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Prehospital Determinants of Emergency Department Mortality in Adult Trauma at a South Sumatran Tertiary Referral Center: A Retrospective Cross-Sectional Study

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ABSTRACT

Background: Prehospital care is a modifiable determinant of trauma survival, but evidence from Indonesian referral centers without integrated emergency medical services is scarce.**Objective:** To examine whether first-responder type, prehospital time, and transport mode were associated with emergency department mortality in adult trauma.**Methods:** This retrospective cross-sectional study (STROBE) analyzed 34 consecutive adult trauma patients at the Emergency Department of Dr. Mohammad Hoesin General Hospital, Palembang. Associations were assessed with Fisher exact tests, exact conditional and Haldane–Anscombe odds ratios (OR), Newcombe risk differences, univariable Firth penalized logistic regression, and a multivariable model. Power, minimum detectable ORs, and reverse fragility indices were calculated.**Results:** Mortality was 20.6% (7/34; 95% CI: 10.3–36.8%). Lay rescuers attended 94.1% (32/34) of patients, 94.1% (32/34) arrived by non-ambulance transport, and 70.6% (24/34) had prehospital times >60 minutes (mean 116.47 ± 55.10 minutes). Death occurred in 6/24 (25.0%) patients with prehospital time >60 minutes versus 1/10 (10.0%) with ≤60 minutes (exact conditional OR 2.92, 95% CI: 0.28–153.08; Firth OR 2.23, 95% CI: 0.38–23.63; Fisher $p = 0.644$), and in 1/2 (50.0%) versus 6/32 (18.8%) for both ambulance transport and medical responder (exact conditional OR 4.09, 95% CI: 0.05–352.85; Firth OR 4.08, 95% CI: 0.29–57.01; $p = 0.374$). No exposure was associated with mortality in multivariable analysis ($p = 0.492$; minimum detectable OR 12.57–69.82; power 12.6–23.1%).**Conclusion:** No prehospital factor was significantly associated with mortality, but wide intervals and low power make these findings uninformative rather than negative; they support lay-responder training and a prospective regional trauma registry.

1. Introduction

Traumatic injury remains a leading cause of premature death and long-term disability worldwide, and its burden falls disproportionately on economically active adults.¹ Injuries cause approximately five million deaths each year, close to one in ten deaths across all age groups,^{1–4} and more than several major communicable diseases (HIV/AIDS, tuberculosis, and malaria) combined.⁴ This burden is concentrated in low- and middle-income countries (LMICs), where rapid motorization, dense urban road networks, and occupational hazards coexist with fragmented or absent prehospital emergency medical services (EMS).⁵ An international survey of prehospital providers and a systematic review of LMIC prehospital care indicate that delay in resource-constrained settings reflects structural health-system deficits—the absence of centralized toll-free dispatch, reliance on untrained bystander first responders, and a lack of dedicated emergency vehicles—as well as geographical barriers.^{5,6} A population-based road-traffic injury registry in Iran that separated prehospital from hospital deaths showed that most road-traffic deaths (62.5%) occurred before hospital arrival.⁷

Indonesia exemplifies this epidemiological transition. Successive institutional series from the Emergency Department

of Dr. Mohammad Hoesin General Hospital (RSUP Dr. Mohammad Hoesin)—the apex tertiary surgical referral center for South Sumatra Province and the principal teaching hospital of the Faculty of Medicine, Universitas Sriwijaya—have documented a trauma case mix concentrated in young and middle-aged men,^{8,9} and thoracic trauma analyses from the same institution indicate that mortality rises with injury severity.^{10,11} Beyond crude case-fatality rates, these institutional audits from Palembang point to a recurring structural problem across Indonesian referral networks: although tertiary resuscitation bays and operating theaters have progressively adopted damage-control surgery and subspecialty trauma protocols, the prehospital continuum that links the roadside or workplace injury scene to the emergency department remains largely unmapped, unstandardized, and unmonitored, because these audits describe in-hospital care only.^{8–11}

Within trauma-system planning, the prehospital interval is conventionally regarded as the most modifiable phase of the acute care pathway, anchored by the golden hour—the proposition that hemorrhage control, airway protection, and circulatory resuscitation delivered within 60 minutes of injury interrupt an otherwise irreversible physiological cascade toward death.^{1,12} The geographical structure of prehospital

delay has been characterized in high-income national registries: a nationwide Swedish cohort study showed that prehospital intervals differ across population-density strata (median response time by about 4 minutes and transport time by 11–15 minutes),¹² and an international survey of prehospital providers identified prolonged scene and transport intervals as frequent delays in mass-casualty incidents.⁶ If elapsed time between injury and hospital arrival acted as an autonomous, uniform determinant of survival, a trauma system that delivers most of its injured patients after the 60-minute threshold should show a monotonic, easily detectable excess in emergency department mortality.^{12,13} Evidence from contemporary trauma registries is more nuanced than a simple linear time–mortality model implies.^{13–17}

In a retrospective cohort of 13,533 trauma patients, Wu et al. showed that the association between on-scene time and in-hospital mortality disappeared once the type and number of prehospital interventions were taken into account, suggesting that what is done during the prehospital phase matters more than elapsed time alone.¹³ A Norwegian national population-based cohort of 28,988 trauma patients found that the longer prehospital intervals of remote areas were not accompanied by excess risk-adjusted mortality, although prolonged on-scene time was associated with higher adjusted odds of death.¹⁴ In contrast, a single-center Thai cohort of 104 major trauma patients transported by an advanced EMS team—the setting most directly comparable to Indonesian provincial referral care—found that scene time over 10 minutes was independently associated with 24-hour mortality (risk ratio 4.47, 95% confidence interval [CI] 1.84–10.83).¹⁵ No association between prehospital time and mortality was found in a Dutch level-I polytrauma cohort,¹⁶ whereas in pediatric trauma the adjusted mortality fell as prehospital time increased up to about 45 minutes and then plateaued.¹⁷ From a pathophysiological standpoint, this divergence between elapsed prehospital time and hospital survival does not invalidate the urgency of early hemorrhage control or airway restoration; rather, it suggests that elapsed time is a composite, noisy exposure that conflates transit distance, scene extrication complexity, bystander recognition delay, and the presence or absence of life-saving interventions en route.^{12–17}

Evaluations of prehospital transport mode and first-responder qualification raise a further epidemiological caveat: confounding by indication.^{18–21} In a nationwide analysis of 14,652 hypotensive trauma patients from the Japan Trauma Data Bank, Yamamoto et al. observed that attendance by a physician-staffed ambulance was associated with higher in-hospital mortality, an excess the authors attributed to delayed definitive hemostasis during a longer prehospital stay after weighting for severity;¹⁸ in less mature systems, the selective deployment of advanced teams to the most physiologically deranged casualties is a further possible explanation. A matched cohort of 5,316 blunt trauma patients from the 2017 US National Trauma Data Bank found similar survival after police and ground ambulance transport.¹⁹ A Swedish national comparison found lower injury-stratified mortality after helicopter than after road-ambulance transport (risk ratios 0.44–0.75),²² whereas a 2026 systematic review and meta-analysis found higher crude mortality with helicopter transport (odds ratio [OR] 1.27) and noted that more severely injured patients are preferentially flown.¹ Trauma prognostication has consequently moved from elapsed time toward early physiological thresholds and composite anatomical–physiological scores, such as early vital-sign thresholds in the Trauma Quality Improvement Program (TQIP),²³ multi-trauma scoring systems in the

emergency department,²⁴ age-augmented Glasgow Coma Scale (GCS) models in traumatic brain injury,²⁵ and prehospital triage tools—the Triage Revised Trauma Score (T-RTS), GAP, MGAP, and the reverse shock index multiplied by the GCS (rSIG)—for predicting resuscitative interventions within 24 hours.²⁶ Integrating these multivariable triage instruments into LMIC emergency departments is particularly relevant when prehospital documentation is rudimentary, because physiological derangement on arrival may reflect the cumulative effect of prehospital hypoxia, hypoperfusion, and ongoing hemorrhage whether the prehospital interval was thirty minutes or three hours.

There is a biological reason why elapsed prehospital time behaves as a noisy surrogate for early mortality risk, and it explains why the present study brings together the Departments of Surgery, Orthopaedics, and Biology. Elapsed time does not injure tissue directly; mechanical energy transfer initiates a hyperacute molecular and cellular cascade whose speed depends on the anatomical injury burden and the host's physiological reserve.^{27–29} Within minutes of severe tissue trauma and hemorrhagic hypoperfusion, accelerated fibrinogen consumption and proteolysis provoke acute trauma-induced coagulopathy (ATC), which independently predicts massive transfusion and death,²⁷ can be predicted on emergency department arrival from early clinical and laboratory parameters,²⁸ and involves coordinated dysregulation of thrombin generation, platelet function, and fibrinolysis.²⁹ At the same time, disturbances in ionized calcium (Ca^{2+}) on emergency department arrival show a parabolic association with coagulopathy, transfusion requirement, and mortality in a large multicenter registry.³⁰

In parallel with hemostatic failure, sympathoadrenal catecholamine surges and tissue ischemia trigger shedding of the endothelial glycocalyx, releasing soluble syndecan-1 and thrombomodulin into the circulation, where their concentrations track injury severity and are associated with acute respiratory distress syndrome, acute kidney injury, disseminated intravascular coagulation, and death or transfusion in pediatric and adult trauma.^{31–33} An exploratory biomarker clustering of the European multicenter ACIT-2 cohort found that clinical trauma-induced coagulopathy and a 28-day mortality of 39% were concentrated in one shock-induced endotheliopathy (SHINE) phenotype characterized by extreme syndecan-1 shedding, factor V depletion, and hyperfibrinolysis despite similar Injury Severity Scores.³⁴ This endothelial–coagulopathic cascade can be modified before hospital arrival: prehospital tranexamic acid (TXA) produces a dose-dependent reduction in circulating syndecan-1,³⁵ its benefit declines with every minute of treatment delay,^{36,37} its timing after traumatic brain injury has been examined in a randomized trial subanalysis,³⁸ and its endothelial biomarker effects differ with age, which underscores the role of host biological reserve.³⁹ Clinically, frailty predicts short-term mortality in older multi-trauma patients beyond chronological age.⁴⁰ Taken together across surgical resuscitation, orthopedic traumatology, and vascular biology, these findings suggest that two patients arriving after an identical 120-minute delay may be in very different biological states: one with an isolated closed tibial shaft fracture and intact reserve may remain hemostatically compensated, whereas another with an unstable pelvic ring disruption or severe traumatic brain injury may already have crossed the threshold of syndecan-1 glycocalyx shedding, factor V proteolysis, fibrinogen exhaustion, and ionized hypocalcemia.^{27,29,34,35,39}

Despite the volume of severe trauma managed at Indonesian tertiary referral hospitals, quantitative analyses of prehospital determinants of emergency department mortality remain scarce, and no prospective provincial trauma registry exists in South Sumatra to benchmark prehospital performance, although design frameworks and cost estimates for LMIC hospital trauma registries, and surveillance priorities for national registries, have been described.⁴¹⁻⁴³ In addition, single-center series with few events are prone to sparse-data bias when analyzed by conventional maximum-likelihood logistic regression,^{44,45} and statistically non-significant *p* values are often misread as evidence that prehospital delays are clinically benign.⁴⁶ To address these clinical and methodological gaps, a team from the Departments of Surgery, Orthopaedics, and Biology at the Faculty of Medicine, Universitas Sriwijaya, conducted the present study at Dr. Mohammad Hoesin General Hospital, Palembang. The objectives were threefold: first, to characterize the demographic, anatomical, physiological, and prehospital exposure profile of consecutive adult trauma patients presenting to this tertiary referral center; second, to quantify the association between first-responder type, prehospital time, and transport mode and emergency department mortality using exact conditional inference, the Haldane-Anscombe correction, Newcombe hybrid score risk differences, and Firth's penalized-likelihood logistic regression alongside multivariable modeling; and third, to quantify the precision achieved—through minimum detectable odds ratios, statistical power, and reverse fragility indices—so that uninformative findings are distinguished from true negative evidence and translated into sample-size estimates for a prospective, biomarker-augmented regional trauma registry.

2. Methods

Study design and reporting

This was a retrospective, hospital-based analytical cross-sectional study. The prehospital exposures and the primary outcome were ascertained within a single acute care episode—from triage arrival in the emergency department to departmental disposition—by structured abstraction of clinical and administrative medical records. Because the study was observational, no intervention was administered, and standard Advanced Trauma Life Support (ATLS) resuscitation and operative protocols were followed according to clinical indication. To ensure transparent reporting of participant selection, exposure definition, the handling of sparse cells, and the precision analysis, the study is reported according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement for cross-sectional studies.⁴⁷ The participant pathway from emergency department screening through eligibility verification, exposure classification, and disposition is shown in Figure 1. From an epidemiological perspective, observational cross-sectional exposure profiling—which captures the joint distribution of prehospital care deficits at hospital arrival—must be distinguished from causal effect estimation, which requires adequate statistical power and control of survivor selection and severity confounding.

