

# Clinical outcomes and opportunities for improvement across modified trauma quality improvement cohorts: a registry-based cohort study

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## Research Article

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# Abstract

## Background

The American College of Surgeons Trauma Quality Improvement Program (TQIP) relies primarily on risk-adjusted mortality benchmarking, yet mortality alone may overlook non-fatal morbidity and care-process failures, particularly in cohorts where outcomes are driven by injury severity. This study aimed to analyse and compare 30-day mortality, cause of death, functional outcomes as measured by the Glasgow Outcome Scale (GOS), and opportunities for improvement (OFIs) identified through peer review across modified TQIP cohorts.

## Methods

Registry-based cohort study of 8,298 trauma patients at Karolinska University Hospital (2013–2023), classified into four modified TQIP cohorts: isolated severe TBI, blunt multisystem with TBI, blunt multisystem without TBI, and penetrating truncal injury. Remaining patients, predominantly lower-severity, formed a non-TQIP reference cohort. Cohort associations with mortality, unfavourable GOS, and OFI were assessed using cumulative sequential logistic regression (reference: non-TQIP cohort), progressively adjusting for patient characteristics, physiological status, and care processes, pooled across 20 imputed datasets. Cause of death, GOS levels, and OFI categories were compared using multinomial logistic regression.

## Results

All TQIP cohorts had elevated odds of mortality and unfavourable GOS. The blunt multisystem cohorts had the highest OFI rates (10.6% with TBI, 16.9% without) and were the only cohorts with elevations across all OFI categories, including potentially preventable deaths and clinical judgement errors. Care-process adjustment (e.g. emergency interventions, time to radiology, care level) reduced their mortality ORs, suggesting potentially modifiable processes. Isolated severe TBI had the highest mortality (52.8%) but no OFI excess, suggesting outcomes driven by primary injury rather than care-process failures. Penetrating truncal injury showed high, potentially preventable, haemorrhage-related mortality but favourable functional recovery in survivors after care-process adjustment.

## Conclusions

Outcome profiles differed across TQIP cohorts and were not captured by mortality alone: isolated severe TBI had the highest mortality but no OFI excess, while the blunt multisystem cohorts had the greatest OFI burden and potentially preventable deaths despite lower mortality. Penetrating truncal injury showed high haemorrhage-related mortality but excellent functional recovery in survivors. Integrating functional

outcomes and structured peer review alongside mortality identifies cohorts amenable to quality improvement and reveals cohort-specific processes to target.

## Background

Trauma remains a leading cause of death and disability worldwide, causing an estimated 4.5 million deaths in 2023 (1) and the primary cause of death among individuals aged 15 to 49 (2). To account for the heterogeneity of the trauma population, the American College of Surgeons (ACS) Trauma Quality Improvement Program (TQIP) defines clinically relevant high-risk cohorts for risk-adjusted mortality benchmarking within and across centres (3–5). In Sweden, cohort-specific benchmarking has confirmed that mortality differences between hospital types are concentrated in specific subgroups rather than reflecting uniform variation in care (6).

However, mortality alone does not distinguish non-preventable from potentially preventable deaths, nor does it capture non-fatal morbidity (7, 8). Functional outcome measures such as the Glasgow Outcome Scale (GOS) address this gap by capturing morbidity among survivors, though GOS is a crude measure, originally developed for traumatic brain injury with follow-up at 6–12 months (9, 10). Structured peer review complements mortality and functional outcome by identifying opportunities for improvement (OFIs), preventable errors such as delays in treatment, clinical judgement errors, or missed diagnoses (11–13). These cohorts are likely to differ not only in expected outcomes but also in the nature of care-process failures and the clinical challenges amenable to intervention.

Characterising these cohort-specific outcome profiles requires combining mortality with measures of morbidity and process errors, yet no previous study has done so across TQIP cohorts. This study aimed to analyse and compare 30-day mortality, cause of death, functional outcomes as measured by the GOS, and OFIs identified through peer review across modified TQIP cohorts.

## Methods

### Study design

We conducted a registry-based cohort study including all trauma patients recorded in both the Karolinska University Hospital trauma registry and the Karolinska trauma care quality database from 1 January 2013 to 28 February 2023. Ethical approval was obtained from the Swedish Ethical Review Authority.

### Setting and participants

The Karolinska University Hospital in Solna is a level 1 equivalent trauma centre, managing approximately 1,200 trauma patients annually (14). The hospital's trauma registry, part of the Swedish Trauma Registry (SweTrau), includes all patients admitted with trauma team activation, regardless of

Injury Severity Score (ISS), and those without activation but with ISS > 9. The registry records data on vital signs, timing, injuries, interventions, demographics, survival status, and functional outcome at discharge from the acute care hospital using the Glasgow Outcome Scale, following the Utstein template (15). The trauma care quality database captures peer review related data, including audit filters, identified OFIs and their categories, cause of death classifications, and decisions on preventable deaths.

