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Prehospital Determinants of Emergency Department Mortality in Adult Trauma at a South Sumatran Tertiary Referral Centre: A Cross-Sectional Study with Penalised-Likelihood Reanalysis

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ABSTRACT

Background: The prehospital phase is considered a modifiable determinant of trauma survival, but evidence from Indonesian tertiary centres with under-developed emergency medical services is scarce.

Objective: To examine whether first-responder type, scene time and transport mode were associated with emergency department mortality in adult trauma.

Methods: This cross-sectional study included 34 consecutive adult trauma patients presenting to the Emergency Department of Dr. Mohammad Hoesin General Hospital, Palembang. Associations were assessed using Fisher exact tests, exact conditional odds ratios (OR), Newcombe risk differences, Firth penalised logistic regression and the originally specified multivariable model. Minimum detectable effects and post-hoc power were calculated.

Results: Mortality was 20.6% (7/34; 95% CI 10.3–36.8). Death occurred in 6/24 patients with scene time >60 minutes versus 1/10 with shorter scene time (OR 2.92, 95% CI 0.28–153.08; p = 0.644), in 1/2 transported by ambulance versus 6/32 otherwise (OR 4.09, 95% CI 0.05–352.85; p = 0.374), and in 1/2 attended by a medical first responder versus 6/32 attended by lay rescuers (p = 0.374). No exposure was independently associated with mortality. The smallest detectable OR was 12.57; realised power was 12.6–23.1%.

Conclusion: No prehospital factor was significantly associated with mortality, but the wide intervals remain compatible with clinically important effects. The findings are uninformative rather than negative and support a prospective, adequately powered, biomarker-augmented regional trauma registry.

1. Introduction

Injury is among the leading causes of premature death worldwide and its burden falls disproportionately on adults of working age. A recent systematic review and meta-analysis of trauma transport places the global toll at approximately five million deaths each year.[1] That burden is concentrated in low- and middle-income countries, where prehospital systems are fragmented or absent: an international survey of prehospital providers found that delay in these settings is driven primarily by the absence of dispatch systems, untrained first responders and lack of transport rather than by distance,[2] and a population-based road-traffic injury registry showed that most deaths occur before the patient ever reaches a hospital.[3] Indonesia is no exception. Successive institutional series from the Emergency Department of Dr. Mohammad Hoesin General Hospital, the tertiary referral centre for South Sumatera, have documented a trauma case-mix dominated by blunt mechanisms in young adult men,[4,5] and mortality after blunt thoracic injury at this centre has been shown to track anatomical severity closely.[6,7]

The prehospital phase is conventionally regarded as the most modifiable segment of the trauma pathway, and the golden hour — the proposition that definitive haemorrhage control and airway protection delivered within sixty minutes of injury interrupt an otherwise irreversible physiological cascade — remains the organising principle of trauma systems worldwide. The geographical structure of prehospital delay is now well described: a national Swedish cohort found that prehospital time intervals varied several-fold across population-density strata,[8] and mass-casualty data confirm that extended prehospital times are associated with adverse outcome.[2] If elapsed time were the operative variable, a system delivering most of its patients beyond sixty minutes should show a correspondingly high mortality.

The contemporary evidence is considerably more equivocal than the golden-hour construct implies. A large retrospective cohort found that the association between on-scene time and in-hospital mortality weakened substantially once the type and number of prehospital interventions were taken into account, so that what is done during the prehospital phase mattered more than how long it lasted.[9] A Norwegian national population-based study found that the markedly longer prehospital times of remote settings were not accompanied by a corresponding excess mortality after case-mix adjustment,[10] and a Southeast Asian cohort reported no independent association between scene time and mortality in major trauma.[11] The same pattern holds in paediatric practice.[12] Analyses of transport mode point in the same direction and add a specific warning: a nationwide Japanese study found that physician-staffed ambulance attendance was associated with *higher* mortality among hypotensive patients with prolonged prehospital stay,[13] a registry comparison found non-ambulance transport no worse than ground ambulance after adjustment,[14] and both a Swedish cohort and a systematic review with meta-analysis concluded that apparent modality effects reflect patient selection rather than the vehicle itself.[1,15] Contemporary risk stratification has moved accordingly towards physiological and composite anatomical-physiological measures.[16–18]

There is a biological reason why elapsed time is an imperfect surrogate for risk, and it is the reason a department of medical biology belongs on a surgical research team. The clock does not injure the patient; the cascade the clock imperfectly indexes does. Within minutes of energy transfer, hypoperfusion and tissue injury deplete fibrinogen and provoke an acute coagulopathy that independently predicts massive transfusion and death,[19] and that is already predictable from routinely available variables at emergency department arrival.[20] Disturbance of ionised calcium at arrival correlates with coagulopathy, transfusion requirement and mortality across large registries.[21] In parallel, shedding of the endothelial glycocalyx releases syndecan-1 into the circulation, where its concentration tracks injury burden and predicts organ failure and death.[22,23] Critically, this cascade is pharmacologically modifiable during transport: prehospital tranexamic acid produces a dose-dependent fall in syndecan-1,[24] its benefit after traumatic brain injury depends on how early it is given,[25] and the endothelial response to it varies with the patient's physiological reserve.[26] The rate at which an individual traverses the cascade is therefore governed by injury burden and reserve rather than by elapsed minutes — a principle confirmed clinically by the finding that frailty, not chronological age or time, governs short-term mortality in older multiple-trauma patients.[27]

Empirical data on prehospital determinants of trauma mortality in Indonesian tertiary referral hospitals remain sparse, and no regional trauma registry exists to generate them, although both the design and the cost of establishing one in a comparable low- and middle-income tertiary institution have now been described.[28,29] Dr. Mohammad Hoesin General Hospital receives trauma patients across a wide geographical catchment and a broad range of injury severity, and therefore offers a natural setting in which to characterise prehospital exposures and their relationship with early mortality. The present study was conducted by an interdisciplinary team drawn from the Department of Surgery, the Department of Orthopaedics and the Department of Biology of the Faculty of Medicine, Universitas Sriwijaya, combining operative,

musculoskeletal and molecular perspectives on a single clinical problem. Our objectives were, first, to describe the prehospital exposure profile of adult trauma patients presenting to this centre; second, to quantify the association between first-responder type, scene time and transport mode and emergency department mortality using effect estimates and compatibility intervals rather than significance testing alone; and third, to determine the precision actually achieved, so that the findings can be interpreted correctly and used to specify an adequately powered successor study.

2. Methods

2.1. Study design and reporting

This was an observational, analytical, hospital-based cross-sectional study. Exposure and outcome status were ascertained at a single point of observation, namely the patient's episode of care in the emergency department, using data abstracted from the medical record. No intervention was administered and no aspect of clinical care was altered for the purposes of the study. The manuscript is reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement for cross-sectional studies.[30] The complete participant pathway, from screening through to the analysed cohort, is presented in Figure 1.

2.2. Setting

The study was conducted in the Emergency Department of Dr. Mohammad Hoesin General Hospital, Palembang, the tertiary referral centre of the Faculty of Medicine, Universitas Sriwijaya, and the principal trauma receiving hospital for the province of South Sumatera. The department receives both direct-from-scene presentations and secondary referrals from primary and secondary facilities across the province, and its trauma case-mix has been characterised in several previous institutional series.[4–6] At the time of the study there was no province-wide physician-staffed emergency medical services system, no centralised dispatch for trauma, and no formal prehospital trauma registry.

2.3. Participants and eligibility

All adult patients presenting to the emergency department with a diagnosis of trauma during the study period were screened. Inclusion criteria were a clinical diagnosis of trauma and age 18 years or older. Exclusion criteria, specified before data collection and shown in Figure 1, were death before initial emergency department assessment (dead on arrival), because such patients cannot contribute an in-department outcome, and prior definitive management at another facility, because the prehospital exposures of interest could not then be attributed unambiguously. Records with incomplete documentation of any of the three prehospital exposures were also ineligible. Eligible patients were recruited by consecutive sampling until the target sample size was met, which minimises selection bias relative to convenience sampling.

2.4. Sample size

The sample size was calculated a priori with the standard formula for the comparison of two independent proportions, assuming a two-sided significance level of 5% ($Z_{\alpha/2}$ = 1.96) and 80% power (Z_{β} = 0.84). The anticipated mortality proportion among patients with high-risk prehospital exposure was set at 65% and that among the comparison group at 30%, values derived from the pre-study literature available to the investigators. This yielded a minimum of 31 patients, to which a 10% margin for incomplete records was added, giving a target of 34 patients. As quantified in Table 5 and in the Discussion, the mortality proportions actually observed were far lower than those assumed, with the consequence that the realised power was substantially below the nominal 80%; this discrepancy is reported explicitly rather than left implicit.