Setting

The study was conducted in the Emergency Department of Dr. Mohammad Hoesin General Hospital (RSUP Dr. Mohammad Hoesin), Palembang, the apex tertiary referral hospital for South Sumatra Province and the primary clinical teaching institution of the Faculty of Medicine, Universitas Sriwijaya. The emergency department serves a provincial population of more than eight million people across urban Palembang and the surrounding rural regencies and receives both direct-from-scene trauma

presentations and secondary inter-facility referrals across the full spectrum of blunt, penetrating, and thermal injury. At the time of data collection, South Sumatra Province had no unified, physician-staffed prehospital EMS network, no universal toll-free trauma dispatch system with automated scene geolocation, and no standardized provincial prehospital trauma registry. Initial scene assistance and transport to the emergency department therefore relied mainly on informal community networks, bystander intervention, and private vehicles. Patients arriving by non-ambulance vehicles typically travel in private cars, commercial three- or four-wheeled vehicles, or police vehicles without en-route oxygen, cervical collars, vacuum splints, intravenous fluids, or pre-arrival notification of the emergency department resuscitation team, whereas, in the authors' experience, the small fraction arriving by ambulance mostly reflects secondary mobilization for patients perceived by bystanders or local officials as critically injured.

Participants and eligibility

We screened every consecutive adult who presented to the Emergency Department of Dr. Mohammad Hoesin General Hospital with an acute physical injury during the study period (Figure 1). To be included, a patient had to be 18 years or older and have a confirmed clinical diagnosis of acute traumatic injury caused by mechanical or thermal energy. Three exclusions were fixed before any data were extracted. Patients pronounced dead on arrival (DOA), before the first emergency department assessment, were excluded because they could not reach an in-department mortality endpoint. Patients already given definitive operative or resuscitative care at a referring primary or secondary facility were also excluded; for them, prehospital time, the initial responder and the primary transport mode could not be separated from the inter-hospital transfer. Finally, records missing any of the three prehospital exposures or the disposition outcome were ineligible. Enrollment was consecutive and stopped when the sample-size target was reached, which limits selection and spectrum bias compared with convenience sampling. Excluding dead-on-arrival casualties and post-stabilization transfers—necessary to define a coherent in-department mortality outcome and to avoid misclassifying primary prehospital times—inevitably conditions the analytical cohort on survival to the emergency department. Institutional triage logs during the study window captured all consecutive patients who completed emergency department registration, met the inclusion criteria, and had complete prehospital documentation, giving an unambiguous analytical cohort for evaluating in-department mortality.

Sample size

The target sample size was calculated before data collection with the standard normal-approximation formula for comparing two independent proportions, with a two-sided significance level of 5% ($\alpha = 0.05$; $Z_{\alpha/2} = 1.96$) and 80% power ($1 - \beta = 0.80$; $Z_{\beta} = 0.84$). On the basis of preliminary high-acuity trauma literature available to the investigators at protocol inception, emergency department mortality was assumed to be 65.0% ($p_1 = 0.65$) among patients with a high-risk prehospital exposure (prolonged prehospital time, non-ambulance transport, or an untrained first responder) and 30.0% ($p_0 = 0.30$) in the favorable reference stratum. These parameters yield 31 patients per exposure group (62 in total); the recruitment target of 34 patients (31 plus a 10% allowance for incomplete records) therefore corresponds to one group. Because the mortality proportions observed in the cohort (25.0% versus 10.0% for prehospital time) were much lower than assumed, the power achieved fell far below the nominal 80%; this discrepancy was quantified explicitly by minimum-detectable-

effect and reverse-fragility analyses rather than left implicit. Specifically, the two-proportion formula assumes a comparison between balanced or pre-specified exposure strata with an absolute risk difference of 35.0 percentage points (65.0% minus 30.0%, an unconditional OR of 4.33); when exposure prevalence is extreme (94.1% non-ambulance and 94.1% lay responder) and the observed risk difference for prehospital time is 15.0 percentage points (25.0% versus 10.0%; unconditional OR 3.00), the information available for the comparison falls sharply, so the precision achieved was quantified after the study.

Variables and definitions

The primary dependent variable was emergency department mortality, defined as all-cause death of an enrolled patient between triage arrival and formal departmental disposition (transfer to the operating theater, intensive care unit, inpatient surgical ward, or mortuary), coded 0 for survived and 1 for died. Three prehospital independent variables were defined a priori. First-responder type was the professional qualification of the person who gave initial physical assistance at the scene, dichotomized as lay (non-medical) bystander versus trained medical or paramedical provider (physician, nurse, or certified paramedic). Prehospital time was the interval from the injury to the first medical care in the emergency department, dichotomized at the golden-hour threshold as 60 minutes or less (≤ 60 minutes) versus more than 60 minutes (> 60 minutes). Transport mode was the vehicle that carried the patient from the scene to the hospital, dichotomized as dedicated ambulance versus non-ambulance vehicle (private car, motorcycle, public transport, or police or commercial vehicle).

Demographic, anatomical, and physiological covariates abstracted for cohort description were age (analyzed continuously and in three categories: young adult, 19–24 years; adult, 25–59 years; older adult, ≥ 60 years), sex, principal injured region (head, extremity, multiple regions, thoracic, or abdominal), injury mechanism (blunt, penetrating, or thermal), incident location (within or outside the Palembang city limits), Injury Severity Score (ISS; the sum of the squares of the highest Abbreviated Injury Scale [AIS] grades in the three most severely injured body regions, categorized as minor [1–9], moderate [10–16], severe [17–24], or critical [≥ 25]), GCS score on arrival (mild [13–15], moderate [9–12], or severe [3–8]), and trauma classification (monotrauma or multiple trauma). Defining prehospital time at the 60-minute boundary aligns the primary exposure with the golden-hour quality metric and suits the granularity of retrospective admission records; because a continuous duration carries more statistical information than a binary indicator, both the continuous distribution (mean, standard deviation, median, interquartile range, and Shapiro–Wilk normality test) and the clustering of values at the boundary were examined.

Data collection

All data were abstracted retrospectively from the emergency department admission register and the individual inpatient and outpatient medical records of Dr. Mohammad Hoesin General Hospital, the sole data source. Prehospital exposure variables (first-responder identity, estimated time of injury, time of emergency department arrival, and arrival vehicle) were extracted from the standardized nursing and surgical triage documentation completed on arrival. Anatomical diagnoses and initial neurological assessments were used to verify the recorded ISS and arrival GCS categories. Data were extracted by the principal surgical investigator with a standardized, pre-piloted abstraction form, followed by programmatic range and internal-consistency checks of all variables before analysis. Because abstraction was performed by a single investigator

without independent duplicate coding, inter-rater reliability statistics (such as Cohen's κ or intraclass correlation coefficients) could not be calculated; this is acknowledged as a limitation. Data on all covariates, the three prehospital exposures, and emergency department mortality were complete for all 34 enrolled patients (100.0%), so no imputation was required. Because the primary objective was to evaluate the three prehospital system exposures with few events (7 deaths), multivariable modeling was restricted to the pre-specified three-exposure model, with exact conditional and Firth penalized-likelihood estimation, rather than over-parameterizing the regression with additional anatomical or physiological covariates.

Statistical analysis

Analyses were performed in IBM SPSS Statistics version 27.0 and cross-verified in Python 3.11 with custom exact, penalized-likelihood, and power-function routines. Categorical variables were summarized as frequencies and percentages, and the overall mortality proportion was given with a Wilson score 95% confidence interval (CI), which maintains nominal coverage with small denominators and skewed proportions. Continuous variables (age, ISS, GCS, and prehospital time) were tested for normality with the Shapiro–Wilk test (statistic W) and summarized both parametrically (mean $\pm$ standard deviation [SD] with 95% CI) and non-parametrically (median, interquartile range [IQR, 25th–75th percentile], and observed range). Bivariate associations between each exposure and mortality were tested primarily with the two-sided Fisher exact test, specified a priori because two of the four expected cell counts were below 5 in every 2×2 table; Pearson χ^2 and likelihood-ratio G^2 statistics were computed for completeness but not used for hypothesis testing.

To guard against estimator instability caused by single-death cells, bivariate effect sizes were quantified with four complementary estimators: (1) the exact conditional maximum-likelihood OR with exact conditional (Fisher) 95% confidence limits; (2) the Haldane–Anscombe OR (adding 0.5 to each cell) with a Wald 95% CI; (3) the absolute risk difference (RD, in percentage points) with Newcombe's hybrid score 95% CI without continuity correction; and (4) the phi coefficient (ϕ , identical to Cramér's V in a 2×2 table). Because sparse cells inflate maximum-likelihood standard errors and bias estimates away from the null,⁴⁴ each exposure was also analyzed by univariable logistic regression with Firth's penalized maximum likelihood,^{45,48–50} which adds a Jeffreys invariant-prior penalty to the score function, removes the first-order $O(n^{-1})$ bias, and gives finite estimates even under quasi-complete separation.^{45,48} Penalized log-likelihoods were maximized by quasi-Newton optimization with a convergence tolerance of 1.0×10^{-6} , and 95% CIs were obtained from the profile penalized likelihood rather than from symmetric Wald approximations.⁴⁵

The pre-specified multivariable logistic regression model containing all three exposures was estimated to evaluate independent effects. Model fit was assessed by the -2 log-likelihood, the likelihood-ratio χ^2 test (the difference between the null and model -2 log-likelihoods), the Cox and Snell R^2 , the Nagelkerke R^2 , sensitivity and specificity at a predicted probability of 0.500, and the events-per-variable (EPV) ratio. Finally, in line with recommendations that non-significant p values be interpreted through confidence (compatibility) intervals and design precision rather than as acceptance of the null hypothesis,^{46,51} three precision metrics were computed for each exposure: (1) the minimum detectable OR (MDE) at 80% power and two-sided $\alpha = 0.05$ for the observed group sizes and unexposed baseline risk; (2) post-hoc power at the observed

unconditional OR; and (3) the reverse fragility index (RFI), defined as the minimum number of outcome reassignments in the exposed group (group sizes fixed) needed to bring the two-sided Fisher exact p value below 0.05.^{51,52} Power and MDE were computed with the two-proportion normal approximation with pooled variance; exact (Fisher) power was also calculated.

The four bivariate estimators were reported alongside Firth's penalized likelihood for a specific reason in sparse-data epidemiology. The unconditional maximum-likelihood OR (ad/bc) is biased away from the null in finite samples and undefined when any cell is zero; the exact conditional estimate conditions on the marginal totals through the non-central hypergeometric distribution; the Haldane-Anscombe estimator adds 0.5 to each cell, which in a saturated 2×2 logistic model coincides with the Jeffreys-prior penalty; and Firth's profile penalized-likelihood intervals reflect the marked skewness of the likelihood surface when the exposed event count is one.^{44,48,53}

Ethical approval

The study protocol was approved by the Health Research Ethics Committee of the Faculty of Medicine, Universitas Sriwijaya / Dr. Mohammad Hoesin General Hospital, Palembang, Indonesia (Reference No.2025/223). Because the analysis involved retrospective abstraction of routinely collected, de-identified clinical records without patient contact or any change to clinical care, the Ethics Committee waived the requirement for individual written informed consent. All patient identifiers and registration numbers were removed before analysis, and all procedures complied with the Declaration of Helsinki.

3. Results

Participant flow and baseline characteristics

Thirty-four consecutive adult trauma patients met all inclusion and exclusion criteria during the study period and formed the analytical cohort. As shown in the STROBE flow diagram (Figure 1), no enrolled patient had missing values for the three prehospital exposures, severity scores, or emergency department disposition, because records without complete exposure and outcome data were excluded by design, and no enrolled patient was excluded from the analysis. The demographic, anatomical, physiological, and outcome characteristics of the cohort are given in Table 1 and Figure 2. Men accounted for 24 patients (70.6%) and women for 10 (29.4%), a male-to-female ratio of 2.4:1 (Figure 2a). The cohort was concentrated in the 25–59-year age group (22 patients,

64.7%), with 7 young adults aged 19–24 years (20.6%) and 5 older adults aged 60 years or above (14.7%). Age was normally distributed (Shapiro–Wilk $W = 0.964$, $p = 0.318$), with a mean of 40.94 ± 17.21 years (95% CI: 34.94–46.95) and a median of 39.50 years (IQR: 26.25–53.75; range 19–79 years). Blunt trauma predominated (30 patients, 88.2%), followed by penetrating trauma (3, 8.8%) and thermal burn injury (1, 2.9%), a blunt-to-penetrating ratio of 10:1.