Since 2017, a structured peer review process has been in place: a specialised nurse screens all trauma cases using audit filters (12, 16) and clinical judgement to flag potential OFIs. A second nurse conducts in-depth reviews of flagged cases, which are then assessed in a multidisciplinary peer review conference held bimonthly. All deaths, including those dead on arrival (DOA), undergo separate review to determine whether the death was trauma-related, classify cause of death, and evaluate preventability. Prior to 2017, the review process was informal and clinician-driven. A full description of the review pathway has been published previously (13).

## **Eligibility criteria**

All patients screened for OFIs in the trauma registry and care quality database during the study period were eligible. Patients under 15 years of age were excluded as they follow separate clinical pathways, and are not included in the peer review process.

## **Outcomes**

There were three primary outcomes: 1) 30-day all-cause mortality (binary: dead within 30 days of hospital arrival versus alive), 2) unfavourable functional outcome at discharge using the Glasgow Outcome Scale (GOS), dichotomised as unfavourable (GOS 1–3: death, vegetative state, or severe disability) versus favourable (GOS 4–5: moderate disability or good recovery), and 3) the presence of any OFI (binary: yes/no) as determined by consensus during the multidisciplinary peer review conference. Opportunities for improvement were defined as preventable errors in patient care that negatively impact outcomes or deviate from established safe clinical practices, assessed relative to optimal care at the study hospital, the Karolinska University Hospital.

Secondary outcomes included: cause of death (classified through peer review as traumatic brain injury, bleeding, multi-organ failure, or other causes), individual GOS levels (death/vegetative state, severe disability, moderate disability, good recovery), and OFI categories. These were grouped into six categories for analysis: missed diagnosis, delay in treatment, clinical judgement errors, inadequate protocols, inadequate resources, and potentially preventable deaths. Definitions, illustrative examples, and subcategory frequencies for each OFI category are provided in Additional file 1. This categorisation follows previously published work at our centre (12) and comparable external classifications (17).

## **Cohort classification**

Patients were classified into four mutually exclusive TQIP-based cohorts following previously published definitions (4, 6): (1) blunt multisystem without TBI, defined as blunt trauma with two or more body regions with Abbreviated Injury Scale (AIS)  $\geq 3$ , without severe TBI; (2) blunt multisystem with TBI, same criteria but including concomitant severe TBI (AIS  $\geq 3$  head injury); (3) isolated severe TBI, defined as severe TBI with emergency department GCS  $\leq 8$  or prehospital intubation with GCS  $\leq 8$ , without multisystem trauma; and (4) penetrating truncal injury, defined as penetrating injuries to central regions (neck, thorax, abdomen) with AIS  $\geq 3$ . Patients not meeting any criteria were classified as a non-TQIP reference cohort. This group predominantly comprises patients with lower injury severity who were evaluated by the trauma team but did not meet the TQIP thresholds. The reference cohort thus represents the lower-severity end of the trauma spectrum and serves as a clinically meaningful baseline for outcome comparisons.

## Statistical analysis

Baseline characteristics were summarised using medians with interquartile ranges for continuous variables and frequencies with percentages for categorical variables. Associations between cohort membership and each of the three primary outcomes were assessed across four sequential logistic regression models with the non-TQIP cohort as reference level in all models: the unadjusted model estimated crude odds ratios; the patient characteristics model adjusted for age, American Society of Anesthesiologists (ASA) physical status, and sex; the physiological model additionally adjusted for systolic blood pressure and respiratory rate; and the care-process model further adjusted for intubation status, emergency procedures, care level, time to emergency procedure, and time to CT. Injury severity (New Injury Severity Score (NISS), AIS) and Glasgow Coma Scale (GCS) were excluded as covariates because they define the cohort classification. The reduction between the physiological and care-process models instead isolates the component of excess risk potentially mediated by modifiable care processes.

Secondary outcomes were assessed using multinomial logistic regression with cohort as the independent variable. Three separate models were fitted: cause of death, modelled as a multinomial outcome where each cause (TBI, bleeding, multi-organ failure, other) was compared against survival as the reference level; GOS level (reference level: good recovery); and OFI category (reference level: no OFI). Covariates from the primary models were not included in the multinomial models due to the rarity of some outcome levels. For the OFI category inadequate protocols, which occurred only in two cohorts, a separate binomial logistic regression was performed.

Sample size adequacy was evaluated using Riley et al. (18). The care-process model included 26 parameters with Cox-Snell  $R^2$  values of 0.238 (mortality), 0.394 (unfavourable GOS), and 0.042 (OFI), yielding minimum required sample sizes of 1,003, 549, and 5,377 respectively (target shrinkage 0.9), all exceeded by our sample of 8,298. Results are reported as odds ratios (OR) with 95% confidence intervals. Analyses were conducted in R (version 4.5.3) (19), with  $p < 0.05$  indicating statistical significance.