2.5. Variables and operational definitions

The dependent variable was mortality, defined as death of a trauma patient occurring from the time of arrival in the emergency department until departmental disposition, coded 0 for survived and 1 for died. Three prehospital exposures were specified a priori. *First-responder type* was the professional background of the person who provided the initial assistance at the scene, dichotomised as lay (non-medical) or medical/paramedical. *Scene time* was the interval from the traumatic event to the receipt of medical care at a health facility, dichotomised at the conventional golden-hour threshold as 60 minutes or less versus more than 60 minutes. *Transport mode* was the vehicle used to convey the patient to the health facility, dichotomised as ambulance or non-ambulance. Covariates recorded were age category, sex, principal injured body region, injury mechanism, incident location, Injury Severity Score (ISS) category, Glasgow Coma Scale (GCS) category and monotrauma versus multiple trauma; their categories and distributions are given in Table 1. ISS was categorised as minor (1–9), moderate (10–16), severe (17–24) or critical (≥25), and GCS as mild (13–15), moderate (9–12) or severe (3–8).

2.6. Data sources and measurement

Data were abstracted from the hospital medical record, which constituted the sole study instrument. Prehospital variables were taken from the triage and admission documentation completed at the point of arrival; injury severity scores were assigned from the documented anatomical diagnoses and the recorded first Glasgow Coma Scale. Abstraction was performed by a single investigator using a structured extraction sheet, and records were checked for completeness and internal consistency before analysis. No independent duplicate abstraction was undertaken, so no inter-rater agreement statistic is available. Complete data on all three exposures and on the outcome were available for every enrolled patient, as recorded in Figure 1, so no imputation was required.

2.7. Statistical analysis

Analyses were performed in IBM SPSS Statistics, with the exact and penalised-likelihood procedures described below conducted in Python 3.11 using SciPy 1.11. Categorical variables were summarised as counts and percentages and continuous variables as means with standard deviations, minima and maxima, as presented in Table 1 and Table 2. The proportion of deaths was accompanied by a Wilson score 95% confidence interval, which retains nominal coverage at small denominators.

Associations between each prehospital exposure and mortality were tested with the two-sided Fisher exact test. This choice was made before analysis on the basis that the expected count was anticipated to fall below five given the exposure prevalences observed at triage, and it was confirmed on inspection: two of the four expected counts were below five in every contingency table, as recorded in the footnote to Table 3. Pearson χ^2 and likelihood-ratio G^2 statistics are reported alongside for completeness but were not used for inference. Effect sizes were expressed in four complementary ways in Table 3 — the exact conditional maximum-likelihood odds ratio with its exact confidence limits, which does not rely on large-sample normality; the Haldane–Anscombe corrected odds ratio; the absolute risk difference with a Newcombe hybrid score interval computed without continuity correction; and the φ coefficient as a measure of association strength. Reporting several estimators is deliberate, because with cell counts as small as one the choice of estimator materially affects the interval; the estimators are not statistically independent and must not be read as mutual corroboration.

Because zero and near-zero cells produce sparse-data bias and inflate standard errors, each exposure was additionally modelled by univariable logistic regression with Firth's penalised likelihood, which applies a Jeffreys-prior penalty to remove first-order bias and guarantees finite estimates under separation.[31] Estimates were obtained by direct maximisation of the penalised log-likelihood with a quasi-Newton optimiser at a convergence tolerance of 10^{-6}, and confidence intervals by root-finding on the profile penalised likelihood at the 95% level rather than by Wald approximation. For a saturated logistic model on a 2 × 2 table the Firth point estimate is algebraically identical to the Haldane–Anscombe corrected odds ratio, so the two entries in Table 3 and Table 4 represent one estimate reported with two interval methods. The multivariable logistic regression model originally specified by the investigators, containing all three prehospital exposures simultaneously, is reported unchanged in Table 4; its coefficients have been re-expressed so that every odds ratio refers to the same exposure direction as the corresponding crude estimate, and the transformation is stated explicitly in that table's footnote.

Because a non-significant result is not evidence of absence, the precision actually achieved was quantified directly and is reported in Table 5 and Figure 4.[32] The primary precision statistic is the *minimum detectable odds ratio* at 80% power and a two-sided α of 0.05, given the realised group sizes and baseline risk, because it depends on the design rather than on the observed result. Post-hoc power at the observed effect size is also reported, but only as an accessible illustration: it is a monotone function of the p value and carries no information beyond it. A reverse fragility index was computed by reassigning outcome status within a fixed group size, in either exposure group, and taking the smallest number of reassignments at which the two-sided Fisher exact test reached $p < 0.05$. Model adequacy was assessed by the events-per-variable ratio. Two-sided p values are reported to three decimal places and interpreted as continuous measures of compatibility rather than as dichotomous decisions.

2.8. Ethics

This study received ethical approval from the Health Research Ethics Committee of the Faculty of Medicine, Universitas Sriwijaya, Palembang, Indonesia. The requirement for individual written informed consent was waived for this retrospective analysis of routinely collected medical-record data, and all data were anonymised before analysis. The study was conducted in accordance with the Declaration of Helsinki.

3. Results

3.1. Participant flow and cohort characteristics

Thirty-four adult trauma patients met the eligibility criteria and were analysed. The participant flow, including the pre-specified exclusions and the completeness of exposure data, is set out in Figure 1; no enrolled patient was subsequently lost from the analysis. The demographic, injury and physiological profile of the cohort is detailed in Table 1 and displayed graphically in Figure 2.

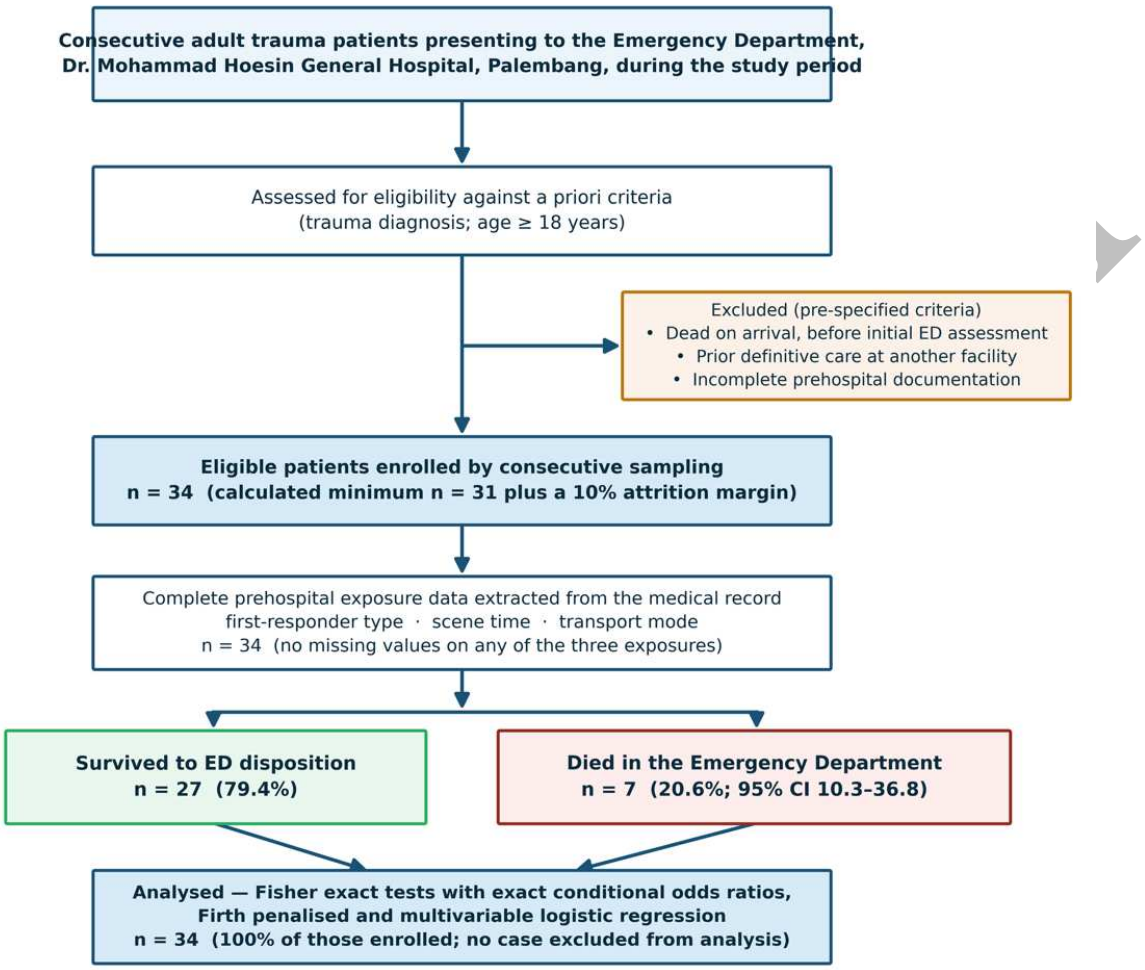

Figure 1. *Participant flow through the study, reported in accordance with the STROBE statement for cross-sectional studies. All 34 eligible patients contributed complete data on the three prehospital exposures and on the outcome.*