The head was the principal injured region in 15 patients (44.1%), followed by the extremities in 7 (20.6%), multiple body regions in 7 (20.6%), the thorax in 3 (8.8%), and the abdomen in 2 (5.9%); thus, 14 of the 34 patients (41.2%) had either primary extremity trauma or multi-region injury involving the musculoskeletal system. Thirty-one incidents (91.2%) occurred within the Palembang city limits and 3 (8.8%) outside the city. Monotrauma was recorded in 27 patients (79.4%) and multiple trauma in 7 (20.6%). Anatomical injury severity spanned the full spectrum from minor to critical, with a mean ISS of 16.50 ± 8.77 (95% CI: 13.44–19.56; median 14.00, IQR: 9.00–22.00; range 4–41; Shapiro–Wilk $W = 0.931$, $p = 0.034$): 11 patients (32.4%) had minor injury (ISS 1–9), 10 (29.4%) moderate injury (ISS 10–16), 6 (17.6%) severe injury (ISS 17–24), and 7 (20.6%) critical injury (ISS ≥ 25). Minor-to-moderate injury (ISS ≤ 16) therefore accounted for 21 patients (61.8%) and severe-to-critical injury (ISS ≥ 17) for 13 (38.2%). The GCS on arrival was negatively skewed toward preserved consciousness (Shapiro–Wilk $W = 0.654$, $p < 0.001$), with a mean of 12.38 ± 4.46 (95% CI: 10.82–13.94; median 15.00, IQR: 10.25–15.00; range 3–15): 24 patients (70.6%) had a mild GCS (13–15), 3 (8.8%) a moderate GCS (9–12), and 7 (20.6%) severe coma (GCS 3–8).

Overall, 7 patients died in the emergency department before inpatient admission or operative transfer, an emergency department mortality of 20.6% (Wilson 95% CI: 10.3–36.8%), and 27 patients (79.4%; Wilson 95% CI: 63.2–89.7%) survived to disposition. The severity distributions in Figure 2 show the divergence between anatomical injury burden and neurological status on arrival: 7 patients (20.6%) were in severe coma (GCS 3–8) and 7 (20.6%) had critical injury (ISS ≥ 25), the same number as the 7 emergency department deaths, whereas nearly two-thirds of the cohort (21/34; 61.8%) had minor-to-moderate injury (ISS 1–16) and more than seven-tenths (24/34; 70.6%) arrived with a mild GCS (13–15). Together with the predominance of blunt (30/34) and urban (31/34; 91.2%) injuries, these figures indicate a predominantly urban, blunt-injury cohort typical of South Sumatra.

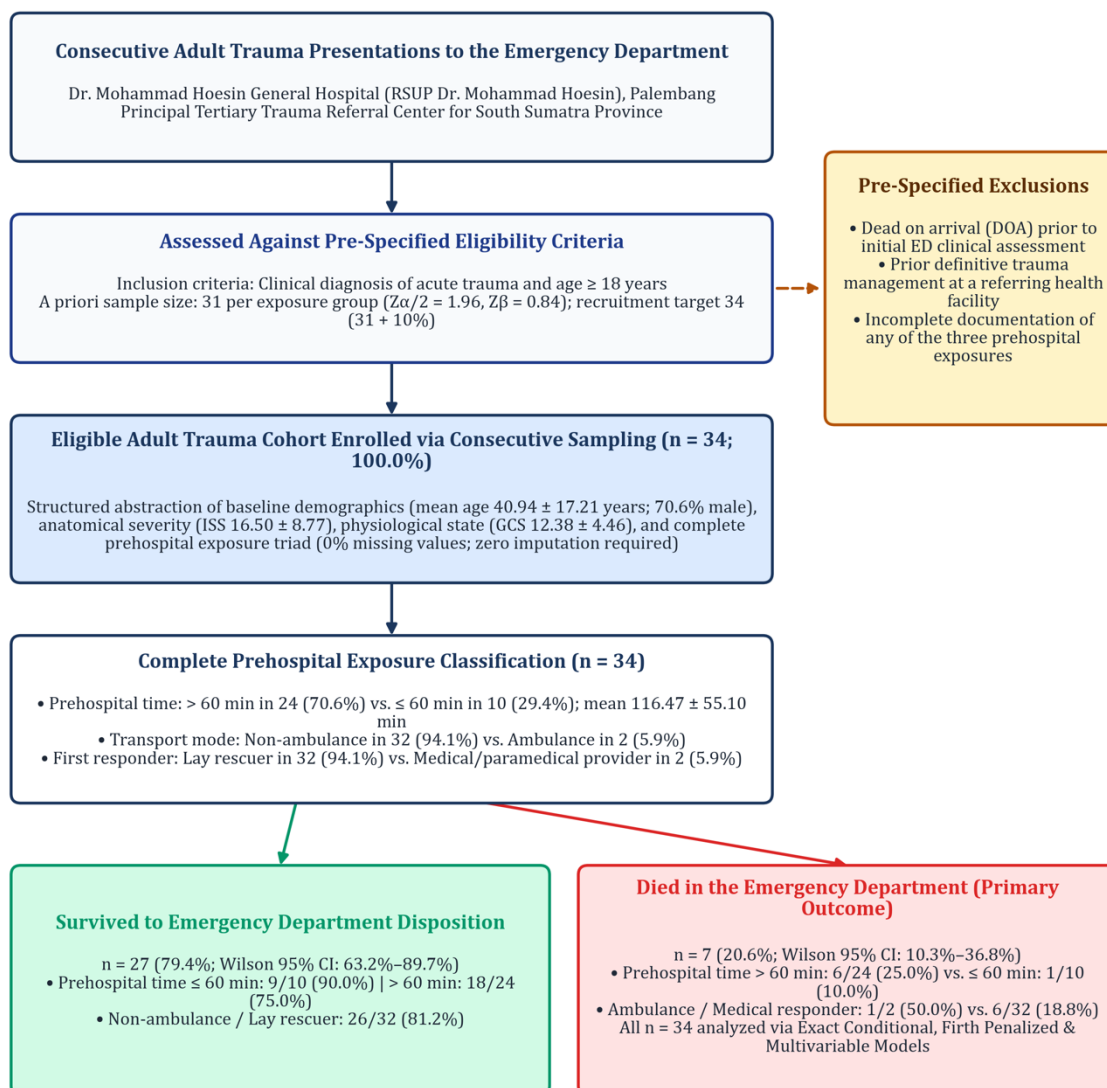


Figure 1. Participant flow through the study, reported in accordance with the STROBE statement for cross-sectional studies. All 34 consecutively enrolled adult trauma patients had complete data on the three prehospital exposures (first-responder type, prehospital time, and transport mode) and on emergency department mortality (7 deaths [20.6%; 95% CI: 10.3–36.8%] and 27 survivors [79.4%]). CI, confidence interval; DOA, dead on arrival; ED, emergency department; GCS, Glasgow Coma Scale; ISS, Injury Severity Score.

Table 1. Demographic, injury, and physiological characteristics of the adult trauma cohort (n = 34).

Characteristic	Category	n (%)	Summary statistics
Age group	Young adult, 19–24 years	7 (20.6)	Mean 40.94 ± 17.21 years (95% CI: 34.94–46.95) Median 39.50 years (IQR: 26.25–53.75; range 19–79) Shapiro–Wilk W = 0.964, p = 0.318
	Adult, 25–59 years	22 (64.7)	
	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)	Extremity or multiple regions: 14 (41.2)
	Extremity	7 (20.6)	
	Multiple regions	7 (20.6)	
	Thoracic	3 (8.8)	
	Abdominal	2 (5.9)	
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 (urban)	31 (91.2)	
	Outside the city (peri-urban/rural)	3 (8.8)	
Injury Severity Score (ISS)	Minor (1–9)	11 (32.4)	Mean 16.50 ± 8.77 (95% CI: 13.44–19.56) Median 14.00 (IQR: 9.00–22.00; range 4–41) ISS ≤16: 21 (61.8); ISS ≥17: 13 (38.2) Shapiro–Wilk W = 0.931, p = 0.034
	Moderate (10–16)	10 (29.4)	
	Severe (17–24)	6 (17.6)	
	Critical (≥25)	7 (20.6)	

Characteristic	Category	n (%)	Summary statistics
Glasgow Coma Scale (GCS)	Mild (13–15)	24 (70.6)	Mean 12.38 ± 4.46 (95% CI: 10.82–13.94)
	Moderate (9–12)	3 (8.8)	Median 15.00 (IQR: 10.25–15.00; range 3–15)
	Severe (3–8)	7 (20.6)	Shapiro–Wilk W = 0.654, p < 0.001
Trauma classification	Monotrauma (single region)	27 (79.4)	
	Multiple trauma (multiple systems)	7 (20.6)	
Emergency department outcome	Survived to disposition	27 (79.4)	Wilson 95% CI: 63.2–89.7%
	Died in the emergency department	7 (20.6)	Wilson 95% CI: 10.3–36.8%

Values are n (%) unless otherwise stated. Continuous variables are summarized as mean ± standard deviation (95% CI) and median n (interquartile range [IQR] and observed range), with Shapiro–Wilk W and p. The CIs for the outcome proportions are Wilson score intervals. CI, confidence interval.

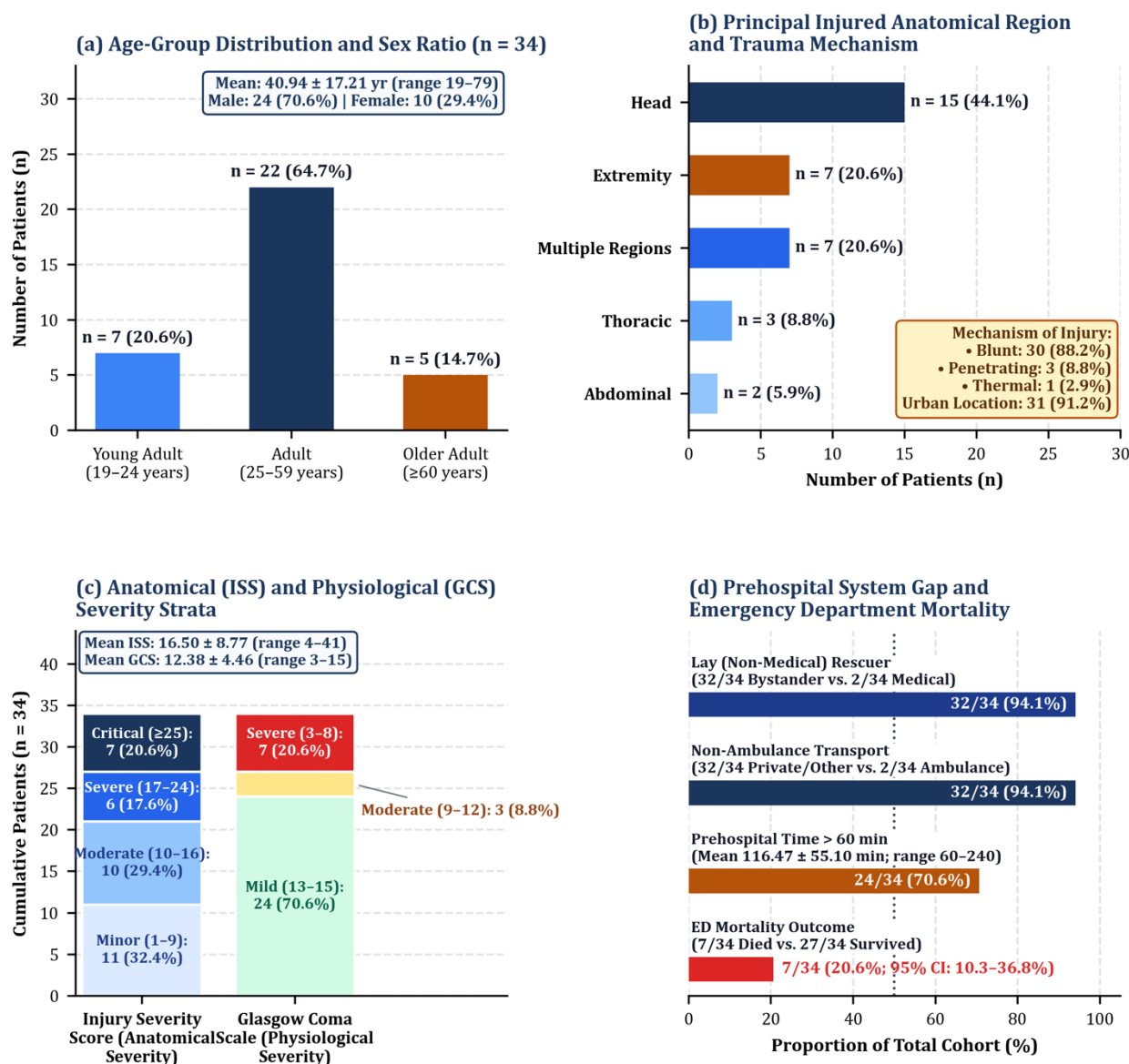


Figure 2. Demographic, anatomical, physiological, and prehospital exposure profile of the adult trauma cohort (n = 34). (a) Age groups and sex; (b) principal injured region and injury mechanism; (c) anatomical severity (Injury Severity Score, ISS) and physiological severity (Glasgow Coma Scale, GCS); and (d) prevalence of the prehospital exposures and emergency department (ED) mortality; the dotted line marks 50% of the cohort.