# Data handling

Missing predictor data were imputed using multiple imputation by chained equations (MICE, 50 iterations) using predictive mean matching (continuous variables and GCS, treated as pseudo-continuous), proportional odds logistic regression (ordinal), logistic regression (binary), and polytomous logistic regression (nominal) (20). Structural missing values were recoded as explicit factor levels prior to imputation: continuous time-to-event variables were decomposed into a binary indicator (procedure performed yes/no; CT performed yes/no) and an ordered categorical variable for elapsed time (0–30, 31–60, 61–120, > 120 minutes for emergency procedures; 0–20, 21–30, 31–60, 61–120, > 120 minutes for CT), with patients who did not undergo the procedure or CT assigned to a “no procedure” or “no CT” level. Outcome variables were included as predictors but not imputed (20, 21). The maximum fraction of missing information (FMI) was 15.2%, yielding a minimum of  $m \geq 16$  (22); we used  $m = 20$  imputed datasets. All models were pooled using Rubin’s rules (23). Convergence was assessed visually via trace plots and quantitatively using the potential scale reduction factor (R-hat) (24) (Additional file 8). All R-hat values were below 1.05 at the final iteration.

## Results

### Participants

This study included 8,298 patients, of whom 5,748 (69.3%) were male. Patients were categorised into the five cohorts: isolated severe TBI ( $n = 246$ , 3%), blunt multisystem with TBI ( $n = 587$ , 7.1%), blunt multisystem without TBI ( $n = 427$ , 5.1%), penetrating truncal injury ( $n = 423$ , 5.1%), and the non-TQIP reference cohort ( $n = 6,615$ , 79.7%). The median age was 43 (IQR 27, 61) years, with the youngest cohort being patients with penetrating truncal injury (28 (IQR 21, 41) years) and the oldest being patients with isolated severe TBI (59 (IQR 38, 74) years). The median NISS was 11 (IQR 3, 22). The study flowchart is presented in Figure 1. Full baseline characteristics and outcome distributions are shown in Table 1.

**Table 1: Baseline characteristics and outcomes by trauma cohort.**

### Mortality and cause of death

Overall, 727 patients (8.8%) died within 30 days. Mortality ranged from 15.9% in blunt multisystem without TBI to 52.8% in isolated severe TBI, compared with 4.1% in the non-TQIP reference cohort (Table 1). Excluding DOA, the largest absolute mortality reduction was in penetrating truncal injury (21.3% to 9.2%) and the smallest in isolated severe TBI (52.8% to 49.6%). Unadjusted odds of mortality were significantly increased in all TQIP cohorts and remained elevated after sequential adjustment for patient characteristics, physiological status, and care processes (Figure 2, Additional files 2–5). The care-process adjustment step reduced ORs across all cohorts, most notably in blunt multisystem without TBI (Figure 2).

Among deaths with a classified cause, TBI was the most common overall, followed by bleeding and multi-organ failure. Cause-of-death patterns differed across cohorts (Figure 3, Additional file 6). Deaths in the TBI-associated cohorts were predominantly TBI-related, particularly in isolated severe TBI (94%), whereas blunt multisystem without TBI showed a heterogeneous distribution across bleeding, multi-organ failure, and other causes. In penetrating truncal injury, bleeding was the dominant cause.

## Functional outcomes

Unfavourable GOS (< 4) at discharge was recorded in 2,896 patients overall. Among the TQIP cohorts, rates ranged from 35% in penetrating truncal injury to 91.1% in isolated severe TBI, compared with 25.8% in the reference cohort (Table 1). Compared to the non-TQIP cohort, all TQIP cohorts had increased unadjusted odds of unfavourable GOS, with the highest OR in isolated severe TBI (29.23 (18.80–45.44)). These associations persisted across adjustment levels, though ORs were substantially reduced after adjusting for care processes (Figure 2, Additional files 3, 5). The OR for penetrating truncal injury crossed below 1.0 after adjusting for care processes (OR 0.87 (0.65–1.17)), while isolated severe TBI remained markedly elevated (OR 10.86 (6.68–17.66)). Among survivors, penetrating truncal injury had the highest proportion of good recovery (13.9%), whereas severe disability predominated in the blunt multisystem cohorts (Additional file 7).