As detailed in Table 1, men accounted for 24 patients (70.6%) and the cohort was concentrated in the economically active age band, with 22 patients (64.7%) aged 25–59 years; the mean age was 40.94 ± 17.21 years (range 19–79), as shown in panel A of Figure 2. Blunt mechanisms predominated, affecting 30 patients (88.2%), with penetrating injury in 3 (8.8%) and thermal injury in 1 (2.9%). The head was the principal injured region in 15 patients (44.1%), followed by the extremities and multiple regions in 7 patients each (20.6%), a distribution presented in panel B of Figure 2. Most incidents occurred within the city limits (31 patients, 91.2%). Monotrauma was recorded in 27 patients (79.4%) and multiple trauma in 7 (20.6%). This case-mix closely reproduces earlier institutional series from the same emergency department.[4,5]

Anatomical severity spanned the full range, with a mean ISS of 16.50 ± 8.77 (range 4–41): 11 patients (32.4%) had minor injury, 10 (29.4%) moderate, 6 (17.6%) severe and 7 (20.6%) critical injury. Physiological derangement was less marked, with a mean GCS of 12.38 ± 4.46 (range 3–15) and 24 patients (70.6%) in the mild category. Both severity distributions are shown side by side in panel C of Figure 2. Seven patients died in the emergency department, giving a mortality of 20.6% (95% CI 10.3–36.8), as recorded in the final rows of Table 1.

3.2. Prehospital exposure profile

The distribution of the three prehospital exposures is presented in Table 2 and summarised graphically in panel D of Figure 2. As Table 2 shows, initial assistance was provided by a lay, non-medical rescuer in 32 patients (94.1%), leaving only 2 patients (5.9%) who received their first care from a medical or paramedical provider. Transport followed the same pattern: 32 patients (94.1%) arrived by a non-ambulance vehicle and 2 (5.9%) by ambulance. Scene time exceeded the 60-minute golden-hour threshold in 24 patients (70.6%), with a mean of 116.47 ± 55.10 minutes and a range from 60 to 240 minutes.

Two features of the scene-time distribution require explicit statement. First, the shortest interval recorded was exactly 60 minutes, so the comparison group defined in Table 2 as 60 minutes or less consists entirely of patients who reached care at that threshold rather than within it; the cohort contains no genuinely early-presenting stratum. Second, the clustering of values at round intervals — a minimum of exactly 60 minutes and a maximum of exactly 240 — indicates that these times were estimated retrospectively at triage rather than timed prospectively. No patient in this cohort combined arrival within the golden hour with ambulance transport and a trained first responder, so the exposure combination regarded internationally as the standard of care was not observed at all.

Table 1. Baseline demographic, injury and physiological characteristics of the study cohort (n = 34).

Characteristic	Category	n (%)	Summary statistic
Age group	Young adult, 19–24 years	7 (20.6)	Mean 40.94 ± 17.21 years
	Adult, 25–59 years	22 (64.7)	Range 19–79 years
	Older adult, ≥60 years	5 (14.7)	
Sex	Male	24 (70.6)	Male-to-female ratio 2.4:1
	Female	10 (29.4)	
Principal injured region	Head	15 (44.1)	Head injury predominant
	Thoracic	3 (8.8)	
	Abdominal	2 (5.9)	
	Extremity	7 (20.6)	
	Multiple regions	7 (20.6)	
Injury mechanism	Blunt	30 (88.2)	Blunt-to-penetrating ratio 10:1
	Penetrating	3 (8.8)	
	Thermal	1 (2.9)	
Incident location	Within the city	31 (91.2)	Predominantly urban
	Outside the city	3 (8.8)	
Injury Severity Score	Minor (1–9)	11 (32.4)	Mean 16.50 ± 8.77
	Moderate (10–16)	10 (29.4)	Range 4–41
	Severe (17–24)	6 (17.6)	
	Critical (≥25)	7 (20.6)	
Glasgow Coma Scale	Mild (13–15)	24 (70.6)	Mean 12.38 ± 4.46
	Moderate (9–12)	3 (8.8)	Range 3–15
	Severe (3–8)	7 (20.6)	
Trauma classification	Monotrauma	27 (79.4)	
	Multiple trauma	7 (20.6)	
Emergency department outcome	Survived	27 (79.4)	
	Died	7 (20.6)	95% CI 10.3–36.8

Notes: Values are n (%) unless otherwise stated. Continuous variables are summarised as mean ± standard deviation with observed range. The confidence interval for the mortality proportion is a Wilson score interval.

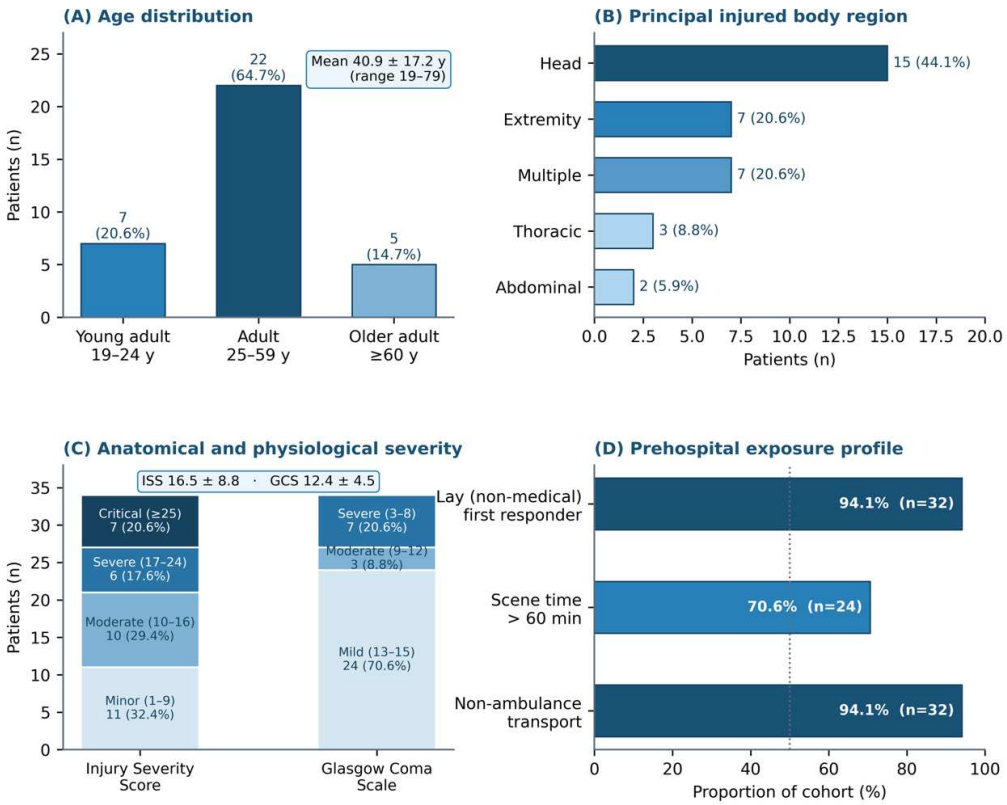

Figure 2. *Demographic, anatomical, physiological and prehospital profile of the cohort. (A) Age distribution; (B) principal injured body region; (C) anatomical severity by Injury Severity Score and physiological severity by Glasgow Coma Scale; (D) prevalence of the three prehospital exposures. Percentages are of the full cohort of 34 patients.*

Table 2. Distribution of the three prehospital exposures among adult trauma patients (n = 34).

Prehospital exposure	Category	n (%)	Continuous summary
First-responder type	Lay (non-medical) rescuer	32 (94.1)	—
	Medical or paramedical provider	2 (5.9)	—
Scene time	≤ 60 minutes (golden hour)	10 (29.4)	Mean 116.47 ± 55.10 minutes
	> 60 minutes	24 (70.6)	Range 60–240 minutes
Transport mode	Ambulance	2 (5.9)	—
	Non-ambulance vehicle	32 (94.1)	—

Notes: Scene time was defined as the interval from the traumatic event to receipt of medical care at a health facility. No patient in this cohort combined a scene time within 60 minutes with ambulance transport and a trained first responder.

