary survey and initial stabilization, consistent with the traditional paradigm of deferring CT in hemodynamically unstable patients to prioritize hemorrhage control.[15] In REACT-2, patients requiring immediate surgery were excluded, imaging remained time-consuming (median, 30 min), and outcomes did not differ from standard

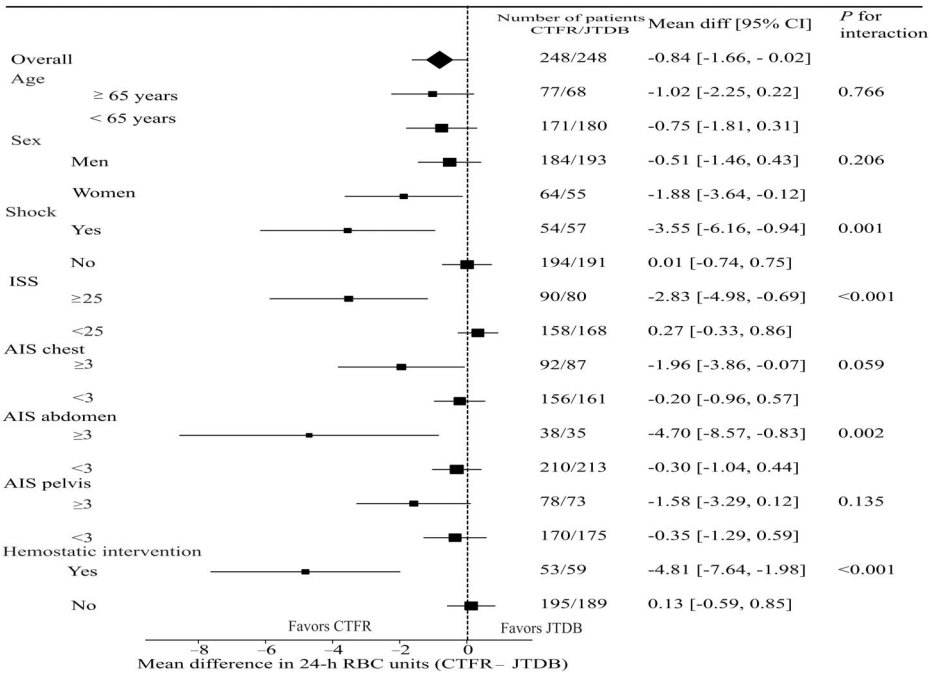

Figure 2. Subgroup analyses of 24-hour red blood cell transfusion. Forest plot showing adjusted mean differences in 24-hour red blood cell (RBC) units comparing CTFR with JTDB in the propensity score–matched cohort. Estimates were obtained using multivariable linear regression. Shock was defined as systolic blood pressure <90 mm Hg or heart rate >120 beats/min on emergency department arrival. Hemostatic intervention indicates receipt of any hemostatic intervention. *p* values shown are for interaction. Analyses were exploratory and not adjusted for multiplicity. The subgroup-specific estimates shown in this figure were derived from the interaction model; direct subgroup analyses for patients with shock are reported in the Results. Points indicate adjusted mean differences; horizontal bars, 95% CIs.

care.[16] In contrast, CTFR operationalized a CT-first sequence in selected patients by placing WBCT before completion of the primary survey within a CT-TRR and reinforcing management with prespecified decision triggers. Scanning was initiated a median of 0.4 minutes after arrival and completed in ~1 minute using a scoutless continuous head-to-pelvis protocol. Radiation output remained within national diagnostic reference levels, supporting the feasibility of this approach. CTFR incorporated bedside safety-based exclusions and attending surgeon confirmation, although CT acquisition–related safety events could not be compared with the JTDB cohort because such data were unavailable.

Current management of blunt solid-organ injury emphasizes selective NOM and angioembolization, both of which rely on CT to characterize injury severity and active bleeding.[17,18] CTFR uses a physiology-guided, two-step imaging strategy: rapid noncontrast WBCT for triage on arrival, including selected patients with physiologic instability, followed by selective contrast-enhanced CT after initial resuscitation when further solid-organ characterization is needed. This approach integrates CT-based decision-making into the earliest phase of resuscitation without delaying definitive hemorrhage control. However, contrast-enhanced CT data were unavailable in the registry comparator.

While prior CT-TRR reports often emphasized the architectural advantage of co-locating infrastructure,[19] CTFR operationalizes the hybrid environment as a protocol-driven CT-first system, linking immediate WBCT on arrival to predefined imaging and physiologic triggers that enable structured triage for anatomy-directed hemorrhage control, consistent with recent evaluations of hybrid ER workflows.[19,20]

The observed associations likely reflect the integrated effect of the CT-first protocol, hybrid ER infrastructure, trauma team experience, staffing, and institutional workflow, which are inherently linked in our setting. Accordingly, our findings should be interpreted as the effect of a system-level resuscitation strategy rather than the isolated effect of CT-first imaging or the CTFR protocol alone. This distinction is important because the protocol depends on immediate CT access, coordinated team activation, rapid image interpretation, interventional radiology and operating room readiness, and predefined CT-to-intervention pathways that may not be reproducible in conventional multiroom settings. Because these center-level factors were not captured in the JTDB, residual confounding by trauma center maturity and infrastructure cannot be excluded. In practical terms, these integrated workflow components are likely essential for achieving similar improvements.