Prehospital exposure profile and system-level care gaps

Table 2 and Figure 2d show how the three prehospital exposures were distributed, and the gap in prehospital care is large. A lay bystander with no training gave the first help at the scene to 32 patients (94.1%; Wilson 95% CI: 80.9–98.4%); a medical or paramedical provider did so for only 2 (5.9%; Wilson 95% CI: 1.6–19.1%). Transport followed exactly the same split. Thirty-two patients (94.1%) came by non-ambulance vehicles (private cars, motorcycles, or informal transport), and 2 (5.9%)

by a dedicated ambulance. For 24 patients (70.6%) the prehospital time was longer than the 60-minute golden-hour threshold, while 10 (29.4%) reached hospital care at or within 60 minutes. Prehospital time was positively skewed (Shapiro–Wilk W = 0.884, p = 0.002). Its mean was 116.47 ± 55.10 minutes (95% CI: 97.25–135.70) and its median 120.00 minutes (IQR: 60.00–150.00; range 60–240 minutes), close to twice the 60-minute golden-hour benchmark.

Two distributional features of the recorded times need explicit statement. First, the shortest prehospital interval in any

record was exactly 60 minutes; the reference stratum of 10 patients classified as ≤ 60 minutes in Table 2 therefore consists entirely of patients who arrived at the 60-minute boundary rather than within the first hour, and the cohort contained no presentation earlier than 60 minutes. Second, the clustering of recorded intervals at multiples of 30 and 60 minutes (from 60 to 240 minutes) reflects retrospective estimation by patients or accompanying bystanders at triage rather than prospective electronic time-stamping. No patient (0 of 34) had the combination of arrival within 60 minutes, ambulance transport, and a trained medical or paramedical first responder, that is, all three favorable exposures. The floor at exactly 60 minutes and the ceiling at 240 minutes indicate that patients in the ≤ 60 -

minute reference group did not experience the rapid 15-to-30-minute urban paramedic response of high-income trauma networks; they represent the fastest-arriving tail of an informal, bystander-driven transport distribution whose central tendency approaches two hours. Furthermore, the identical counts of patients attended by a medical or paramedical responder (2, 5.9%) and of patients transported by ambulance (2, 5.9%) reflect close coupling in the prehospital field: in Palembang, formal medical or paramedical personnel rarely reach a trauma patient outside an ambulance transfer, so transport mode and responder qualification largely capture one health-system exposure.

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

Prehospital exposure	Category	n (%)	Summary statistics
First-responder type	Lay (non-medical) rescuer	32 (94.1)	Wilson 95% CI: 80.9–98.4%
	Medical or paramedical provider	2 (5.9)	Wilson 95% CI: 1.6–19.1%
Prehospital time	≤ 60 minutes (golden-hour threshold)	10 (29.4)	Mean 116.47 $\pm$ 55.10 minutes (95% CI: 97.25–135.70)
	> 60 minutes (prolonged prehospital time)	24 (70.6)	Median 120.00 minutes (IQR: 60.00–150.00; range 60–240) Shapiro–Wilk W = 0.884, p = 0.002
Transport mode	Ambulance	2 (5.9)	Wilson 95% CI: 1.6–19.1%
	Non-ambulance vehicle	32 (94.1)	Wilson 95% CI: 80.9–98.4%
All three favorable exposures	Prehospital time ≤ 60 min + ambulance + medical responder	0 (0.0)	No patient had all three

Prehospital time was the interval from the injury to the first medical care in the emergency department. Because the minimum observed prehospital time was exactly 60 minutes, all 10 patients in the ≤ 60 -minute category reached care at the 60-minute boundary rather than within it. CI, confidence interval; IQR, interquartile range.

Bivariate associations between prehospital exposures and mortality

The bivariate contingency distributions and complementary effect estimates are detailed in Table 3. Among the 24 patients whose prehospital time exceeded 60 minutes, 6 died in the emergency department (25.0%; Wilson 95% CI: 12.0–44.9%), compared with 1 of the 10 patients who reached hospital care at 60 minutes (10.0%; Wilson 95% CI: 1.8–40.4%). This corresponded to an absolute risk difference of +15.0 percentage points (Newcombe hybrid score 95% CI: –18.1 to +36.5), an unconditional crude OR of 3.00 (95% CI: 0.31–28.84), an exact conditional maximum-likelihood OR of 2.92 (exact 95% CI: 0.28–153.08), a Haldane–Anscombe OR of 2.23 (Wald 95% CI: 0.32–15.49), $\phi = 0.169$ (Pearson $\chi^2 = 0.971$; likelihood-ratio $G^2 = 1.081$; minimum expected cell count = 2.06), and a two-sided Fisher exact p value of 0.644. For transport mode, 1 of the 2 patients transported by ambulance died (50.0%; Wilson 95% CI: 9.5–90.5%), compared with 6 of the 32 patients transported by non-ambulance vehicles (18.8%; Wilson 95% CI: 8.9–35.3%), an absolute risk difference of +31.2 percentage points (Newcombe 95% CI: –12.5 to +73.0), an unconditional crude OR of 4.33 (95% CI: 0.24–79.59), an exact conditional OR of 4.09 (exact 95% CI: 0.05–352.85), a Haldane–Anscombe OR of 4.08 (Wald 95% CI: 0.36–45.87), $\phi = 0.182$ (Pearson $\chi^2 = 1.124$; $G^2 = 0.917$; minimum expected cell count = 0.41), and a Fisher exact p value of 0.374. The contingency table for first-responder type was

numerically identical to that for transport mode: 1 death among the 2 patients attended by a medical or paramedical responder versus 6 deaths among the 32 attended by lay rescuers, with the same effect estimates and p value.

Across all three exposures, every point estimate exceeded 1.00, yet every 95% CI included the null value (1.00, or 0.0 percentage points for risk differences) and spanned two to three orders of magnitude. Because the exposed strata for ambulance transport and medical first responder each contained only two patients (one survivor and one death) and had identical cell counts, these comparisons are descriptive proportions governed by severe sparse-data instability and near-collinearity rather than independent causal effect estimates. The four estimators in Table 3 and Figure 3 illustrate how sensitive small-sample contingency inference is to the choice of estimator. For prehospital time (6/24 versus 1/10 deaths), the unconditional sample OR is $(6 \times 9)/(18 \times 1) = 3.00$, the exact conditional estimate is 2.92 with an asymmetric exact CI, and the Haldane–Anscombe estimate is $(6.5 \times 9.5)/(18.5 \times 1.5) = 2.23$ with a symmetric log-scale Wald CI. For ambulance transport and medical first responder (1/2 versus 6/32 deaths), the corresponding values are $(1 \times 26)/(1 \times 6) = 4.33$, 4.09, and $(1.5 \times 26.5)/(1.5 \times 6.5) = 4.08$. Newcombe's hybrid score method for the risk difference avoids the overshoot below –100% or above +100% that affects Wald risk-difference intervals when a denominator is two.

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

Prehospital exposure	Died/total (%) [Wilson 95% CI]	Risk difference, % (Newcombe 95% CI)	Exact conditional OR (exact 95% CI)	Haldane– Anscombe OR (Wald 95% CI)	ϕ	p ^a
Prehospital time > 60 min	6/24 (25.0) [12.0–44.9]	15.0 (–18.1 to 36.5)	2.92 (0.28–153.08)	2.23 (0.32–15.49)	0.169	0.644
Prehospital time ≤ 60 min (ref.)	1/10 (10.0) [1.8–40.4]	Reference	1.00	1.00	—	—
Ambulance transport	1/2 (50.0) [9.5–90.5]	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) [8.9–35.3]	Reference	1.00	1.00	—	—

Prehospital exposure	Died/total (%) [Wilson 95% CI]	Risk difference, % (Newcombe 95% CI)	Exact conditional OR (exact 95% CI)	Haldane- Anscombe OR (Wald 95% CI)	ϕ	p^a
Medical first responder	1/2 [50.0] [9.5–90.5]	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] [8.9–35.3]	Reference	1.00	1.00	—	—

OR, odds ratio; CI, confidence interval; ϕ , phi coefficient (equivalent to Cramér's V for a 2×2 table); ref., reference category. ^aTwo-sided Fisher exact test, pre-specified because two of four expected cell counts were below five in every contingency table (minimum expected counts 2.06, 0.41, and 0.41, respectively). 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. Unconditional crude ORs were 3.00 (95% CI: 0.31–28.84) for prehospital time >60 min and 4.33 (95% CI: 0.24–79.59) for ambulance transport and medical first responder. Risk differences use the Newcombe hybrid score method without continuity correction.

Multivariable Likelihood-Ratio Test: $\chi^2 = 2.407$, df = 3, p = 0.492 | Cox & Snell $R^2 = 0.068$ | Nagelkerke $R^2 = 0.107$

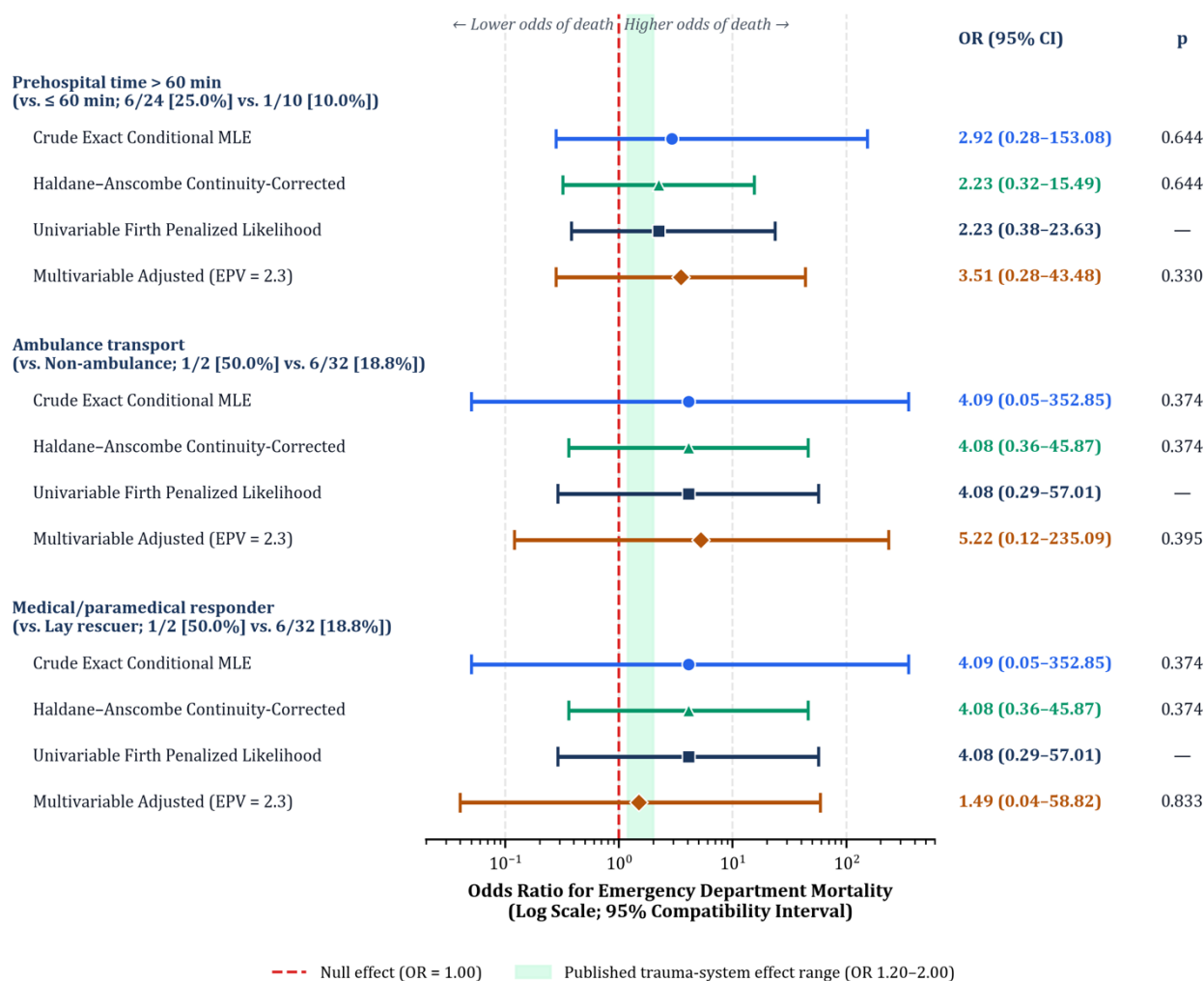


Figure 3. Multi-estimator forest plot of the association between each prehospital exposure and emergency department mortality (n = 34; 7 deaths), showing the exact conditional maximum-likelihood odds ratio (OR), the Haldane-Anscombe OR, the univariable Firth penalized-likelihood OR, and the multivariable adjusted OR with 95% compatibility intervals on a log scale. p values: Fisher exact test for the crude estimators and Wald test for the multivariable model. All intervals cross unity and span two to three orders of magnitude. EPV, events per variable; MLE, maximum-likelihood estimate.