3.3. Association between prehospital exposures and mortality

Bivariate results are set out in full in Table 3 and displayed graphically in Figure 3. As Table 3 shows, among the 24 patients with a scene time longer than 60 minutes, 6 died (25.0%; 95% CI 11.9–44.6), compared with 1 of the 10 patients reaching care within 60 minutes (10.0%; 95% CI 1.8–40.4). The absolute risk difference was 15.0 percentage points (95% CI −18.1 to 36.5), the exact conditional odds ratio 2.92 (95% CI 0.28–153.08) and the Fisher exact p value 0.644. Among the 2 patients transported by ambulance, 1 died (50.0%), against 6 of 32 transported otherwise (18.8%; 95% CI 8.9–35.3), giving a risk difference of 31.2 percentage points (95% CI −12.5 to 73.0), an exact conditional odds ratio of 4.09 (95% CI 0.05–352.85) and p = 0.374. The first-responder comparison was numerically identical by construction, with 1 death among the 2 patients attended by a medical provider and 6 among the 32 attended by lay rescuers (p = 0.374). Every point estimate in Table 3 lies above unity and every confidence interval includes the null.

The transport-mode and first-responder comparisons must be read as descriptive proportions rather than as effect estimates. Each rests on an exposed stratum of two patients of whom one died, and the reverse fragility index of 1 reported in Table 5 means that a single different outcome would have reversed the qualitative conclusion. The estimators shown for these two rows of Table 3 and the corresponding intervals in Figure 3 are given for completeness and are not interpretable as evidence about ambulance transport or about trained first responders. It should also be noted that the two contingency tables are numerically identical, which is consistent with the same two patients constituting the exposed stratum for both variables; if so, these are not two exposures but one exposure measured twice.

Table 3. Bivariate association between prehospital exposures and emergency department mortality, with four complementary effect estimators (n = 34).

Exposure	Died / total (%)	Risk difference, % (95% CI)	Exact conditional OR (95% CI)	Haldane–Anscombe OR (95% CI)	φ	p[a]
Scene time > 60 min	6/24 (25.0)	15.0 (−18.1 to 36.5)	2.92 (0.28–153.08)	2.23 (0.32–15.49)	0.169	0.644
Scene time ≤ 60 min (ref.)	1/10 (10.0)	Reference	1.00	1.00	—	—
Ambulance transport	1/2 (50.0)	31.2 (−12.5 to 73.0)	4.09 (0.05–352.85)	4.08 (0.36–45.87)	0.182	0.374
Non-ambulance (ref.)	6/32 (18.8)	Reference	1.00	1.00	—	—
Medical first responder	1/2 (50.0)	31.2 (−12.5 to 73.0)	4.09 (0.05–352.85)	4.08 (0.36–45.87)	0.182	0.374
Lay rescuer (ref.)	6/32 (18.8)	Reference	1.00	1.00	—	—

Notes: OR, odds ratio; CI, confidence interval; φ, phi coefficient (equivalent to Cramér's V for a 2 × 2 table); ref., reference category. [a]Two-sided Fisher exact test, pre-specified because two of four expected cell counts were below five in every table (minimum expected counts 2.06, 0.41 and 0.41 respectively). Corresponding Pearson χ^2 values were 0.971, 1.124 and 1.124 and likelihood-ratio G^2 values 1.081, 0.917 and 0.917. Risk differences use the Newcombe hybrid score method. The first-responder and transport comparisons are numerically identical because the two contingency tables have the same cell counts.

3.4. Penalised and multivariable modelling

The omnibus likelihood-ratio test is the appropriate global statistic for the multivariable model, and as reported in Table 4 it does not reject the composite hypothesis that the three prehospital exposures jointly carry no information about mortality (χ^2 = 2.394, df = 3, p = 0.495). This statistic was not reported in the original analysis and was recovered from the Cox and Snell R^2 using the relationship $R^2_{CS} = 1 - \exp(-G^2/n)$.

Firth penalised univariable estimates, reported in Table 4 and plotted as the middle row of each group in Figure 3, shrank the crude effects towards the null and produced far narrower intervals than the exact conditional method: 2.23 (95% CI 0.38–23.63) for prolonged scene time and 4.08 (95% CI 0.29–57.01) for both ambulance transport and medical first responder. The multivariable model containing all three exposures simultaneously identified none of them as an independent predictor. Re-expressed so that each estimate refers to the exposed category, the adjusted odds ratios in Table 4 were 3.51 (95% CI 0.28–43.48; p = 0.330) for scene time longer than 60 minutes, 5.22 (95% CI 0.12–235.09; p = 0.395) for ambulance transport and 1.49 (95% CI 0.04–58.82; p = 0.833) for a medical first responder. The model explained little of the observed variation, with a Cox and Snell R^2 of 0.068 and a Nagelkerke R^2 of 0.107. Although overall classification accuracy was 82.4%, Table 4 shows that this was achieved almost entirely by predicting survival for nearly every patient: specificity was 100% (27 of 27) but sensitivity for death only 14.3%, with 1 of 7 deaths correctly classified. The near-certain collinearity between transport mode and first-responder type noted in section 3.3 provides a further reason for the instability of the individual coefficients, independent of the events-per-variable problem.

Table 4. Univariable Firth penalised and multivariable logistic regression models for emergency department mortality (n = 34; 7 events).

Exposure	Firth penalised OR (95% CI)[a]	β	SE	Wald	Adjusted OR (95% CI)[b]	p
Scene time > 60 min	2.23 (0.38–23.63)	1.256	1.289	0.949	3.51 (0.28–43.48)	0.330
Ambulance transport	4.08 (0.29–57.01)	1.653	1.942	0.724	5.22 (0.12–235.09)	0.395
Medical first responder	4.08 (0.29–57.01)	0.397	1.888	0.044	1.49 (0.04–58.82)	0.833
Constant	—	−0.827	1.818	0.207	0.44	0.649

Notes: Model fit: −2 log likelihood 32.167; Cox and Snell R^2 0.068; Nagelkerke R^2 0.107. Omnibus likelihood-ratio test: χ^2 = 2.394, df = 3, p = 0.495. Classification at a cut value of 0.500: sensitivity 14.3% (1 of 7 deaths), specificity 100% (27 of 27 survivors), overall accuracy 82.4%. OR, odds ratio; CI, confidence interval; SE, standard error. [a]Univariable logistic regression with Firth's penalised likelihood; intervals obtained by profile penalised likelihood. [b]Multivariable model containing all three exposures simultaneously, as originally specified by the investigators. Coefficients β are shown with the sign reported by the analysis software; the adjusted odds ratios have been re-expressed so that each refers to the exposed category named in the first column, matching the direction of the crude estimates in Table 3. The events-per-variable ratio of this model is 2.3.

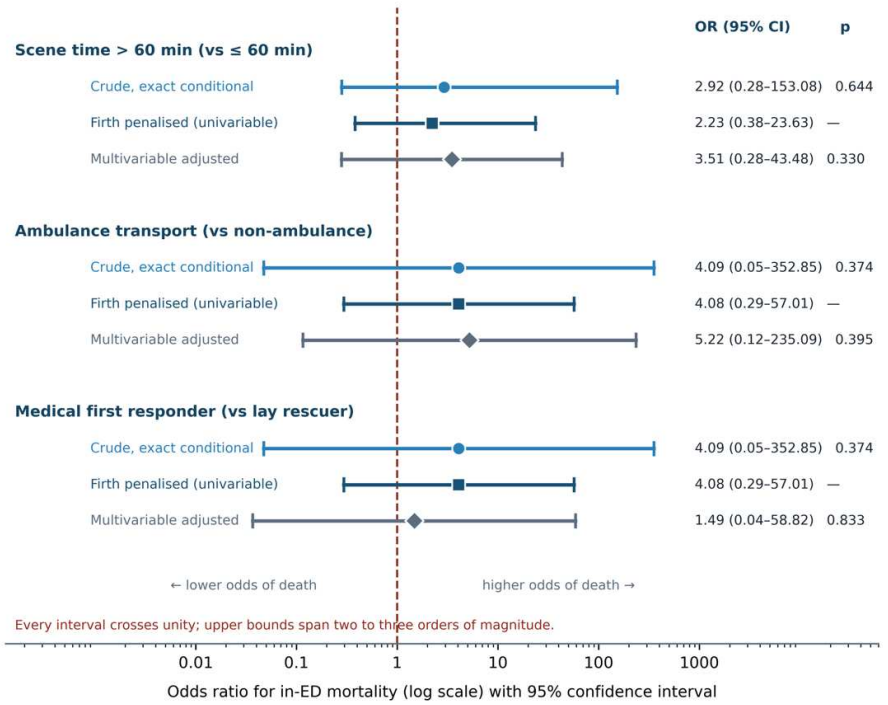

Figure 3. *Forest plot of the association between each prehospital exposure and emergency department mortality, showing the crude exact conditional odds ratio, the univariable Firth penalised estimate and the multivariable adjusted estimate. Adjusted estimates have been re-expressed to refer to the exposed category. All intervals cross unity and their upper bounds span two to three orders of magnitude.*