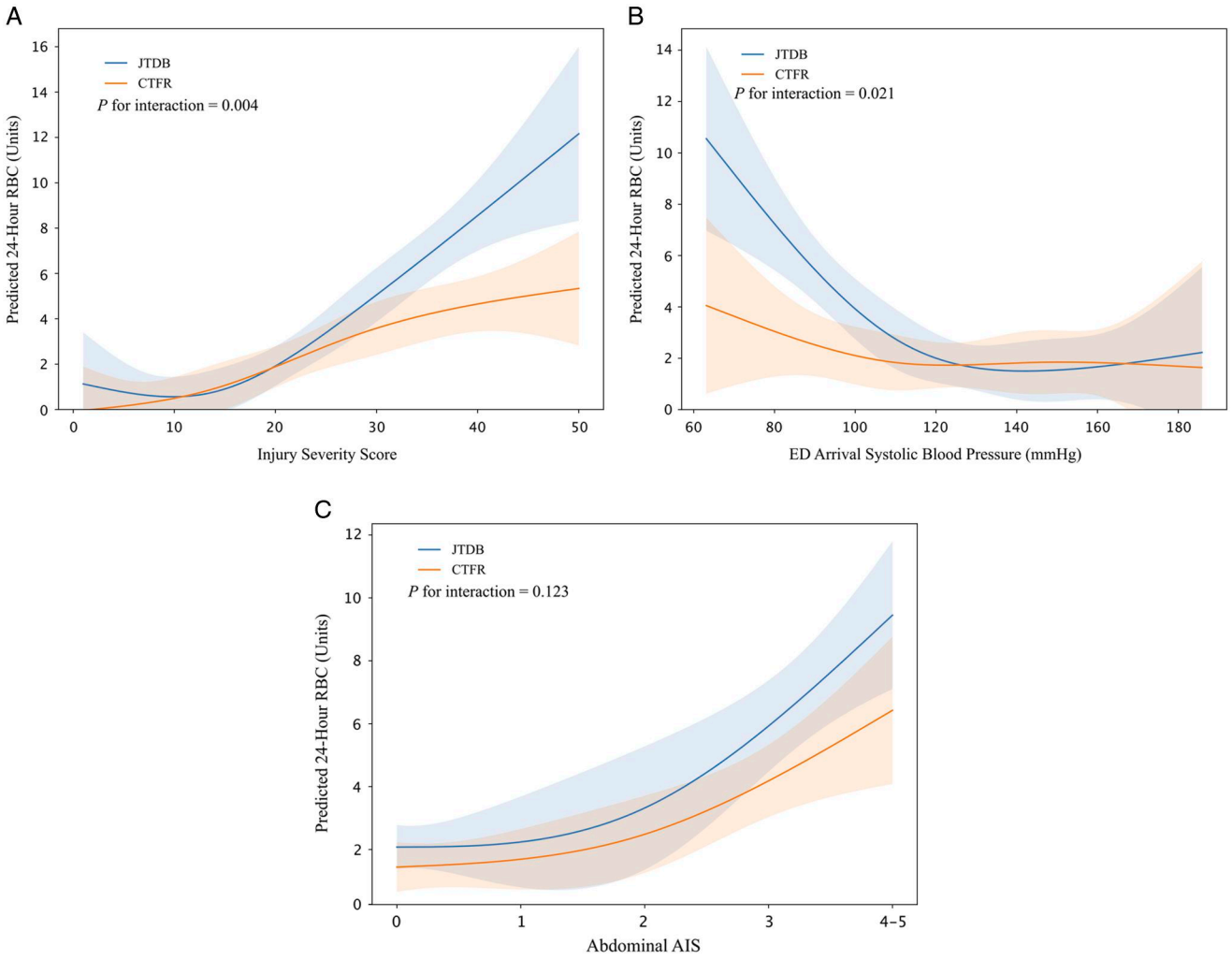

Figure 3. Effect modification of 24-hour red blood cell transfusion. Restricted cubic spline models evaluating effect modification of the association between CTFR and 24-hour red blood cell (RBC) transfusion requirements according to Injury Severity Score (*A*), systolic blood pressure on emergency department arrival (*B*), and maximum abdominal AIS severity (*C*) in the propensity score–matched cohort. *p* values are for the global interaction between CTFR and each modifier modeled with restricted cubic splines. Shaded regions indicate 95% CIs. Knot placement followed standard practice. SBP, systolic blood pressure.