Univariable Firth and multivariable logistic regression

To reduce small-sample bias and evaluate independent associations, univariable Firth penalized-likelihood regression and standard multivariable logistic regression were performed (Figure 3; Table 4). Firth's penalty shrank the crude maximum-likelihood point estimates toward the null and substantially narrowed the upper confidence bounds: the univariable Firth OR was 2.23 (profile penalized-likelihood 95% CI: 0.38–23.63) for prehospital time >60 minutes and 4.08 (95% CI: 0.29–57.01) for both ambulance transport and a medical or paramedical first responder. In a saturated univariable logistic model for a 2×2 table, Firth's point estimate is algebraically identical to the Haldane-Anscombe OR (2.23 and 4.08),⁵³ whereas its profile intervals reflect the asymmetry of the likelihood surface. When

all three exposures were entered together, the likelihood-ratio test showed that they jointly carried no significant information about emergency department mortality ($\chi^2 = 2.407$, df = 3, p = 0.492; –2 log-likelihood 32.167 against a null value of 34.575). The model explained only a negligible fraction of outcome variance (Cox and Snell $R^2 = 0.068$; Nagelkerke $R^2 = 0.107$). Expressed so that every adjusted OR refers to the exposed category, the estimates were: prehospital time >60 minutes, $\beta = 1.256$, SE = 1.289, Wald $\chi^2 = 0.949$, adjusted OR = 3.51 (95% CI: 0.28–43.48; p = 0.330); ambulance transport, $\beta = 1.653$, SE = 1.942, Wald $\chi^2 = 0.724$, adjusted OR = 5.22 (95% CI: 0.12–235.09; p = 0.395); medical first responder, $\beta = 0.397$, SE = 1.888, Wald $\chi^2 = 0.044$, adjusted OR = 1.49 (95% CI: 0.04–58.82; p = 0.833); and intercept, $\beta = -2.480$, SE = 1.185, Wald $\chi^2 = 4.381$, exp(β) = 0.08 (p = 0.036).

At a predicted-probability threshold of 0.500, overall classification accuracy was 76.5% (26 of 34 patients), lower than the 79.4% achieved by predicting survival for everyone: the model predicted survival for 33 of the 34 patients, and the single patient with a predicted probability of death above 0.500 survived, giving a specificity of 96.3% (26 of 27 survivors) and a sensitivity of 0% (0 of 7 deaths). With 7 deaths and 3 covariates, the EPV ratio was 2.3—far below the conventional minimum of 10 for asymptotic stability—and the collinearity between ambulance transport and medical first-responder attendance compounded the problem. Three features of Table 4 explain the combination of coefficient inflation and near-zero explanatory

power. First, adding the three covariates reduced the null $-2 \log$ -likelihood (34.575) to 32.167, by only 2.407 units on 3 degrees of freedom—well below the critical value of 7.815 for significance at $\alpha = 0.05$. Second, entering transport mode and first-responder type—which share identical margins and cell counts—together makes the information matrix nearly singular and inflates the standard errors of their slopes to 1.942 and 1.888. Third, because baseline survival was 79.4% (27/34), a model that classifies almost every patient as a survivor attains an apparently high accuracy while detecting no death, which shows why overall accuracy is misleading in imbalanced clinical data.

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

Exposure	Firth penalized OR (profile 95% CI) ^a	β	SE	Wald χ^2	Adjusted OR (95% CI) ^b	P
Prehospital 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
Intercept	—	−2.480	1.185	4.381	0.08 ^c	0.036

Model fit: $-2 \log$ -likelihood = 32.167 (null model 34.575); Cox and Snell $R^2 = 0.068$; Nagelkerke $R^2 = 0.107$. Likelihood-ratio test: $\chi^2 = 2.407$, $df = 3$, $p = 0.492$. Classification at a predicted-probability cut value of 0.500: sensitivity 0% (0 of 7 deaths), specificity 96.3% (26 of 27 survivors), overall accuracy 76.5% (26 of 34). OR, odds ratio; CI, confidence interval; SE, standard error. ^aUnivariable logistic regression with Firth's penalized likelihood; profile penalized-likelihood intervals at a convergence tolerance of 1.0×10^{-6} ; penalized likelihood-ratio $p = 0.395$, 0.267, and 0.267. ^bMultivariable model containing all three exposures; adjusted ORs refer to the exposed category in Table 3. ^c $\exp(\beta)$. Events per variable = 2.3 (7 events/3 covariates).

Precision analysis: power, minimum detectable effects, and reverse fragility

Because a non-significant p value in a small sample cannot distinguish the true absence of an effect from inadequate design sensitivity, the precision achieved by the study was quantified (Table 5; Figure 4). With the observed group sizes (24 versus 10 for prehospital time; 2 versus 32 for ambulance transport and medical first responder) and a two-sided α of 0.05, post-hoc power at the observed unconditional ORs was 12.6% for prehospital time >60 minutes (OR 3.00) and 23.1% for ambulance transport and medical first responder (OR 4.33) by the normal approximation, and 4.4% and 15.2% under the Fisher exact test. The minimum detectable OR—the smallest true effect that this sample of 34 patients with 7 deaths could have detected with 80% power at $\alpha = 0.05$ —was 12.57 for prehospital time and 69.82 for ambulance transport and medical first responder by the normal approximation; under the Fisher exact test it was about 16.2 for prehospital time, and 80% power could not be reached at any OR for the two-patient strata (maximum 60.7%). Figure 4b sets these thresholds against the observed ORs and the range of effects reported for trauma systems (OR 1.20–2.00):^{18,20,54} every observed and every

plausible trauma-system effect lies well below the detection threshold of the present sample.

The reverse fragility index complements these power estimates. Whereas the forward fragility index counts how many outcome events in a significant trial must be changed to make $p \geq 0.05$, the reverse fragility index counts how many outcomes in a non-significant study must be changed, within fixed group sizes, to make $p < 0.05$.⁵² For prehospital time (6/24 versus 1/10 deaths; $p = 0.644$), 7 of the 18 survivors in the >60-minute group would have had to die instead (13/24 [54.2%] versus 1/10 [10.0%]; two-sided Fisher exact $p = 0.024$)—an RFI of 7, equal to the total number of deaths in the study; if outcomes in both groups may change, 4 reassignments suffice (9/24 versus 0/10; $p = 0.034$). For ambulance transport and medical first responder (1/2 versus 6/32 deaths; $p = 0.374$), changing the single survivor in the exposed stratum to a death (2/2 [100.0%] versus 6/32 [18.8%]; two-sided Fisher exact $p = 0.050$ [0.0499]) crosses the significance threshold, an RFI of 1 that shows how fragile inference on a two-patient stratum is. Table 5 and Figure 4 therefore show that the null results reflect severe imprecision rather than evidence that prehospital exposures do not influence trauma mortality.

Table 5. Precision analysis: power, minimum detectable effect, and reverse fragility index for each prehospital exposure (n = 34).

Prehospital exposure	Exposed/unexposed (n)	Observed crude OR	Post-hoc power, % (normal / Fisher exact) ^a	Minimum detectable OR at 80% power (normal / Fisher exact) ^a	Reverse fragility index ^b
Prehospital time >60 min	24/10	3.00	12.6 / 4.4	12.57 / 16.2	7
Ambulance transport	2/32	4.33	23.1 / 15.2	69.82 / not attainable ^c	1
Medical first responder	2/32	4.33	23.1 / 15.2	69.82 / not attainable ^c	1

OR, odds ratio. ^aTwo-sided $\alpha = 0.05$ and the observed group sizes; normal, two-proportion normal approximation with pooled variance; Fisher exact, exact power of the two-sided Fisher test. Post-hoc power refers to the unconditional effect observed (OR = 3.00 and 4.33); the minimum detectable OR is the smallest true effect detectable with 80% power. ^bSmallest number of outcome changes in the exposed group (group sizes fixed) needed for the two-sided Fisher exact test to reach $p < 0.05$; if outcomes in both groups may change, the index for prehospital time is 4. ^cMaximum attainable power 60.7%. Published trauma-system effect sizes lie between approximately 1.2 and 2.0,^{18,20,54} well below every detectability threshold in this table.

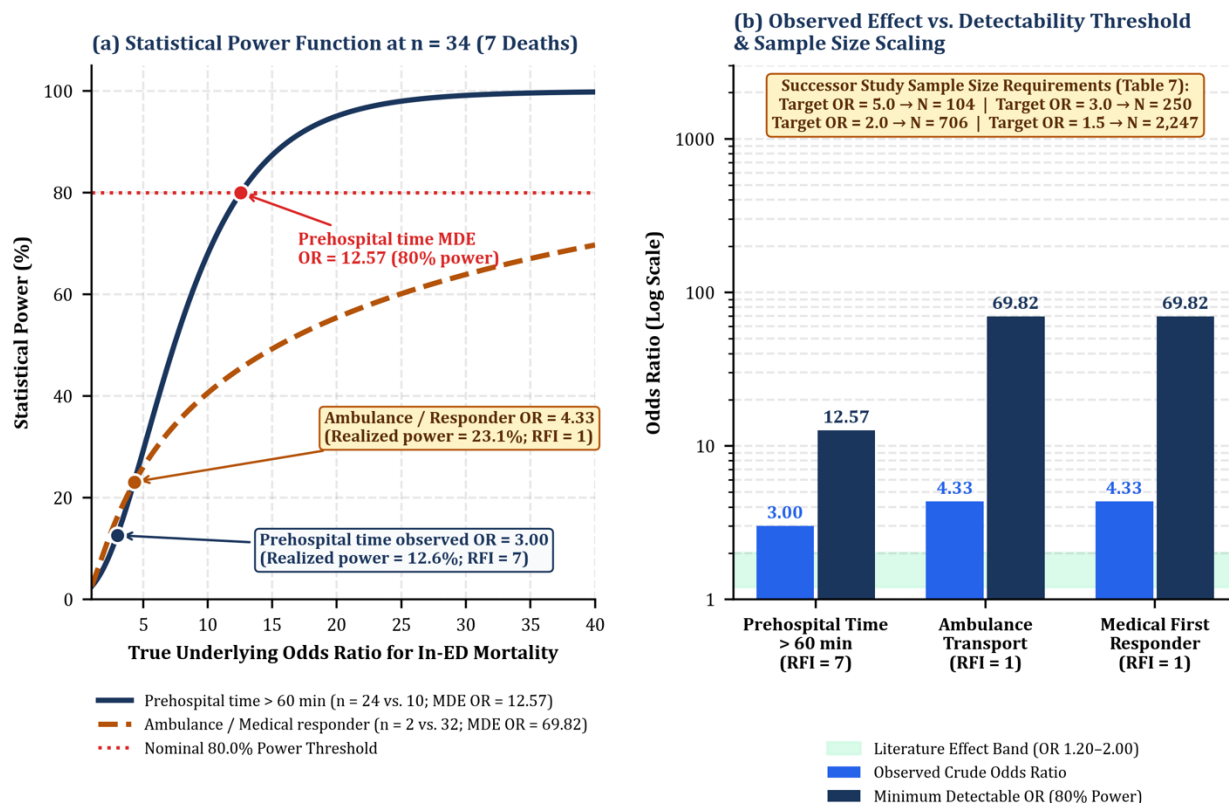


Figure 4. Precision achieved by the present study design (n = 34; 7 deaths). (a) Power (two-proportion normal approximation with pooled variance) as a function of the true odds ratio (OR), with the observed effects marked for prehospital time (OR = 3.00; power 12.6%; reverse fragility index [RFI] = 7) and ambulance/medical responder (OR = 4.33; power 23.1%; RFI = 1); and (b) observed ORs compared with the minimum OR detectable at 80% power (12.57 and 69.82) and the published trauma-system effect band (OR 1.20–2.00). MDE, minimum detectable effect; ED, emergency department.