3.5. Precision, power and fragility

Table 5 and Figure 4 quantify what this sample was capable of detecting. As Table 5 records, the smallest odds ratio detectable at 80% power and a two-sided α of 0.05 was 12.57 for scene time and 69.82 for the two exposures with only two patients in the exposed stratum, while post-hoc power at the effect sizes actually observed was 12.6% for scene time and 23.1% for transport mode and first-responder type. Panel B of Figure 4 places the observed and detectable effects on the same axis and makes the gap between them immediately visible: the study was equipped to identify only effects an order of magnitude larger than the 1.2 to 2.0 range that trauma-system studies actually report. The reverse fragility index was 7 for scene time and 1 for both transport mode and first-responder type; that is, seven of the eighteen survivors with prolonged scene time would have had to die instead for the scene-time result to reach significance, whereas for the other two exposures a single different outcome would have sufficed. With 7 events and 3 covariates, the events-per-variable ratio of the multivariable model was 2.3, well below the value at which regression coefficients can be regarded as stable. Together these figures establish that the null findings reflect the precision of the design rather than a demonstrated absence of association.

Table 5. Precision analysis: realised statistical power, minimum detectable effect and reverse fragility for each prehospital exposure.

Exposure	Exposed / unexposed (n)	Observed OR	Post-hoc power (%)	Minimum detectable OR at 80% power	Reverse fragility index
Scene time > 60 min	24 / 10	3.00	12.6	12.57	7
Ambulance transport	2 / 32	4.33	23.1	69.82	1
Medical first responder	2 / 32	4.33	23.1	69.82	1

Notes: OR, odds ratio. Power was computed for the effect size actually observed, at a two-sided α of 0.05 and the realised group sizes. The minimum detectable odds ratio is the smallest true effect this design could have identified with 80% power. The reverse fragility index is the smallest number of patients whose outcome status would have to change for the Fisher exact test to reach p < 0.05. Published trauma-system effect sizes lie between approximately 1.2 and 2.0, well below every detectability threshold in this table.

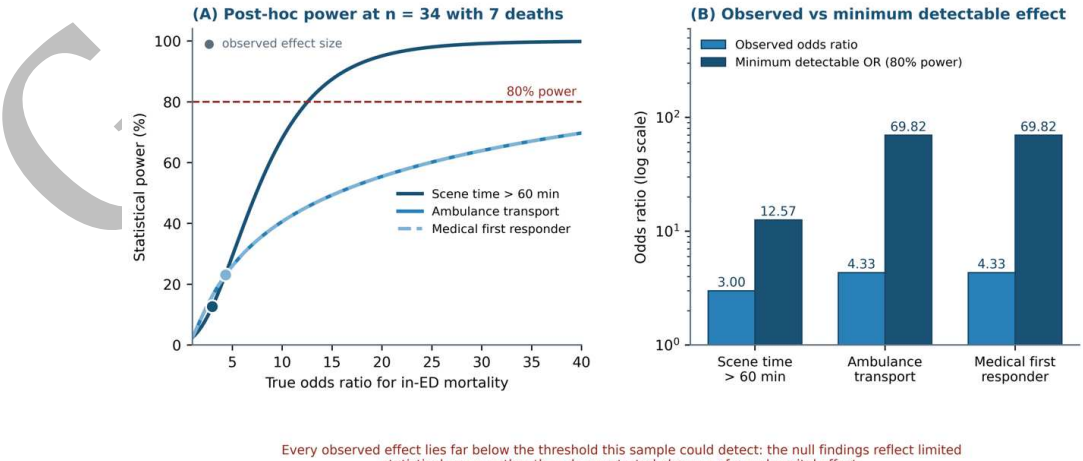

Figure 4. *Precision achieved by the present design. (A) Post-hoc statistical power as a function of the true odds ratio, with the observed effect size marked for each exposure; (B) observed odds ratios compared with the minimum odds ratio detectable at 80% power. Every observed effect lies far below the detectability threshold, establishing that the null findings reflect limited power rather than demonstrated absence of an association.*

4. Discussion

The most important result of this study is descriptive rather than inferential. In a cohort drawn from the principal tertiary referral centre of South Sumatera, 94.1% of trauma patients received their first assistance from a bystander with no medical training, 94.1% travelled to hospital in a vehicle that was not an ambulance, and 70.6% did not reach medical care within the golden hour, with a mean scene time of 116.47 minutes — almost double the threshold around which international trauma systems are organised. Not one patient experienced the combination of exposures that constitutes the international standard of care. Against that background, emergency department mortality was 20.6% and none of the three prehospital exposures examined was significantly associated with death. The absence of a statistical association should not be allowed to obscure the magnitude of the system gap that these proportions describe.

4.1. Interpreting a null result

Four considerations govern the interpretation of these findings. First, and decisively, the study was not powered to detect any plausible effect. Sample size had been calculated on the assumption of mortality proportions of 65% and 30% in the exposed and unexposed groups; the proportions observed, shown in Table 3, were 25.0% and 10.0%, so the effect the design was built to find was several times larger than the effect observed. The consequence is arithmetically severe: as Table 5 records, the realised power was between 12.6% and 23.1%, and the smallest detectable odds ratio ranged from 12.57 to 69.82. Because published trauma-system effects fall between roughly 1.2 and 2.0, a study of this size could not have detected the true effect even if it were exactly as large as the literature suggests. A non-significant p value obtained under these conditions carries essentially no evidential weight against the hypothesis, and the correct reading is the interval estimate, which for scene time is compatible with anything from a meaningful protective effect to a 153-fold increase in the odds of death.[32]

Second, the exposure distribution was extremely unbalanced, as Table 2 makes plain. Only two patients were transported by ambulance and only two were attended by a medical first responder, so both comparisons rest on a single death in a stratum of two, with a reverse fragility index of 1. Third, the cohort was skewed towards lesser injury: as Table 1 shows, 61.8% of patients had an ISS in the minor or moderate range and 70.6% presented with a mild GCS. Prehospital care exerts its greatest influence in patients with time-critical haemorrhage or airway compromise, and a cohort composed largely of physiologically stable patients dilutes any such effect towards the null — precisely the pattern reported when prehospital time was examined across severity strata in larger cohorts.[9,10]

Fourth, the direction of the likely confounding deserves explicit reasoning. All three exposures are plausibly associated with injury severity in the same direction: an ambulance is more likely to be summoned and a trained responder more likely to attend when a patient appears gravely injured, and extrication from a severe incident takes longer, lengthening the interval to first medical care. Injury severity is in turn strongly associated with death, as demonstrated repeatedly in this very emergency department.[6,7,33] Every crude estimate in Table 3 lies above unity, and confounding by severity alone would be expected to produce exactly that pattern. Because the aggregated data available for this analysis do not cross-tabulate injury severity against mortality, this explanation cannot be tested here; it is, however, more parsimonious than any causal interpretation. A further consequence of the absence of an Indonesian trauma registry is that the 20.6% mortality reported in Table 1 cannot be benchmarked against a risk-adjusted national expectation — an evidentiary gap that is itself part of what this study documents.[28]

4.2. Comparison with the published literature

The direction of these results is consistent with the more rigorous contemporary literature, and Table 6 places the present findings alongside the principal comparator studies. The single closest analogue is the cohort analysis showing that the association between on-scene time and in-hospital mortality weakened substantially once the type and number of prehospital interventions were taken into account,[9] which is precisely the interpretation this study's data support. A national population-based Norwegian study found that the far longer prehospital times of remote settings did not produce a corresponding excess mortality,[10] and a Southeast Asian cohort — the most comparable health system to the present one — found no independent association between scene time and mortality in major trauma.[11] The same null relationship has been reported in paediatric trauma.[12] Where prehospital time does vary systematically, as across Swedish population-density strata, the variation is geographical rather than prognostic.[8]

The counterintuitive observation in Table 3 that both patients transported by ambulance and both patients attended by a medical responder had a 50.0% mortality has a well-described explanation. A nationwide Japanese study found that physician-staffed ambulance attendance was associated with higher in-hospital mortality among hypotensive patients following prolonged prehospital stay — a textbook demonstration of confounding by indication rather than of iatrogenic harm.[13] A large registry comparison found non-ambulance transport no worse than ground ambulance after adjustment,[14] and both a Swedish cohort and a systematic review with meta-analysis concluded that apparent transport-modality effects reflect patient selection, as the final column of Table 6 records.[1,15] In an environment where ambulance use runs at 5.9%, as Table 2 records, this selection is likely to be extreme: the two ambulance-transported patients are best understood as the two whose condition at the scene was most alarming.