Generalizability of CTFR to centers without CT-integrated resuscitation rooms is therefore limited. In such settings, the transferable element may not be the physical layout itself, but rather the protocolized linkage between rapid CT access, early physiologic risk stratification, predefined decision triggers, and definitive hemorrhage-control pathways. Implementation would likely require immediate or near-immediate CT access without delaying lifesaving bedside interventions, coordinated trauma team activation, rapid image interpretation, attending trauma surgeon–led decision-making, and reliable interventional radiology and operating room readiness. Centers without these capabilities should not interpret CTFR as support for routine CT before stabilization; instead, any adaptation should ensure that CT-to-intervention pathways can be executed safely and without delaying hemorrhage control.

In our matched cohort, CTFR was associated with less frequent laparotomy (5.6% vs. 11.7%) and a shorter time to needle among patients undergoing endovascular hemostasis. Accordingly, the shorter time to first hemostatic intervention may have been driven primarily by earlier endovascular hemostasis and earlier modality selection rather than by faster operative incision times. Together, these findings—less frequent laparotomy and greater use of pelvic angioembolization—are consistent with a possible shift toward earlier anatomy-directed endovascular hemorrhage control after immediate WBCT, in line with prior hybrid ER reports.[11] One hypothesis is that immediate WBCT may reduce diagnostic uncertainty

by more rapidly distinguishing pelvic from intraperitoneal hemorrhage, thereby facilitating earlier modality selection. In contrast, in conventional workflows, sonographic free fluid in hemodynamically unstable pelvic fractures can lead to emergent laparotomy when the bleeding source is uncertain.[21] Our findings are consistent with the possibility that immediate WBCT may help clarify the bleeding compartment earlier and support triage toward targeted pelvic angioembolization.

Limitations

This study has limitations. First, residual confounding may remain despite propensity score matching, and exclusion of JTDB patients with missing covariates may introduce selection bias. Because time-to-hemostasis was evaluated only among patients receiving hemostatic intervention, conditioning on postexposure events may introduce selection bias. CTFR-related adverse events during scanning were not systematically captured, precluding formal safety assessment. Because the registry lacks comparable bedside safety exclusions, and activation criteria are partly clinician-judged, activation rates and exclusions could not be reproduced in JTDB. This may have introduced selection bias, including potential bias favoring CTFR if patients requiring immediate bedside stabilization or judged unsafe for immediate CT were excluded from the CTFR pathway but remained eligible in the registry comparator. Second, this study was conducted at a single high-resource CT-TRR center and compared with a heterogeneous national registry cohort. Although propensity score matching balanced measured patient-level variables, the JTDB does not capture detailed center-level information on trauma center designation, staffing, trauma team experience, interventional radiology readiness, operating room access, CT-TRR availability, or institutional CT-to-intervention workflow. Accordingly, we could not determine how many comparator centers had CT-TRR or comparable CT-first capabilities; inclusion of such centers in the comparator cohort may have attenuated between-group differences, but also limits attribution of the findings specifically to the CTFR protocol. Therefore, these infrastructure variables could not be incorporated into the propensity score model or outcome models, and residual system-level confounding cannot be excluded. The observed associations may reflect the combined contribution of the CTFR protocol, integrated infrastructure, and trauma center maturity rather than the isolated effect of CT-first imaging alone. To partly address this concern, we conducted sensitivity analyses restricting the JTDB comparator to high-volume facilities, which yielded directionally consistent results; however, hospital volume does not fully substitute for detailed center-level infrastructure data. Third, mortality analyses were limited by sample size; therefore, survival effects require evaluation in larger multi-institutional studies. Finally, the direct shock subgroup analysis was performed within the matched cohort using a parsimonious adjustment model, whereas Figure 2 presents interaction estimates; therefore, slight differences in effect size are expected despite consistent overall interpretation. These subgroup findings should be interpreted as exploratory and require confirmation in larger studies.

CONCLUSIONS

Implementation of CTFR was associated with lower 24-hour RBC transfusion requirements, shorter time to hemostatic intervention, and fewer laparotomies compared with a matched JTDB cohort. These associations appeared more pronounced and potentially more clinically relevant among patients presenting with shock, although CTFR should be interpreted as a system-level strategy and multi-institutional studies are needed to evaluate generalizability and survival effects.

AUTHORSHIP

S.M. participated in conception, study design, and drafting of the manuscript. S.M., S.S., and M.S. participated in data acquisition. S.M., M.A., and T.F. participated in data analysis and interpretation. S.M., M.A., T.F., M.S., and S.S. participated in critical revision. All authors participated in final approval.

DISCLOSURE

All authors have completed and submitted JTACS Conflict of Interest Disclosure forms, which are provided as Supplemental Digital Content, http://links.lww.com/TA/F542.

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