## Opportunities for improvement

Opportunities for improvement were identified in 493 patients (5.9%), with rates highest in the blunt multisystem cohorts (Table 1). Blunt multisystem without TBI had the highest unadjusted OFI elevation (OR 4.11 (3.11–5.43)), persisting across all adjustment levels, and was the only cohort with significantly elevated odds across all OFI categories, including inadequate protocols, which was not significantly elevated in any other cohort (Figure 2, Table 2). Isolated severe TBI showed no significant OFI excess despite the highest mortality, though the category analysis identified elevated odds of delays in treatment. In penetrating truncal injury and blunt multisystem with TBI, clinical judgement errors and potentially preventable deaths were the predominant categories, with blunt multisystem with TBI showing the highest odds of potentially preventable deaths across all cohorts (Table 2). Each TQIP cohort thus displayed a distinct outcome profile across mortality, functional outcomes, and OFI categories (Figure 3).

**Table 2: OFI categories by TQIP cohort.**

## Discussion

This study examined 30-day mortality, functional outcome (GOS), and OFIs across modified TQIP cohorts. All TQIP cohorts carried substantially elevated risks of both mortality and unfavourable functional outcomes that persisted after sequential adjustment. Care-process adjustment reduced ORs across all cohorts, but this statistical signal converged with elevated OFI rates only in the blunt multisystem cohorts, identifying them as the populations where quality improvement efforts have the greatest potential.

Blunt multisystem without TBI had the highest OFI rate, the only significant elevations across all OFI categories, and a reduced mortality OR after adjusting for care processes (OR 2.4 to 1.69) consistent with care-process mediation. Despite a median NISS of 34, these patients presented with apparent physiological stability: a median GCS of 15, median SBP of 125 mmHg, median respiratory rate of 18 breaths/min, and the lowest ICU admission rate among the TQIP cohorts (46%). Yet their multisystem injuries require complex interventions across multiple organ systems — 59% underwent emergency procedures, spanning the widest range of intervention types across cohorts (Table 1) — creating more decision points where errors in clinical judgement, protocols, and resource allocation can occur. This complexity, combined with the apparent physiological stability, may compound the risk by leading to underestimation of injury severity and insufficient escalation of care, ultimately resulting in the highest rate of potentially preventable deaths (25,26). The cause-of-death distribution in this cohort was notably heterogeneous, with multi-organ failure featuring prominently alongside bleeding, consistent with a pattern in which initially stable patients deteriorate over time without adequate monitoring. That multi-organ failure was a substantial cause of death despite this low rate of ICU admission, combined with elevated OFIs in care level and inadequate resources, suggests that earlier escalation to higher-level care could mitigate preventable deterioration.

Beyond mortality, the blunt multisystem cohorts carried a disproportionate burden of unfavourable functional outcomes, signalling morbidity and rehabilitation needs that mortality statistics do not capture. Elevated odds of missed injuries may contribute to both delayed deterioration and the high proportion of patients discharged with severe disability. Longitudinal data from the same centre show that blunt multisystem without TBI remains the only cohort with persistently elevated OFI risk despite an overall decrease in OFI incidence over time (27). Blunt multisystem with TBI showed a similar but less pronounced pattern, with OFIs predominantly driven by clinical judgement errors and potentially preventable deaths, consistent with the complexity of managing concurrent neurosurgical and general surgical priorities (28).

In contrast, isolated severe TBI showed no significant OFI excess despite the highest mortality (53%) and unfavourable GOS rate. Adjusting for care processes reduced ORs but, in the absence of elevated OFI rates, this likely reflects necessary care responses (ICU admission and intubation) rather than modifiable quality gaps. Deaths were predominantly TBI-related (94%), and despite the highest mortality this cohort had the lowest proportion of DOA among the TQIP cohorts (8 [3.3%]), reflecting injuries that are not acutely lethal but progress over hours to days — consistent with the paradoxical increase in mortality OR after adjusting for physiological status, which confirms that preserved haemodynamics on presentation

do not exclude severe brain injury. Nevertheless, the category analysis identified significantly elevated odds of delays in treatment. Although CT times were the fastest across cohorts, time to definitive neurosurgical intervention was prolonged, suggesting that reducing this interval is the principal modifiable target in a cohort where the primary brain injury otherwise largely determines outcomes. The absence of elevated odds of potentially preventable deaths aligns with the preventable death literature, in which severe TBI is consistently classified as the predominant cause of non-preventable trauma mortality (17,29), and with prognostic studies identifying initial neurological status as the dominant outcome determinant (30,31).