The apparent contradiction with studies that do find a time effect is largely a matter of study base. Analyses anchored at the scene capture the patients who die before hospital arrival; analyses anchored at the hospital, such as the present one, are conditioned on survival to the emergency department. A road-traffic injury registry that separated prehospital from hospital deaths demonstrated that most fatalities occur before arrival,[3] which is the mechanism by which this survivor effect operates and which Figure 1 makes explicit through the exclusion of dead-on-arrival patients. Contemporary practice has moved accordingly towards physiological and composite risk stratification rather than elapsed time: early vital signs identify 24-hour mortality risk far more effectively than time-based metrics,[16] composite anatomical-physiological scores outperform single-domain scores in emergency department trauma populations,[17] adding age to the Glasgow Coma Scale improves mortality discrimination after head injury,[18] and a simple bedside product of shock index and Glasgow Coma Scale performs comparably to a full anatomical-physiological model.[34]

4.3. The molecular basis of a time-independent result

It must be stated at the outset that **no biological marker was measured in this study**. No lactate, base deficit, fibrinogen, ionised calcium, viscoelastic assay or syndecan-1 concentration was available. What follows, and the framework presented in Figure 5, is a mechanistic interpretation drawn entirely from published work; the kinetics shown in panel B of Figure 5 are illustrative values taken from other populations and are placed against this cohort's mean scene time for orientation only. Prevalence figures for coagulopathy derive from cohorts of severely injured patients and do not transfer directly to the present cohort, in which 61.8% of patients had minor or moderate injury.

With that qualification, the mechanistic account explains why elapsed time behaves so poorly as a univariable prognostic marker. As Figure 5 sets out, mechanical energy transfer initiates cellular injury and hypoperfusion, which deplete fibrinogen and provoke an acute coagulopathy that is independently associated with massive transfusion and death.[19] That coagulopathy is predictable at emergency department arrival from routinely available early variables,[20] and its severity is indexed by a disturbance in ionised calcium that correlates with transfusion requirement and mortality across a large multicentre registry.[21] In parallel, degradation of the endothelial glycocalyx releases syndecan-1, whose circulating concentration tracks injury burden and predicts poor outcome,[22] and which, together with soluble thrombomodulin and the receptor for advanced glycation end products, is associated with acute respiratory distress syndrome, acute kidney injury and death.[23] The convergence of these processes with hypothermia and acidosis constitutes the lethal triad depicted in Figure 5.

Table 6. The present findings in the context of the principal contemporary studies of prehospital determinants of trauma mortality.

Study	Design and setting	n	Principal finding	Consistency with the present study
Wu et al., 2025	Retrospective cohort, trauma centre	23,668	Association between on-scene time and in-hospital mortality weakened substantially after adjustment for the type and number of prehospital interventions	Consistent; supports interventions rather than elapsed time as the operative factor
Nilsbakken et al., 2023	National population-based cohort, Norway	6,187	Markedly longer prehospital times in remote settings were not accompanied by a corresponding excess mortality after case-mix adjustment	Consistent; prolonged prehospital time need not translate into higher mortality
Siripakarn et al., 2023	Retrospective cohort, tertiary centre, Thailand	405	Scene time was not independently associated with mortality in major trauma transported by emergency medical services	Consistent; closest regional analogue in a comparable Asian health system
Rickenbach et al., 2024	Retrospective cohort, paediatric trauma	1,494	Prehospital time was not associated with mortality after adjustment for injury severity	Consistent; severity dominates outcome in hospital-based cohorts
Yamamoto et al., 2021	Nationwide cohort, Japan	10,168	Physician-staffed ambulance attendance was associated with higher mortality among hypotensive patients following prolonged prehospital stay	Consistent; explains the paradoxical direction of the transport-mode estimate here
Rahhal et al., 2025	National registry cohort, United States	1,229,976	Non-ambulance transport was not associated with worse adjusted survival than ground ambulance transport in blunt trauma	Consistent; non-ambulance arrival is not inherently harmful once severity is accounted for
Anvari et al., 2026	Systematic review and meta-analysis	—	The survival advantage of one transport modality over another remains unproven after pooling	Consistent; transport-mode effects unresolved even at meta-analytic scale
Homayoun et al., 2022	Road traffic injury registry, Tabriz, Iran	4,860	The majority of road-traffic deaths occurred before hospital arrival	Explains the survivor conditioning that attenuates prehospital effects in this design
Present study, 2026	Cross-sectional, single tertiary centre, Palembang, Indonesia	34	Mortality 20.6%; no prehospital exposure significantly associated with death; minimum detectable OR 12.57	Uninformative rather than negative; intervals remain compatible with meaningful effects

Notes: OR, odds ratio. Sample sizes are those reported in the respective source publications and are shown to indicate the order of magnitude of each study. Consistency is assessed with respect to the direction and interpretation of the effect estimates, not with respect to statistical significance.

The rate at which an individual traverses this cascade is governed by injury burden and physiological reserve, not by elapsed minutes. A young patient with an isolated tibial fracture and a scene time of two hours may be biologically untouched, while an older patient with a haemorrhagic pelvic fracture may be irretrievably coagulopathic within thirty; the finding that frailty rather than chronological age governs short-term mortality in older multiple-trauma patients makes the same point clinically.[27] This heterogeneity is the biological reason why crude scene time performs poorly as a univariable predictor. It also identifies where the therapeutic leverage actually lies, because the interventions with demonstrated prehospital benefit act on the cascade rather than on the clock. Prehospital tranexamic acid produces a dose-dependent fall in syndecan-1, directly linking a deliverable prehospital drug to the endothelial limb of the cascade;[24] its benefit after traumatic brain injury depends on how early it is given;[25] and the endothelial response varies with the patient's physiological reserve, arguing for stratified rather than uniform administration.[26] That the PATCH-Trauma trial found improved early survival without improved six-month functional outcome illustrates how demanding the contemporary evidentiary standard has become, and how far the field has moved beyond transport speed as an end in itself.[35]

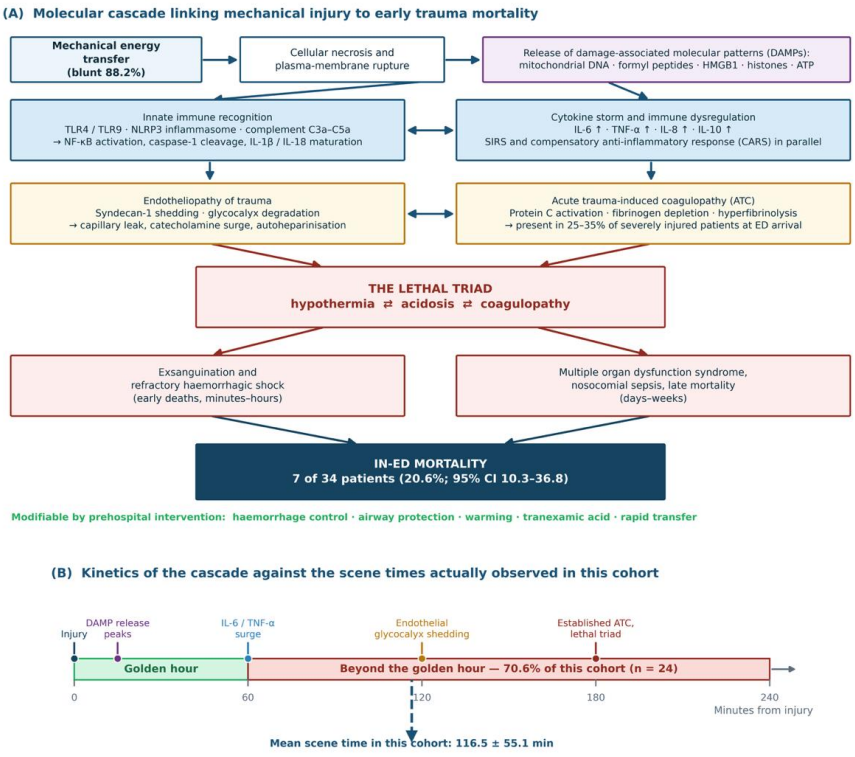

Figure 5. *Molecular framework linking mechanical injury to early trauma mortality. (A) The cascade from damage-associated molecular pattern release through innate immune activation, endotheliopathy and acute trauma-induced coagulopathy to the lethal triad and death; (B) the published kinetics of that cascade set against the scene times observed in this cohort. The framework is a synthesis of published mechanisms; no biological markers were measured in the present study.*