4. Discussion

The principal contribution of this study is descriptive, epidemiological, and methodological rather than inferential. In a consecutive cohort of 34 adult trauma patients at the apex tertiary surgical referral hospital of South Sumatra Province, 94.1% (32/34) received initial on-scene assistance from an untrained lay bystander, 94.1% (32/34) were transported in a non-ambulance vehicle, and 70.6% (24/34) had prehospital times over 60 minutes, with a mean of 116.47 ± 55.10 minutes (median 120.00 minutes)—nearly twice the 60-minute benchmark.^{1,5,12} No patient had the combination of arrival within 60 minutes, ambulance transport, and a trained medical or paramedical responder. Against this background, emergency department mortality was substantial at 20.6% (7/34; 95% CI: 10.3–36.8%), yet neither prehospital time >60 minutes (exact conditional OR 2.92; Firth OR 2.23; $p = 0.644$), ambulance transport (exact conditional OR 4.09; Firth OR 4.08; $p = 0.374$), nor a medical first responder (exact conditional OR 4.09; Firth OR 4.08; $p = 0.374$) was significantly associated with mortality in bivariate or multivariable analysis (likelihood-ratio $p = 0.492$). Rather than allowing $p > 0.05$ to obscure the prehospital deficit documented here, the precision and Firth penalized-likelihood analyses explain why these null findings arise and how they should guide regional trauma-system reform. In LMIC surgical settings where single-center dissertations and institutional series are often the only local data, reporting the precision of a small cohort helps prevent an underpowered $p > 0.05$ from being read as reassurance about prehospital delay, while preserving the descriptive baseline needed to design a registry of several hundred patients.^{41–43}

Four epidemiological and biostatistical considerations govern the interpretation of these neutral tests. First, and most

decisively, the study was severely underpowered to detect any realistic clinical or health-system effect. The original sample-size calculation (31 per group) assumed mortality rates of 65.0% in the exposed stratum and 30.0% in the reference stratum; the observed mortality for prolonged versus shorter prehospital time was 25.0% (6/24) and 10.0% (1/10). As shown in Table 5 and Figure 4, this gap between assumed and observed event rates reduced power to 12.6%–23.1% (normal approximation) and raised the minimum detectable OR at 80% power to 12.57 for prehospital time and 69.82 for transport mode and first-responder type. Because adjusted ORs reported for prehospital time and transport exposures in large trauma-system studies are of the order of 1.2–2.0,^{18,20,54} a sample of 34 patients with 7 deaths was unlikely to detect an effect of plausible clinical magnitude. A non-significant p value obtained with low power carries little evidential weight in favor of the null hypothesis;^{46,51} the appropriate summary is the compatibility interval, which for prolonged prehospital time (exact 95% CI: 0.28–153.08; Firth profile 95% CI: 0.38–23.63) is compatible with anything from a moderate protective effect to a more than twenty-fold increase in the odds of emergency department death. Second, the extreme exposure imbalance (2/34 patients [5.9%] with ambulance transport or a medical responder) meant that those two contingency tables rested on a single death among two exposed patients (RFI = 1) and were closely coupled, which made multivariable adjustment (EPV = 2.3) unreliable even with Firth penalization.⁴⁵ Third, the cohort was skewed toward non-critical injury: 61.8% (21/34) had minor or moderate ISS (≤ 16) and 70.6% (24/34) a mild GCS on arrival (13–15), which dilutes any time-sensitive hemorrhagic or airway mortality signal toward the null.⁵⁵ Fourth, all point estimates in the bivariate and multivariable models exceeded unity (OR 2.23–5.22), a pattern typical of confounding by

indication and severity: where ambulance use is only 5.9%, ambulances and medical responders are selectively summoned to the most severely injured casualties, and complex vehicular extrication in severe crashes simultaneously prolongs prehospital time and increases mortality.^{18,54,56}

Confounding by indication (also termed selective referral or triage channeling) may be especially strong where EMS coverage is limited.^{20,21} When 94.1% of injured people travel to hospital in private cars or on motorcycles, the rare decision to summon and wait for an ambulance or medical responder (5.9% of cases) is almost never random; it is usually prompted by overt catastrophic injury, deep coma, or hemodynamic collapse at the scene. Unadjusted or under-adjusted comparisons between ambulance and non-ambulance arrivals therefore compare the most severely injured patients with walking-wounded or moderately injured casualties, which may generate positive crude ORs (4.08–5.22) through baseline severity rather than through any effect of the vehicle.^{1,18,20,21}

When set against contemporary trauma-systems research (Table 6), the findings agree with some comparator cohorts and not with others. In the retrospective cohort of Wu et al. ($n = 13,533$), the covariate-adjusted association between on-scene time and in-hospital mortality disappeared after adjustment for the type and number of prehospital interventions, which themselves had mixed associations with mortality.¹³ The Norwegian national cohort of Nilsbakken et al. ($n = 28,988$) showed that the longer prehospital intervals of remote regions did not carry excess case-mix-adjusted mortality, although prolonged on-scene time did.¹⁴ Berkeveld et al. ($n = 342$) in the Netherlands found no association between prehospital time and mortality,¹⁶ whereas Siripakarn et al. ($n = 104$) in Thailand found scene time over 10 minutes independently associated with 24-hour mortality.¹⁵ In pediatric trauma (Rickenbach et al., $n = 60,670$), adjusted mortality fell with prehospital time up to about 45 minutes and then plateaued.¹⁷ The 50.0% (1/2) mortality among ambulance-transported and medically attended patients in the present cohort resembles the nationwide Japanese cohort of Yamamoto et al. ($n = 14,652$), in which physician-staffed ambulance care was associated with higher mortality in hypotensive patients—an excess the authors attributed to a longer prehospital stay.¹⁸ Rahhal et al. ($n = 5,316$)

found no survival penalty for police transport relative to ground ambulance in US trauma centers,¹⁹ Lapidus et al. found lower injury-stratified mortality with helicopter transport in Sweden,²² and the meta-analysis by Anvari et al. found higher crude mortality with helicopter transport while noting the selection of more severely injured patients.¹

The apparent discrepancy between hospital-based studies with null time effects and scene-based registries with high prehospital lethality is explained by survivor conditioning (left truncation): in the East Azerbaijan road-traffic injury registry of Sadeghi-Bazargani et al. ($n = 7,347$ deaths), 62.5% of road-traffic deaths occurred before hospital arrival.⁷ Because the present study excluded dead-on-arrival casualties by design, the cohort is conditioned on surviving a mean prehospital interval of 116.47 minutes without formal EMS care, and physiological and composite anatomical-physiological measures—early TQIP vital-sign thresholds,²³ emergency department multi-trauma scores,²⁴ age-adjusted GCS,²⁵ prehospital GAP/MGAP/T-RTS tools,²⁶ and the rSIG, validated in children⁵⁷—are potentially better discriminators of early hospital death than elapsed time, although this was not tested here. Comparison with these studies (Table 6) and with regional Southeast Asian trauma epidemiology suggests shared structural deficits across developing trauma networks in Indonesia, Thailand, Iran, and sub-Saharan Africa.^{5,7,15,41–43} In mature trauma systems such as those of Sweden, Norway, and the Netherlands, prehospital EMS coverage is broad, response times are short, and physician-staffed or paramedic teams provide advanced on-scene care, including helicopter retrieval in rural areas; under those conditions, variations in scene or transport time largely reflect on-scene procedures or rural geography.^{12,14,16,22} By contrast, in South Sumatra and comparable LMIC catchments where 94.1% of patients arrive by lay bystander transport without any en-route intervention, prolonged prehospital times (mean 116.47 minutes) may act as a filter: the most severely bleeding or asphyxiating casualties die at the roadside or in transit (survivor conditioning),⁷ whereas the survivors who reach hospital are a selected subpopulation whose outcomes may depend more on ISS and GCS than on differences between 60 and 120 minutes of transit.^{10,11}

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

Study and year	Design and setting	Sample size (n)	Principal finding	Consistency with the present study
Wu et al., 2025 ¹³	Retrospective cohort, four hospitals, Taiwan	13,533	Association between on-scene time and in-hospital mortality disappeared after adjustment for the type and number of prehospital interventions	Consistent; supports specific interventions rather than elapsed time as the operative factor
Nilsbakken et al., 2023 ¹⁴	National population-based cohort, Norway	28,988	Longer prehospital times in remote settings not accompanied by excess adjusted mortality; prolonged on-scene time associated with higher adjusted mortality	Partly consistent; remoteness was not harmful, but on-scene time mattered
Siripakarn et al., 2023 ¹⁵	Retrospective single-center cohort, EMS-transported major trauma, Thailand	104	Scene time >10 min independently associated with 24-h mortality (risk ratio 4.47; 95% CI 1.84–10.83)	Not consistent; closest regional Southeast Asian analogue
Berkeveld et al., 2021 ¹⁶	Retrospective cohort, level I trauma center, Netherlands	342	Mean prehospital times nearly identical in survivors (45.3 min) and non-survivors (44.9 min; adjusted $p = 0.156$)	Consistent; no linear prehospital time–mortality gradient in hospital-arriving polytrauma
Rickenbach et al., 2024 ¹⁷	Retrospective registry cohort (TQIP), pediatric trauma, United States	60,670	Non-linear association: adjusted mortality fell as prehospital time rose to about 45 min, then plateaued	Partly consistent; no simple linear time effect
Yamamoto et al., 2021 ¹⁸	Nationwide cohort of hypotensive patients, Japan (JTDB)	14,652	Physician-staffed ambulance associated with higher in-hospital mortality (adjusted OR 1.22), attributed by the authors to a longer prehospital stay	Same direction as the ambulance and medical-responder estimates; mechanism differs
Rahhal et al., 2025 ¹⁹	Matched cohort, 2017 National Trauma Data Bank, United States	5,316	Similar survival after police and ground ambulance transport in blunt trauma	Consistent; non-ambulance arrival is not inherently harmful

Study and year	Design and setting	Sample size (n)	Principal finding	Consistency with the present study
Lapidus et al., 2023 ²²	National cohort, helicopter vs. road ambulance, Sweden	74,032	Lower injury-stratified mortality with helicopter transport (risk ratios 0.44–0.75) despite longer prehospital times	Not directly comparable; transport mode can matter when advanced care is delivered en route
Anvari et al., 2026 ¹	Systematic review and meta-analysis	Pooled (29 studies)	Higher crude in-hospital mortality with helicopter transport (OR 1.27); benefit uncertain	Consistent; transport-mode effects remain unresolved even at meta-analytic scale
Sadeghi-Bazargani et al., 2022 ⁷	Road traffic injury registry, East Azerbaijan Province, Iran	7,347	62.5% (4,589/7,347) of road-traffic deaths occurred before hospital arrival	Explains survivor conditioning (left truncation of DOA cases) that attenuates prehospital effects in hospital-based designs
Present study, 2026	Retrospective cross-sectional study, Palembang, Indonesia	34	ED mortality 20.6% (7/34); 94.1% lay rescuer; 94.1% non-ambulance; 70.6% prehospital time >60 min (mean 116.47 min); no significant association; MDE OR 12.57	Uninformative rather than negative; wide compatibility intervals remain compatible with clinically important effects