Penetrating truncal injury presented a distinctive bimodal pattern of high mortality alongside excellent functional recovery in survivors, reflecting injuries where haemorrhage is either rapidly lethal, as evidenced by the highest proportion of DOA (51 [12.1%]), or surgically controllable (32). After adjusting for care processes, the OR for unfavourable GOS crossed below 1.0, suggesting that fast, definitive surgical haemostasis, with the shortest time to intervention across cohorts, drives favourable recovery in survivors. The mortality OR increased after adjusting for patient characteristics (from 6.35 to 13.72), indicating that the young, low-comorbidity profile of these patients masks the true lethality of the injuries; once this demographic advantage is accounted for, the excess mortality is even greater. Although the overall OFI elevation was reduced after full adjustment, the OFI category analysis revealed significantly elevated odds for clinical judgement errors and potentially preventable deaths. Bleeding was the dominant cause of death, and haemorrhage is consistently identified as the leading cause of potentially preventable trauma mortality (17,33,34). The co-occurrence of haemorrhage-dominated deaths with elevated odds of clinical judgement errors and potentially preventable deaths suggests that care failures in this cohort may carry disproportionate consequences (35).

The inverse relationship between mortality and OFI risk challenges the use of mortality as a sole quality indicator (36). The TQIP framework uses risk-adjusted mortality benchmarking across centres to identify underperformance (4), but this approach can detect that a cohort underperforms without characterising why. As illustrated by the blunt multisystem without TBI cohort, care-process failures that manifest primarily as morbidity rather than death may go unrecognised, consistent with evidence from our centre that opportunities for improvement concentrate in physiologically stable patients who survive (37) and that mortality-based case selection fails to capture them (13). Gruen et al. (33) identified distinct error patterns contributing to trauma mortality concentrated in specific phases of care; our findings extend this by demonstrating that the distribution of OFIs across cohorts is not proportional to mortality. Preventable death reviews remain valuable (7), but mortality alone does not capture the full spectrum of care-process failures. Integrating functional outcomes and process indicators alongside mortality is necessary for comprehensive quality assessment.

## Strengths and limitations

This study benefits from a large cohort spanning 10 years with structured peer review and simultaneous assessment of mortality, functional outcomes, and process errors across standardised trauma cohorts. The sequential modelling framework introduces care-process variables in the final step, which enabled us to estimate the excess risk potentially attributable to modifiable processes. Additionally, the peer review process may itself contribute to quality improvement through a Hawthorne effect: the awareness that all cases are subject to systematic screening and multidisciplinary peer review can drive behavioural change among all healthcare personnel, independent of the specific errors identified.

Several limitations merit consideration. The OFI identification relies on audit filters and nurse-led case reviews with limited individual predictive ability (16,38), and no combination of filters captures all care-process errors. Observed OFI rates may therefore partly reflect detection bias, where cohorts with previously identified high-impact OFIs receive sustained scrutiny while others may be under-scrutinised; a low observed OFI rate should not be interpreted as confirmation that care-process errors are absent.

Additionally, some OFI categories had limited case numbers, particularly in the smaller cohorts, and the resulting effect estimates should be interpreted as signals identifying areas for quality improvement attention rather than precise measures of magnitude. The GOS was assessed at discharge from the primary admitting hospital; discharge includes transfer to another facility, home, or death. Discharge GOS therefore reflects functional status at a variable and often early timepoint rather than at the standardised 6–12 month follow-up for which the scale was designed, and may over- or underestimate long-term disability.

When comparing DOA-excluded mortality rates with national Swedish data (NISS > 15) (6), isolated severe TBI (49.6% versus 50%) and blunt multisystem without TBI (10.8% versus 9%) were comparable, supporting the external validity of findings in these cohorts. Blunt multisystem with TBI mortality was higher (21.8% versus 12%), likely reflecting the injury severity at a level 1 referral centre, and penetrating trauma mortality was lower (9.2% versus 20%), consistent with high-volume surgical haemostasis capacity. These differences should be considered when generalising to other settings.

## Conclusion

Outcome profiles differed markedly across TQIP cohorts and were not captured by mortality alone: isolated severe TBI had the highest mortality yet one of the lowest OFI rates, while the blunt multisystem cohorts, particularly without TBI, had the greatest OFI burden and potentially preventable deaths despite lower mortality. Penetrating truncal injury showed high haemorrhage-related mortality but excellent functional recovery in survivors. Risk-adjusted mortality benchmarking alone cannot characterise these differences. Integrating functional outcomes and structured peer review alongside mortality not only identifies which cohorts are amenable to quality improvement but also reveals potential cohort-specific processes to target.

## Abbreviations

ACS: American College of Surgeons; ASA: American Society of Anesthesiologists; CI: Confidence interval; CT: Computed tomography; GCS: Glasgow Coma Scale; GOS: Glasgow Outcome Scale; ICU: Intensive care unit; ISS: Injury Severity Score; NISS: New Injury Severity Score; OFI: Opportunity for improvement; OR: Odds ratio; SweTrau: Swedish Trauma Registry; TBI: Traumatic brain injury; TQIP: Trauma Quality Improvement Program

## Declarations

The requirement for informed consent was waived by the Swedish Ethical Review Authority (approval numbers 2021-02541 and 2021-03531) due to the use of pseudoanonymised registry data collected as part of routine care and supported under Chapter 7 of the Swedish Patient Data Act (2008:355). Participants had the right to request removal of their data from the registry at any time.