4.4. Interdisciplinary and institutional implications

The collaboration between the Department of Surgery, the Department of Orthopaedics and the Department of Biology at the Faculty of Medicine, Universitas Sriwijaya allows one clinical problem to be addressed from three complementary directions, and Figure 6 sets out the integrated pathway we propose for Dr. Mohammad Hoesin General Hospital. The surgical perspective defines the resuscitation and damage-control decisions that follow arrival. The orthopaedic perspective governs the timing of definitive skeletal fixation, which contemporary evidence bases on physiological state rather than elapsed time since injury, and which this centre's own pelvic ring fracture series has shown to be driven by physiological derangement and injury burden.[33] The biological perspective supplies the measurable markers by which physiological reserve can be phenotyped rather than inferred: fibrinogen, ionised calcium and, in a research setting, syndecan-1.[19,21,22] As Figure 6 indicates, none of these requires infrastructure beyond that already present in a tertiary Indonesian laboratory, and a bedside perfusion sign such as central capillary refill time — independently associated with trauma mortality — requires no equipment at all.[36] Structured scoring systems have already been shown to stratify operative need and outcome in an Indonesian blunt trauma cohort, so the approach is not foreign to this setting.[37] That composite anatomical-physiological scores outperform single-domain scores establishes the methodological precedent for the integrated risk phenotype shown in the central lane of Figure 6.[17,34]

Three implications follow for practice in South Sumatera. First, given that 94.1% of patients in Table 2 were first attended by a lay rescuer, the highest-yield intervention available to this health system is not the procurement of ambulances but the systematic training of lay first responders in the small number of manoeuvres that alter early mortality: external haemorrhage control, airway positioning, spinal protection and prevention of heat loss. This is consistent with international survey evidence attributing prehospital delay in low- and middle-income settings primarily to untrained responders and absent dispatch rather than to distance.[2] Second, the near-absence of ambulance transport identifies a structural deficiency in provincial emergency medical services that cannot be remedied within a single hospital and requires provincial coordination of dispatch, vehicle availability and pre-alert to the receiving centre. Third, and most immediately actionable, establishing a prospective regional trauma registry linking prehospital timings, injury severity, physiological parameters and outcome would convert questions of this kind from underpowered single-centre exercises into adequately powered analyses. Both a practical design framework and a costing for exactly such a registry in a comparable tertiary low- and middle-income institution have been published,[28,29] so neither feasibility nor affordability remains a valid objection. The registry feedback loop shown on the right of Figure 6 is the mechanism by which system performance would be monitored.

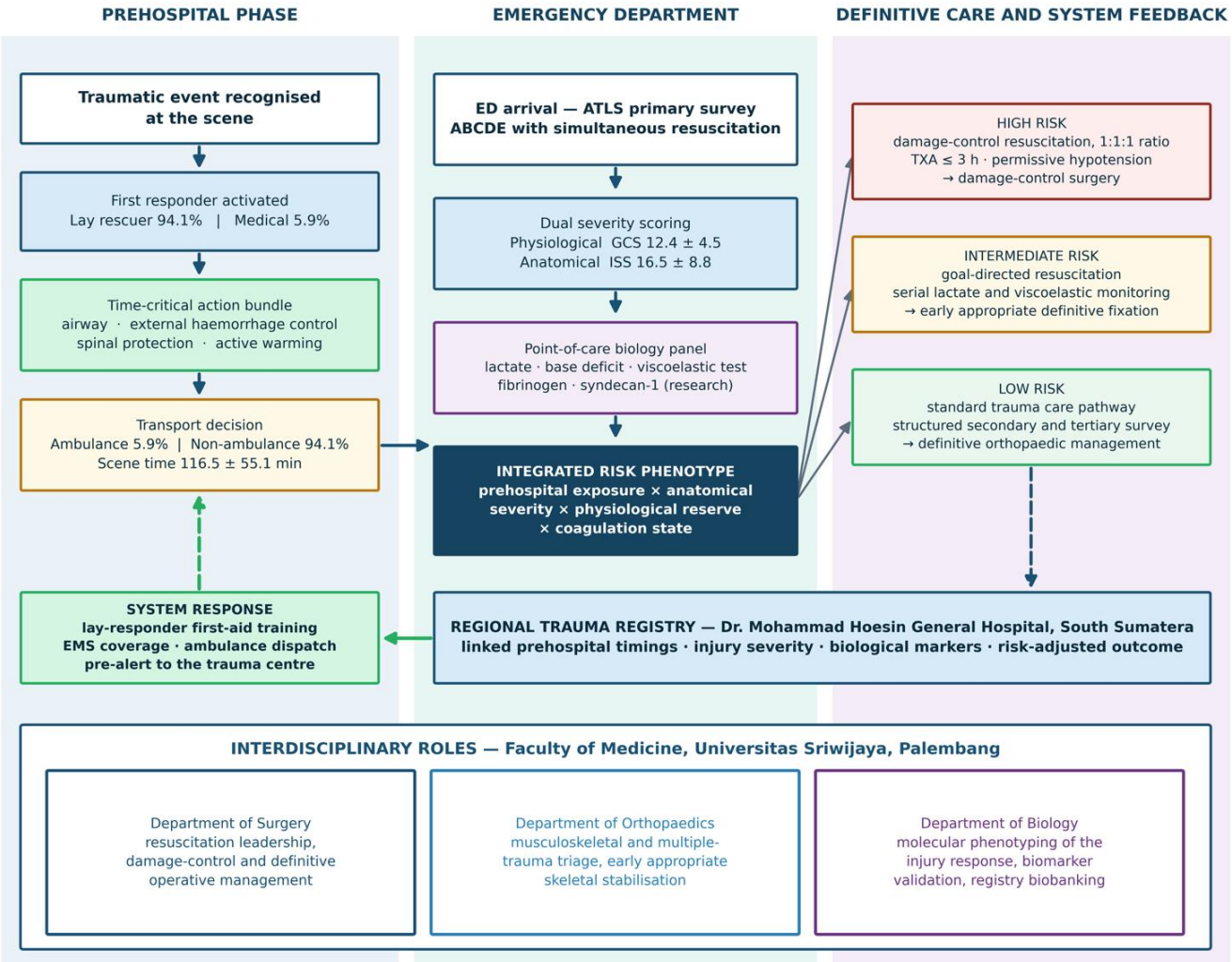

Figure 6. *Proposed integrated prehospital, emergency department and definitive care pathway for adult trauma at Dr. Mohammad Hoesin General Hospital, showing the complementary contributions of the Departments of Surgery, Orthopaedic and Biology, Faculty of Medicine, Universitas Sriwijaya, and the registry feedback loop through which system performance would be monitored.*

4.5. Implications for musculoskeletal trauma

More than two-fifths of this cohort had a musculoskeletal component to their injury: as Table 1 records, the extremities were the principal injured region in 7 patients (20.6%) and a further 7 (20.6%) had multiple-region injury. For these patients the consequences of a mean interval of 116.47 minutes from injury to first medical care extend well beyond mortality, and a study confined to death as an outcome necessarily misses most of the harm. An open fracture sustained at the roadside and first seen by a clinician nearly two hours later has passed outside every recommended window for antibiotic administration and wound irrigation. A limb splinted — or more often not splinted — by an untrained bystander for that interval carries an elevated risk of compartment syndrome and of secondary neurovascular injury during transport in a private vehicle. An unsplinted femoral shaft fracture continues to bleed throughout, contributing to precisely the physiological derangement that later governs whether early definitive fixation is safe; this centre's own analysis of pelvic ring fractures found that physiological derangement and injury burden, rather than elapsed time, determined in-hospital mortality.[33] Prehospital delay therefore influences orthopaedic decision-making indirectly, through the physiological state in which the patient arrives, rather than through any fixed time limit. This is an additional reason why prehospital system improvement matters at this centre even though the mortality analysis was null, and it identifies limb salvage, infection and functional outcome as endpoints a successor study should capture.