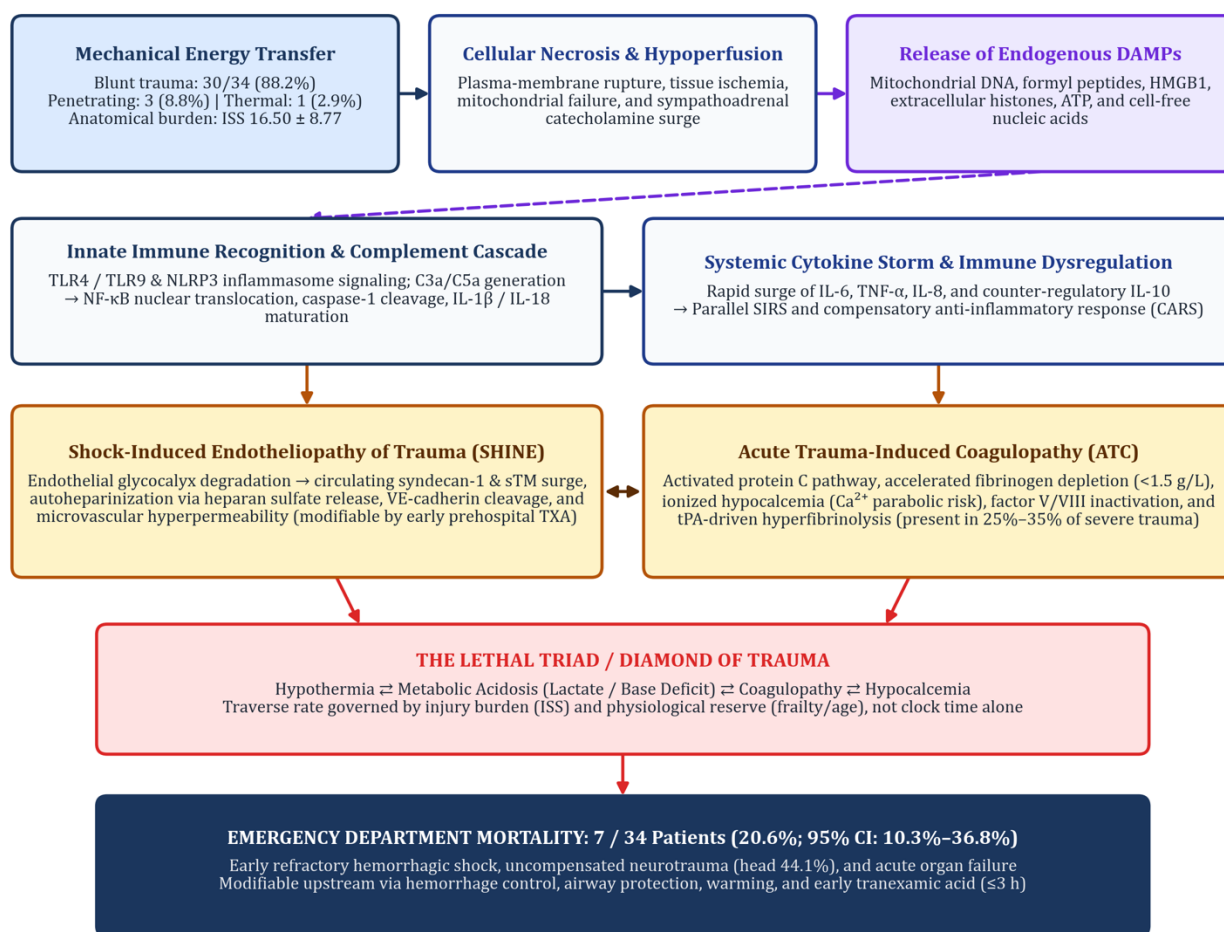
CI, confidence interval; DOA, dead on arrival; ED, emergency department; EMS, emergency medical services; JTDB, Japan Trauma Data Bank; MDE, minimum detectable effect; OR, odds ratio; TQIP, Trauma Quality Improvement Program. Sample sizes are those reported in the source publications (eligible or analyzed patients; Sadeghi-Bazargani et al.: deaths). Consistency is assessed with respect to the direction, confounding structure, and epidemiological interpretation of the estimates rather than dichotomous *p*-value thresholds.

No circulating biomarkers—arterial lactate, base deficit, plasma fibrinogen, ionized calcium, viscoelastic testing, or soluble syndecan-1—were measured in this retrospective cohort; the molecular framework summarized in Figure 5 offers a possible biological explanation of why prehospital time alone performs poorly as a univariable predictor of emergency department death. As depicted in Figure 5a, mechanical energy transfer (blunt in 88.2% of this cohort) causes immediate cellular necrosis, plasma-membrane rupture, and tissue hypoperfusion, releasing damage-associated molecular patterns (DAMPs)—including mitochondrial DNA, formyl peptides, high-mobility group box-1 (HMGB1), extracellular histones, and adenosine triphosphate—into the circulation. These DAMPs engage pattern-recognition receptors (TLR4, TLR9, and the NLRP3 inflammasome) and activate the complement cascade (C3a and C5a), driving nuclear factor- κ B (NF- κ B) translocation, caspase-1 cleavage, and a biphasic release of pro-inflammatory (IL-6, TNF- α , IL-8) and counter-regulatory (IL-10) cytokines.^{58,59} Downstream, this neurohumoral and inflammatory surge converges on two coupled vascular-hemostatic axes: SHINE and ATC.^{27,29,34} In the vascular lumen, catecholamine-driven cleavage of the endothelial glycocalyx sheds syndecan-1 and heparan sulfate into plasma (with endogenous autoheparinization and capillary leak) and increases soluble thrombomodulin (sTM), which complexes with thrombin to activate protein C.^{31–34} Activated protein C inactivates factors Va and VIIIa and neutralizes plasminogen activator inhibitor-1 (PAI-1), releasing tissue plasminogen activator (tPA)-driven hyperfibrinolysis alongside rapid fibrinogen depletion (<1.5 g/L)^{29,60} and ionized calcium derangement.³⁰ Together with hypothermia and hypoperfusion-induced metabolic acidosis, ATC forms the lethal triad (or lethal diamond when ionized calcium is included) shown in Figure 5, which can culminate in refractory hemorrhagic shock or organ dysfunction. As shown in Figure 5b, when published estimates of the timing of DAMP release, the early IL-6 and TNF- α surge, syndecan-1 shedding, and established ATC^{60–62} are set against the cohort's prehospital time distribution (mean 116.47 $\pm$ 55.10 minutes; 70.6% >60 minutes), patients with severe injury and low physiological reserve would be expected to traverse this cascade well before reaching hospital, whereas patients with isolated minor injuries (61.8% with ISS $\leq$ 16 in this series) may tolerate a two-hour

delay with little endothelial or hemostatic perturbation.³⁴ This biological heterogeneity—illustrated by the high-mortality SHINE phenotype identified by Johansson et al.³⁴ and by trials showing that prehospital tranexamic acid lowers syndecan-1³⁵ and 28-day mortality (a secondary outcome),⁶³ with a benefit that declines with delay and is not seen after 3 hours^{36,60}—suggests that interventions targeting the molecular cascade matter more than transport speed alone.

The three collaborating departments—Surgery, Orthopaedics, and Biology—meet in the pathophysiology of trauma-induced coagulopathy and shock-induced endotheliopathy.^{27,29,34} When blunt energy (88.2% of this cohort) disrupts soft tissue, long bone, and parenchymal organs, the release of nuclear and mitochondrial DAMPs (HMGB1, histones H3 and H4, and mitochondrial DNA) activates endothelial Toll-like receptors and neutrophil extracellular traps (NETs), amplifying systemic inflammation.^{58,64} At the same time, hemorrhagic hypovolemia triggers an intense sympathoadrenal surge in circulating adrenaline (median 950–1,370 pg/mL in the two highest-shedding SHINE phenotypes),³⁴ which activates endothelial metalloproteinases and heparanases that cleave the luminal glycocalyx. The resulting rise in circulating syndecan-1 (a cut-off of 130.4 ng/mL identified overt disseminated intravascular coagulation in one series)³³ strips the endothelium of its antithrombotic and barrier function, releases heparan sulfate proteoglycans that cause autoheparinization, and exposes sTM.^{31–34} As thrombin binds sTM on the denuded endothelium, protein C is converted into activated protein C, which cleaves activated factors V and VIII and consumes PAI-1, unleashing tPA-mediated hyperfibrinolysis and rapid fibrinogen depletion.^{27,29} Tissue hypoperfusion also drives anaerobic glycolysis and lactic acidemia, and ionized hypocalcemia (<1.10 mmol/L), measured before transfusion in 30,183 TraumaRegister DGU patients, showed a parabolic relationship with coagulopathy and mortality.³⁰ Because tranexamic acid inhibits plasminogen activation and attenuates syndecan-1 shedding in a dose-dependent manner, with age-dependent endothelial biomarker responses,^{35,39,60} measuring biological markers on arrival may be more informative for surgical resuscitation than recording elapsed time alone.

(a) Molecular and Pathophysiological Cascade Linking Mechanical Energy Transfer to Early Emergency Department Mortality



(b) Published Molecular Kinetics Set Against the Observed Prehospital-Time Distribution in South Sumatra (n = 34)

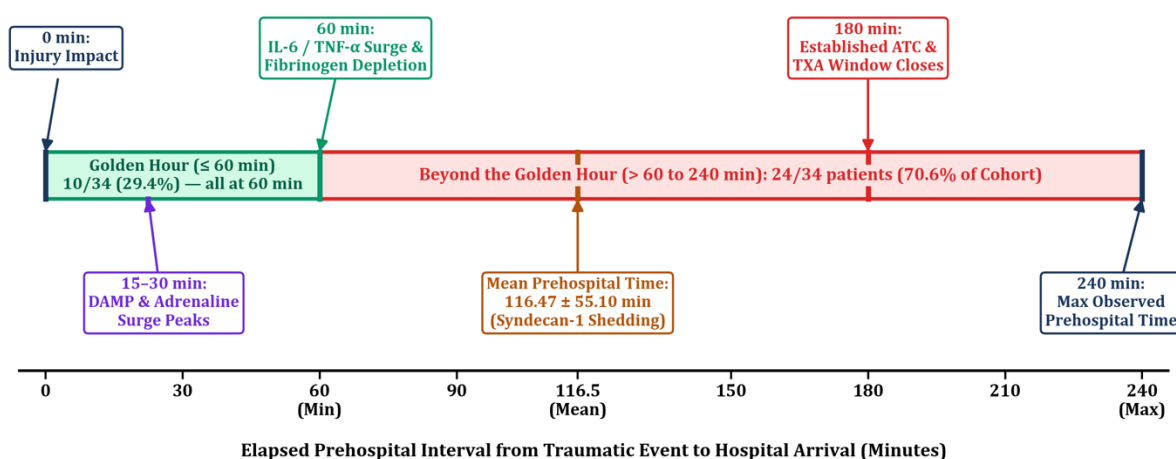


Figure 5. Conceptual molecular and pathophysiological framework linking mechanical injury to early emergency department death (not measured in this cohort). (a) The cascade from mechanical energy transfer and release of damage-associated molecular patterns (DAMPs) through innate immune activation, shock-induced endotheliopathy of trauma (SHINE; syndecan-1 shedding), and acute trauma-induced coagulopathy (ATC; fibrinogen depletion and ionized hypocalcemia) to the lethal triad and death; and (b) a schematic of the published time course of that cascade set against the prehospital times observed in this cohort (mean 116.47 ± 55.10 minutes; range 60–240 minutes). TXA, tranexamic acid.

To translate these epidemiological and biological insights into institutional reform at Dr. Mohammad Hoesin General Hospital, Figure 6 outlines an integrated, three-lane clinical and research pathway uniting the Departments of Surgery, Orthopaedics, and Biology at the Faculty of Medicine, Universitas Sriwijaya. In the surgical domain, emergency department reception should pair

the structured ATLS primary survey with immediate physiological risk stratification, incorporating simple equipment-free perfusion markers such as central capillary refill time (CRT), which secondary analyses of CRASH-2 associate with adult trauma mortality,⁶⁵ alongside locally evaluated scoring systems such as the Thoracic Trauma Severity

Score (TTSS)¹¹ and the Blunt Abdominal Trauma Scoring System (BATSS),⁶⁶ both of which still require validation. In the orthopedic domain—which concerns the 41.2% of this cohort with primary extremity trauma (7/34; 20.6%) or multi-region trauma (7/34; 20.6%)—a mean prehospital delay of 116.47 minutes without trained splinting or wound coverage has consequences beyond mortality. Open long-bone fractures arriving nearly two hours after injury without sterile dressing or early antibiotic prophylaxis carry higher risks of deep infection, compartment syndrome, and neurovascular compromise,⁶⁷ and unsplinted femoral shaft or unstable pelvic ring fractures may continue to bleed during private-vehicle transport, producing the acidosis, coagulopathy, and shock that determine whether Early Appropriate Care (EAC) definitive osteosynthesis or damage-control external fixation is indicated—a relationship suggested by the institution's 4.5-year analysis of pelvic ring fracture mortality, which proposed delays in care as a contributor.⁶⁸ In the biology domain, a rapid point-of-care panel at triage (arterial lactate, base deficit, Clauss fibrinogen, and ionized calcium, supplemented by research biobanking of circulating syndecan-1 and soluble thrombomodulin) would allow objective phenotyping of physiological reserve and endotheliopathy on arrival (Figure 6).^{27,30,31,34}

The orthopedic perspective in Figure 6 is particularly relevant because 41.2% of the patients had primary extremity or multi-region injuries and 44.1% head trauma. In

musculoskeletal trauma care, the debate between Early Total Care (ETC) and Damage Control Orthopaedics (DCO) has evolved into EAC, in which the choice between definitive intramedullary nailing or open reduction and internal fixation within 24–36 hours and staged external fixation depends not on elapsed prehospital hours but on objective resuscitation endpoints—lactate <4.0 mmol/L, arterial pH ≥ 7.25 , or base excess ≥ -5.5 mmol/L—and on the absence of progressive coagulopathy.^{69,70} The prehospital environment nevertheless shapes whether a patient arrives within the safe physiological window for EAC: when a patient with an unstable pelvic ring fracture or a comminuted femoral shaft fracture endures 116.47 minutes of unsplinted transport in a private car, repeated fracture-site motion may enlarge retroperitoneal and thigh hematomas, accelerate fat embolization and DAMP release, and deepen lactic acidosis and coagulopathy on arrival, a hypothesis that requires prospective testing. For open Gustilo–Anderson type II and III fractures, which are common after motorcycle collisions, a two-hour delay without sterile dressing, removal of gross contamination, or prompt intravenous cephalosporin or aminoglycoside administration allows bacterial adherence to devitalized bone well before surgical debridement can begin.⁶⁷ Thus, even when prehospital delay does not alter emergency department survival detectably in a small sample, it may influence limb salvage, fracture-related infection, compartment syndrome, and intensive care length of stay—endpoints that should be captured in the successor registry (Figure 6).