## Ethics approval and consent to participate

This study was approved by the Swedish Ethical Review Authority (approval numbers: 2023-02975-02, 2021-03531, 2021-02541). The need for informed consent was waived by the ethical review authority due to the retrospective registry-based design.

## Consent for publication

Not applicable.

## Availability of data and materials

The datasets analysed during the current study are not publicly available due to containing sensitive patient information protected under Swedish law. Data access requests can be directed to the corresponding body at Karolinska University Hospital and are subject to approval by the Swedish Ethical Review Authority.

## Competing interests

The authors declare that they have no competing interests.

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# Authors' contributions

Conceptualisation: JA, MGW. Study design: JA, MGW, LS, OB. Data acquisition: JA, MGW. Statistical methodology and analysis: JA, MJ. Data interpretation: JA, MGW, LS, OB, MJ. Peer review process expertise: LS, OB. Funding acquisition: JA, MGW. Supervision: MGW. Drafting the manuscript: JA. Critical revision: all authors. All authors read and approved the final manuscript.

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Tables

Table 1 and 2 are available in the Supplementary Files section.

Figures

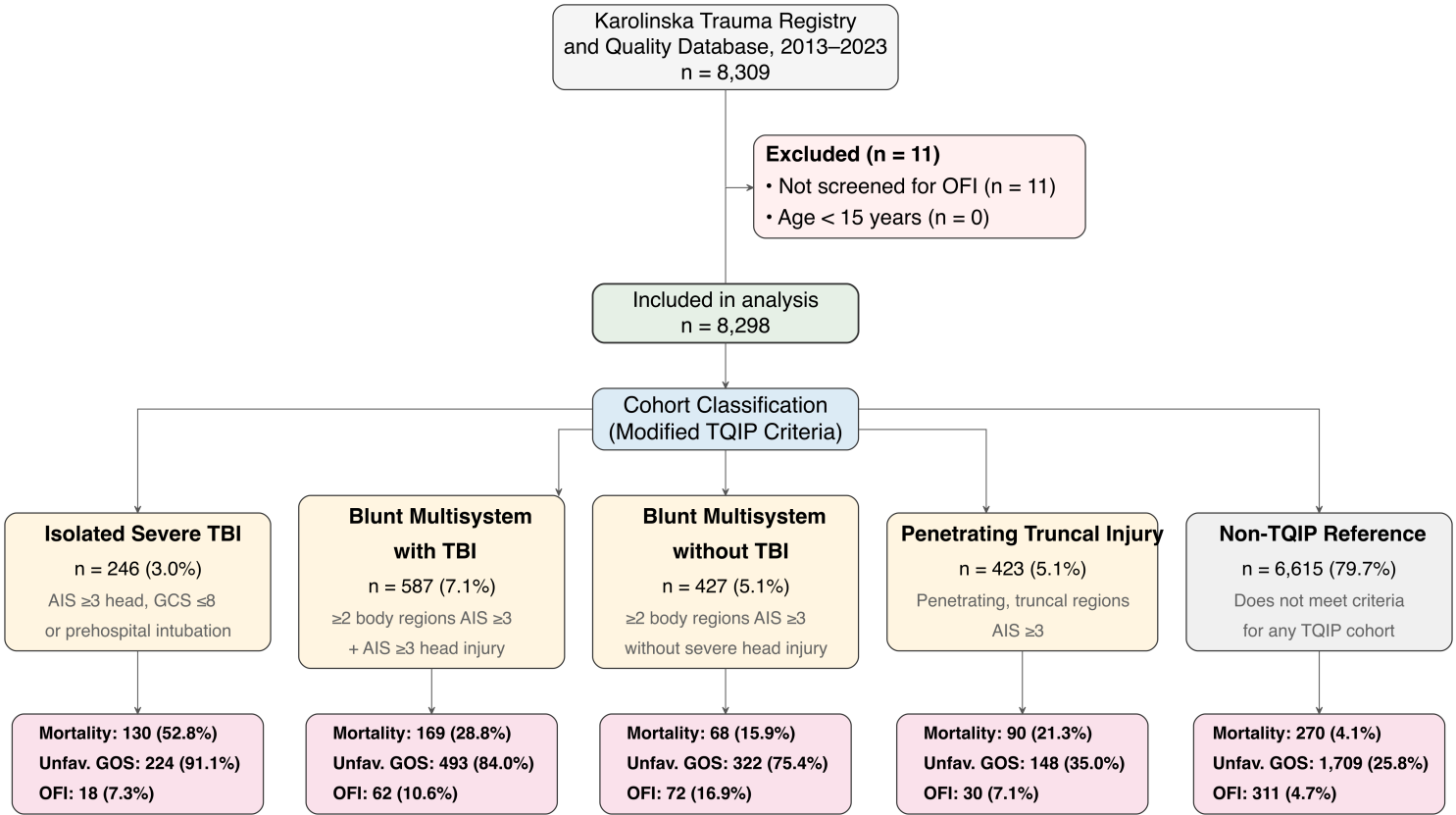


Figure 1

Study flowchart showing patient inclusion, cohort classification, and outcomes. Cohort percentages = % of total included patients. Outcome percentages = % within each cohort.