4.6. Lessons for surgical practice

Four practical conclusions follow from this analysis, and they are worth stating plainly rather than leaving to be inferred. The first is that in this system almost the whole of the first hour after injury is spent outside medical care, so hospital-based quality improvement — however well executed — cannot address the largest single source of delay in the pathway. The leverage lies upstream of the emergency department doors, and a centre that measures only what happens after arrival will systematically overlook it. The second follows directly from the exposure profile in Table 2. With 94.1% of patients first attended by a lay rescuer and the same proportion arriving without an ambulance, systematic training of lay first responders in external haemorrhage control, airway positioning, spinal protection and prevention of heat loss is a higher-yield intervention than vehicle procurement; it is also achievable within existing provincial resources rather than being contingent on new capital expenditure, which matters in a health system where capital is the binding constraint.

The third conclusion concerns measurement. Elapsed prehospital time should not be used as an isolated quality indicator, because it is a weak univariable predictor for the biological reasons set out above and because the interventions with demonstrated benefit act on the injury cascade rather than on the clock. A service that shortened its transport times while delivering no haemorrhage control, no warming and no early antifibrinolytic therapy would improve its metric without improving its outcomes, and the metric would conceal rather than reveal that failure. The fourth conclusion is a caution about the present study itself. This manuscript must not be cited as evidence that prehospital factors do not affect trauma mortality. With the realised power and the minimum detectable effects reported in Table 5, it could not have detected an association of the magnitude the literature actually reports; it is an uninformative study rather than a negative one, and that distinction is arithmetical rather than rhetorical.

4.7. Strengths

The study has several genuine strengths. Consecutive sampling over a defined period reduces selection bias relative to convenience sampling, and as Figure 1 records, complete data were available on every exposure and on the outcome for all 34 patients, so no imputation was necessary and no case was lost to analysis. The exposures were defined operationally before data collection and abstracted with a structured instrument. The analysis has been strengthened beyond the original protocol by the use of exact rather than asymptotic inference, by the reporting of four complementary effect estimators in Table 3, by Firth penalised modelling to address sparse-data bias, and by explicit quantification of the precision achieved in Table 5 and Figure 4. Reporting the minimum detectable effect, the realised power and the reverse fragility index alongside the p values allows a reader to distinguish an uninformative study from a negative one, a distinction frequently blurred in the trauma literature. Finally, the study provides the first quantified description of the prehospital exposure profile of trauma patients reaching this provincial referral centre, which has value independent of the hypothesis test.

4.8. Limitations

The limitations are substantial and must govern interpretation. The sample of 34 patients with 7 deaths is far too small for the questions posed, and the resulting imprecision, quantified in Table 5, is the dominant feature of the results rather than an incidental caveat. The design is cross-sectional, so associations cannot be interpreted causally. The study was conducted at a single tertiary referral centre and its findings may not generalise to primary or secondary facilities, still less to the population of injured people who never reach hospital. Because dead-on-arrival patients were excluded by design, as Figure 1 shows, and referred patients were excluded because their prehospital exposures could not be attributed, the cohort is conditioned on survival to the emergency department, which systematically attenuates any true effect of prehospital delay. The number of patients screened and excluded under each criterion was not recorded and therefore could not be reported in Figure 1, and the recruitment period is not documented in the source records; both are STROBE requirements that the corresponding author should supply. No physiological variable at first contact or at arrival — systolic blood pressure, heart rate, respiratory rate, shock index, oxygen saturation or temperature — was available, so no Revised Trauma Score, shock index or TRISS-derived probability of survival could be computed, even though these are the measures contemporary evidence identifies as most informative.[16,36] The prehospital exposures were captured only in dichotomous form; response time, transport time, the nature and number of interventions delivered en route, and the distance from the scene were not recorded, even though it is precisely the interventions rather than the duration that larger studies find to matter.[9] Scene time was reconstructed from admission documentation rather than from a dispatch record and is therefore subject to recall and documentation error, most probably non-differential and biasing towards the null; the same applies to first-responder type and transport mode, which were abstracted by a single investigator without duplicate coding. The continuous scene-time values were not available for this analysis, so the information loss caused by dichotomisation could not be recovered. The multivariable model contained three covariates for seven events, an events-per-variable ratio of 2.3, and it could not be adjusted for injury severity, physiological status or age because the necessary cross-tabulations were not available in the aggregated dataset. For the same reason, receiver operating characteristic analysis, time-to-event modelling and subgroup analysis by injury severity were not performed; presenting such analyses without patient-level linkage would have been misleading, and they are deliberately omitted rather than approximated. No biological markers were measured, so the framework in Figure 5 is a conceptual synthesis of published mechanisms and not a finding of this study. In-hospital complications and the operative course of the 27 survivors were not captured, because the outcome was restricted to the emergency department episode.

4.9. Recommendations for the successor study

The design of an adequately powered successor study follows directly from the figures reported here, and Table 7 converts them into a specification. Such a study should record continuous rather than dichotomised prehospital intervals, document the interventions delivered before arrival,[9] capture physiological variables at first contact including a bedside perfusion sign,[16,36] and prespecify adjustment for injury severity, Glasgow Coma Scale, age and mechanism.[17,18] It should incorporate a point-of-care biological panel comprising lactate, base deficit, fibrinogen and ionised calcium, with syndecan-1 as a research measure, so that the molecular phenotype is measured rather than assumed.[19–22] Outcomes should extend beyond emergency department mortality to 24-hour and 30-day survival and to graded in-hospital complications. Analytically, penalised or Bayesian methods should be planned from the outset rather than applied retrospectively,[31] and results reported as effect estimates with compatibility intervals rather than as significance decisions.[32] The vehicle for all of this is a prospective regional trauma registry, for which both a design framework and a costing are already available.[28,29]

Table 7. Precision attainable at increasing sample sizes, and the sample size required to detect effects of the magnitude reported in the trauma-systems literature.

Total sample size	Exposed / unexposed (n)	Smallest OR detectable at 80% power	Target OR	Total sample size required
34 (present study)	24 / 10	12.57	1.5	2,247
100	71 / 29	5.11	2.0	706
300	212 / 88	2.76	3.0	250
500	353 / 147	2.25	5.0	104
1,000	706 / 294	1.81		
2,000	1,412 / 588	1.53		

Notes: OR, odds ratio. All calculations assume the mortality of 10.0% observed in the unexposed stratum, the exposure prevalence of 70.6% observed in this cohort, a two-sided α of 0.05 and 80% power. The left-hand columns show what a study of a given size could detect; the right-hand columns show the size required to detect a given effect. Published trauma-system effects lie between approximately 1.2 and 2.0, implying that several hundred to several thousand patients would be required — attainable only through multicentre recruitment or a sustained provincial registry.

5. Conclusion

Among 34 adult trauma patients presenting to the Emergency Department of Dr. Mohammad Hoesin General Hospital, Palembang, emergency department mortality was 20.6% and neither scene time, transport mode nor first-responder type was significantly associated with death. The realised power of 12.6–23.1% and a minimum detectable odds ratio of 12.57, reported in Table 5 and Figure 4, establish that these are uninformative rather than negative findings, and the confidence intervals in Table 3 remain compatible with clinically important effects. The descriptive results are unambiguous: 94.1% of patients were first attended by a lay rescuer, 94.1% arrived without an ambulance and 70.6% reached care beyond the golden hour. Strengthening lay first-responder training, developing provincial emergency medical services and establishing the prospective biomarker-augmented regional trauma registry outlined in Figure 6 are the priorities that follow, and an adequately powered multicentre study of the size specified in Table 7 is required before any conclusion about prehospital determinants of trauma mortality in South Sumatera can be drawn.

6. Declarations

Ethical approval and consent to participate. This study received ethical approval from the Health Research Ethics Committee of the Faculty of Medicine, Universitas Sriwijaya, Palembang, Indonesia. The requirement for individual written informed consent was waived for this retrospective analysis of anonymised medical-record data. The study was conducted in accordance with the Declaration of Helsinki.

Consent for publication. Not applicable; no individually identifiable patient data are presented.

Availability of data and materials. All aggregated data supporting the conclusions of this article are contained within Table 1 to Table 5. Further detail is available from the corresponding author on reasonable request.

Competing interests. The authors declare that they have no competing interests.

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Authors' contributions. IK conceived the study, collected and interpreted the data and drafted the manuscript. RL supervised the clinical and musculoskeletal aspects of the study design and critically revised the manuscript. ZM supervised the methodology and the biological interpretation and critically revised the manuscript. All authors read and approved the final manuscript.

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