Proposed Interdisciplinary Prehospital, Emergency Department, and Regional Trauma Registry Pathway
Dr. Mohammad Hoesin General Hospital & Faculty of Medicine, Universitas Sriwijaya (Surgery • Orthopaedics • Biology)

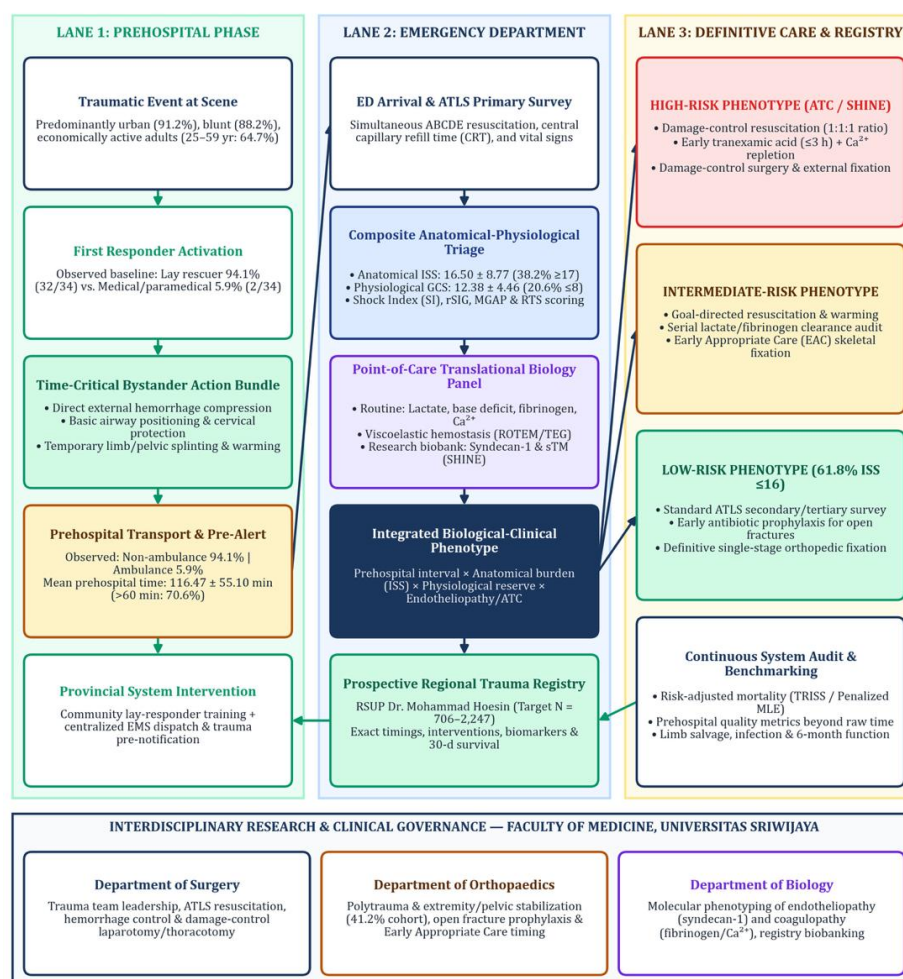


Figure 6. Proposed interdisciplinary prehospital, emergency department, and definitive trauma care pathway for Dr. Mohammad Hoesin General Hospital, illustrating the complementary roles of the Departments of Surgery, Orthopaedics, and Biology at the Faculty of Medicine,

Universitas Sriwijaya, together with the prospective regional trauma registry feedback loop. ATLS, Advanced Trauma Life Support; ED, emergency department; EMS, emergency medical services.

The strengths and limitations of this study must be weighed transparently, and they lead directly to the sample-size estimates for a successor provincial registry presented in Table 7. Strengths include consecutive sampling over a defined clinical window, complete data for all 34 patients without imputation, multi-estimator bivariate analysis (exact conditional MLE, Haldane–Anscombe correction, and Newcombe score intervals), Firth penalized-likelihood regression to reduce small-sample bias,^{45,48,50} and explicit quantification of precision through minimum detectable ORs and reverse fragility indices.^{51,52} The main limitation is the small sample of 34 patients with 7 deaths (EPV = 2.3), which limited power to 12.6%–23.1% (normal approximation; 4.4%–15.2% by the exact test) and produced wide compatibility intervals. Other limitations are the single-center cross-sectional design; survivor bias from the exclusion of dead-on-arrival casualties and secondary referrals; the absence of screening-log counts for excluded subgroups; retrospective triage estimation of prehospital times, with clustering at multiples of 30 minutes between 60 and 240 minutes; single-investigator chart abstraction without inter-rater κ ; the lack of continuous arrival vital signs (precluding the Revised Trauma Score [RTS] or the Trauma and Injury Severity Score [TRISS]) and of prehospital intervention logs; and the absence of biomarker measurements and of 30-day functional and orthopedic follow-up after disposition.

To overcome these limitations, Table 7 gives the sample sizes required for a prospective regional trauma registry at Dr. Mohammad Hoesin General Hospital, assuming the observed unexposed mortality (10.0%), the observed prevalence of prolonged prehospital time (70.6%), two-sided $\alpha = 0.05$, and 80% power: $n = 100$ reduces the minimum detectable OR from 12.57 to 5.11 and $n = 300$ to 2.76, whereas detecting ORs of 2.0 and 1.5 with 80% power requires 706 and 2,247 patients, respectively. Because the baseline mortality of 10.0% rests on 1 death among 10 patients (Wilson 95% CI: 1.8–40.4%), these

projections are approximate. As registry design frameworks, cost models, and WHO minimum dataset recommendations for LMIC hospitals have been described,^{41–43} a prospective, biomarker-augmented South Sumatran trauma registry combined with systematic community lay-responder first-aid training is a feasible priority for provincial surgical policy.

Implementing the registry roadmap in Table 7 at Dr. Mohammad Hoesin General Hospital requires a phased, resource-conscious strategy suited to South Sumatra's health economy.^{41–43} In Phase 1 (immediate community and triage action), the Faculty of Medicine, Universitas Sriwijaya, could partner with municipal traffic police, motorcycle-taxi (ojek) associations, and community volunteers in Palembang—who currently constitute the lay first-responder workforce of 94.1%—to deliver structured half-day training in four zero-capital interventions: direct manual compression and improvised tourniquet application for extremity hemorrhage, jaw-thrust airway positioning with manual cervical spine stabilization, improvised rigid splinting of long-bone fractures, and thermal blanket wrapping to prevent hypothermia.^{5,71,72} At the same time, emergency department triage documentation at RSUP Dr. Mohammad Hoesin could be upgraded at no capital cost to require exact screening and exclusion logs, continuous vital signs, central CRT,⁶⁵ the shock index (SI), rSIG,⁵⁷ and GAP/MGAP scores,²⁶ alongside the TTSS¹¹ and BATSS⁶⁶ scores. In Phase 2 (biomarker-augmented registry deployment), using published LMIC registry costing and architecture models,^{41–43} a digital trauma registry embedded in the emergency department workflow could prospectively enroll consecutive trauma arrivals toward the Table 7 milestones of 706 (for OR 2.0) and 2,247 patients (for OR 1.5), pairing routine point-of-care lactate, base deficit, fibrinogen, and ionized calcium assays with targeted ELISA biobanking of syndecan-1 and soluble thrombomodulin in severe trauma (ISS ≥ 16) to establish a molecularly phenotyped regional trauma registry in Indonesia.

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 (N)	Exposed/unexposed split (n) (at 70.6% exposure prevalence)	Smallest OR detectable at 80% power (MDE)	Target literature OR	Total sample size required for 80% power ($\alpha = 0.05$)
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	—	—

OR, odds ratio; MDE, minimum detectable effect. Columns 1–3 give the MDE at each total sample size; columns 4–5 give the total sample size required for each target OR (the two sets of rows are independent). All calculations use the two-proportion normal approximation with pooled variance and assume the baseline mortality of 10.0% observed in the unexposed stratum (prehospital time ≤ 60 minutes; 1/10, Wilson 95% CI 1.8–40.4%), the exposure prevalence of 70.6% observed in this cohort, a two-sided α of 0.05, and 80% power. Published trauma-system effect sizes lie between approximately 1.2 and 2.0,^{18,20,54} implying that 706 to 2,247 patients are required, which is attainable through sustained prospective provincial trauma registry recruitment.

5. Conclusion

Among 34 consecutive adult trauma patients at the Emergency Department of Dr. Mohammad Hoesin General Hospital, Palembang, emergency department mortality was 20.6% (7/34; 95% CI: 10.3–36.8%), and none of the three prehospital exposures—prehospital time > 60 minutes (exact conditional OR 2.92; Firth OR 2.23; $p = 0.644$), ambulance transport (exact conditional OR 4.09; Firth OR 4.08; $p = 0.374$), or a medical or paramedical first responder (exact conditional OR 4.09; Firth OR 4.08; $p = 0.374$)—was significantly associated with mortality in bivariate or multivariable analysis (likelihood-ratio $p = 0.492$). With power of 12.6%–23.1% (normal approximation; 4.4%–15.2% by the exact test), minimum detectable ORs of 12.57–

69.82 at 80% power, and reverse fragility indices of 1 to 7, these neutral tests are uninformative rather than negative, and their wide compatibility intervals remain compatible with clinically substantial effects. The descriptive findings are clear: 94.1% (32/34) of patients received initial care from an untrained lay bystander, 94.1% (32/34) arrived by non-ambulance transport, 70.6% (24/34) reached hospital care after the 60-minute golden hour (mean prehospital time 116.47 ± 55.10 minutes), and no patient (0/34) received all three favorable prehospital exposures. Regional priorities should be community lay-responder training in hemorrhage control, airway positioning, and fracture splinting; provincial EMS coordination; and a prospective, biomarker-augmented regional trauma registry enrolling 706 to 2,247 patients to detect ORs of 2.0 to 1.5 with

80% power. By combining exact conditional inference, Firth penalized-likelihood estimation, and formal minimum-detectable-effect and reverse-fragility analysis with an interdisciplinary surgical, orthopedic, and biological framework, this study provides a baseline and a sample-size framework for trauma-system development in South Sumatra.

6. Declarations

Ethics Approval and Consent to Participate

This study was approved by the Health Research Ethics Committee of the Faculty of Medicine, Universitas Sriwijaya / Dr. Mohammad Hoesin General Hospital, Palembang, Indonesia (Reference No.2025/223). The requirement for individual written informed consent was waived for this retrospective analysis of de-identified medical-record data. The study was conducted in accordance with the Declaration of Helsinki.

Consent for Publication

Not applicable; no individually identifiable patient data, images, or clinical details are reported.

Availability of Data and Materials

Aggregated data supporting the conclusions of this article are presented in Tables 1–7 and Figures 1–6. De-identified patient-level data are available from the corresponding author on reasonable request.

Artificial Intelligence Use Statement

None.

Author Contribution Statement

I.K.: Conceptualization, Data curation, Formal analysis, Investigation, Writing – original draft. R.L.: Methodology, Supervision, Writing – review & editing. Z.M.: Methodology, Supervision, Writing – review & editing. All authors reviewed and approved the final manuscript.

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Conflict of Interest / Competing Interests

The authors declare that they have no competing interests.

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