AIS = Abbreviated Injury Scale; GCS = Glasgow Coma Scale; GOS = Glasgow Outcome Scale; OFI = Opportunity for Improvement; TBI = Traumatic Brain Injury; TQIP = Trauma Quality Improvement Program.

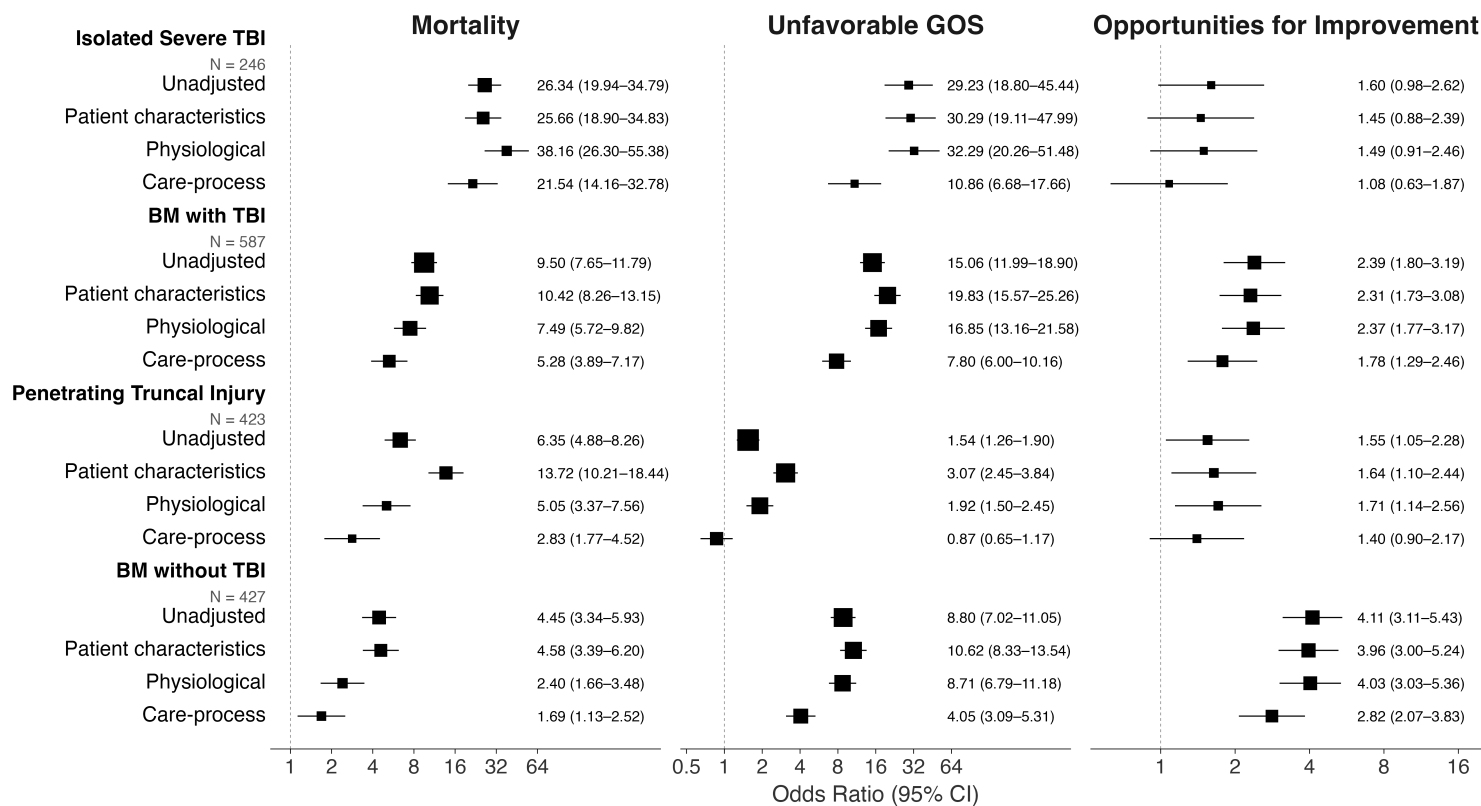


**Figure 2**

Forest plot of odds ratios from cumulative sequential logistic regression models for 30-day mortality, unfavourable GOS, and opportunities for improvement. Each model retains all covariates from the preceding step.

*Reference: Non TQIP-cohort. Each model includes all covariates from previous steps. Unadjusted: cohort only. Patient characteristics: + age, ASA, sex. Physiological: + SBP, respiratory rate. Care-process: + intubation, emergency procedure, care level, time to procedure, time to CT.*

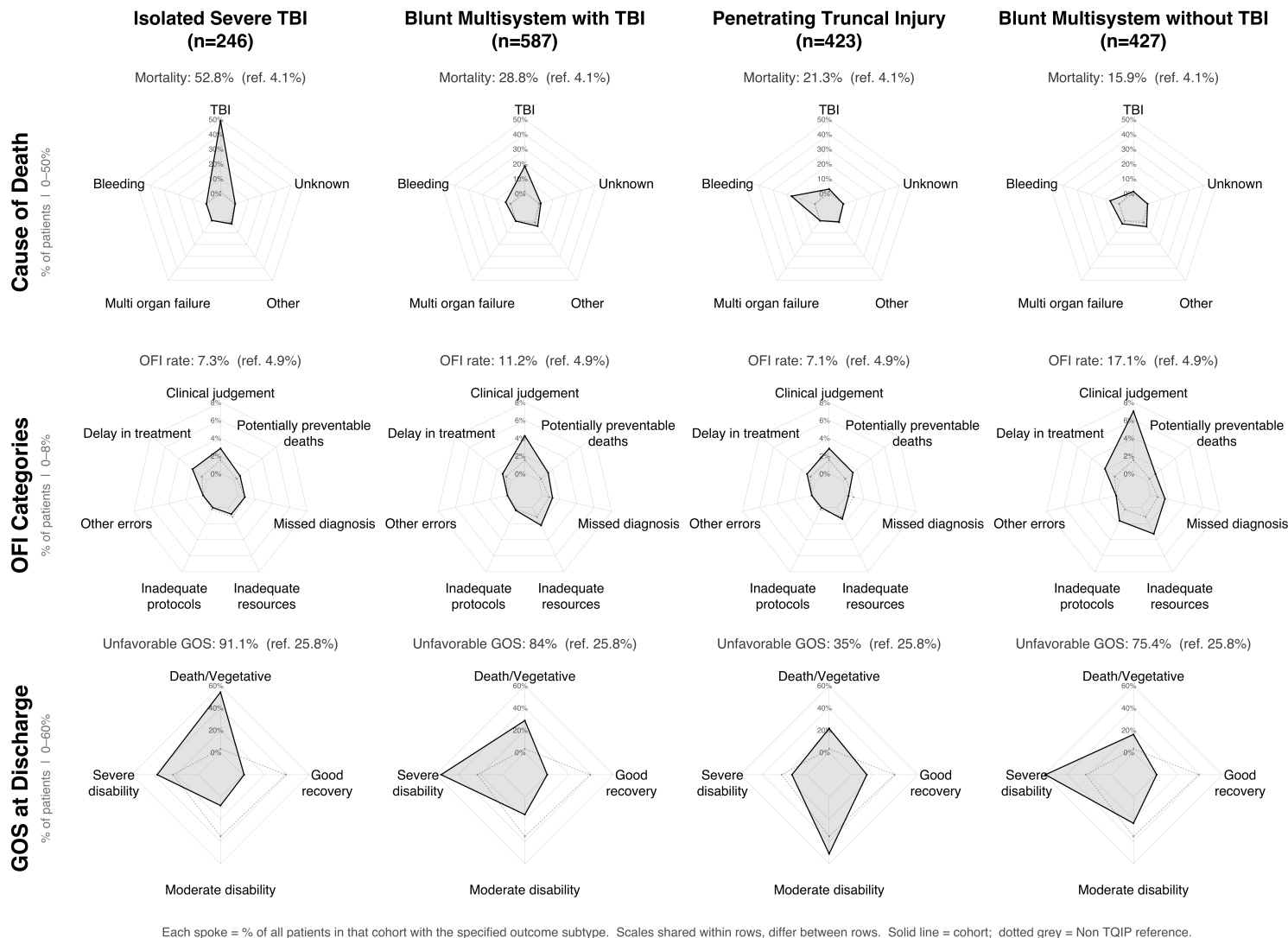


**Figure 3**

Radar charts of outcome category distributions by TQIP cohort. Percentage of patients in each cohort with specific outcome categories for cause of death, OFI categories, and GOS levels.

*Solid lines represent TQIP cohorts; dotted grey lines represent the non-TQIP reference.*

## Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [supplementarysjtrem.docx](#)
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