

Original Research Article

Evaluating trauma management approaches and determinants of mortality among hemodynamically unstable patients with severe multiple injuries: a retrospective cohort study

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ABSTRACT

Background: In settings with limited resources, hemodynamically unstable patients with severe multiple injuries continue to have a high mortality rate due to trauma, which is a major preventable cause of death globally. This study evaluated determinants of in-hospital mortality in this population.

Methods: This retrospective cohort study included 1000 hemodynamically unstable patients having an Injury Severity Score (ISS) of 16 or more, admitted to a tertiary care center in Tamil Nadu, India, between January 2024 and January 2026. Injury patterns, physiological parameters, trauma scores, management interventions, transfusion practices and outcomes were retrieved from hospital records. Independent predictors of in-hospital mortality were found using multivariable binary logistic regression, and adjusted odds ratios (AOR) with 95% confidence intervals (CI) have been calculated.

Results: Mean age was 38.4 years and 77.6% were male. Road traffic accidents (63.5%) and blunt trauma (87.4%) predominated. In-hospital mortality was 35.0%, 24-hour mortality 11.4%, and exsanguination accounted for 7.0%. Independent predictors of death were higher ISS (AOR 1.12 per point; 95% CI 1.05 to 1.20), serum lactate above 6 mmol/L (AOR 3.84), Glasgow Coma Scale below 8 (AOR 3.12), delay to definitive haemostasis beyond 120 minutes (AOR 2.96), massive transfusion (AOR 1.58), mechanical ventilation (AOR 1.91) and vasopressor requirement (AOR 2.28). A ratio of 1:1 or higher between fresh frozen plasma and red blood cells was protective (AOR 0.42). Damage control surgery, interventional radiology and computed tomography before haemostasis were not independent predictors.

Conclusions: Injury severity, depth of shock, delayed haemostasis and unbalanced transfusion were the principal determinants of mortality. Early risk stratification, rapid hemorrhage control and balanced transfusion may improve survival.

Keywords: Trauma, Hemorrhagic shock, Multiple trauma, Blood transfusion, Mortality, Injury severity score

INTRODUCTION

Trauma is a major health problem and a primary cause of mortality and disability worldwide. The "World Health Organization (WHO)" evaluates that injuries and violence cause 4.4 million fatalities annually, or almost 8% of all deaths worldwide. Among most common causes of death for those between ages of 5 and 29 are road traffic injuries, homicide, and suicide.¹

Severe multiple injuries are frequently accompanied by hemorrhagic shock and hemodynamic instability, which are major contributors to early trauma-related death. Despite advances in prehospital care, resuscitation, surgical technique and critical care, uncontrolled hemorrhage remains most important preventable cause of death after injury.² Early recognition of life-threatening injuries and prompt hemorrhage control are therefore central to modern trauma care.

Over the past two decades, trauma management has evolved from sequential treatment to coordinated multidisciplinary systems involving emergency physicians, trauma surgeons, anesthesiologists, interventional radiologists and intensivists.³ Contemporary practice emphasizes rapid assessment, damage control resuscitation, permissive hypotension, balanced blood component therapy, and timely hemorrhage control.⁴⁻⁶ Reducing the time to definitive haemostasis and optimizing transfusion practice can improve survival in severe trauma with hemorrhagic shock.^{7,8}

Nevertheless, mortality among haemodynamically unstable trauma patients remains substantial, particularly in those with a high injury burden and profound physiological derangement.⁹ The effectiveness of trauma management may vary between institutions according to resource availability, organizational structure and adherence to protocols. Evidence assessing the effect of trauma management on mortality rates among hemodynamically unstable patients with severe multiple injuries continues to be limited, especially in tertiary care settings, despite research from India describing injury patterns and management outcomes.^{10,11}

The present study was therefore undertaken to evaluate trauma management strategies and their association with clinical outcomes among haemodynamically unstable patients having severe multiple injuries, for identifying independent determinants of mortality, including injury severity, timing of definitive haemostatic intervention, and transfusion practice.

METHODS

Study design and setting

This retrospective cohort research has been carried out in Trauma Ward and Emergency Department of a tertiary care center in Salem, Tamil Nadu, India, and included patients admitted between January 2024 and January 2026. Protocol approved by Institutional Ethics Committee. The retrospective analysis employed anonymised hospital information; thus, informed consent was not needed. Clinical photographs in this article were published with written informed consent from each patient or their legal representative.

Participants

All patients presenting with severe trauma and hemodynamic instability during the study period were screened. Patients with hemodynamic instability at presentation, severe trauma with an ISS of 16 or above, and a need for active resuscitation, such as airway management, blood transfusion, or shock treatment, were all eligible. Patients were excluded if they arrived in

cardiopulmonary arrest, had a non-traumatic cardiac arrest, had an ISS below 16, remained hemodynamically stable throughout the initial assessment, or had records too incomplete to permit calculation of trauma scores or determination of the primary outcome.

Sample size calculation

Based on a related study by Otsuka H et al the sample size of 1000 was determined.¹² With an absolute precision of 3%, estimated mortality rate among hemodynamically unstable individuals is 0.364. The significance level was set at 5%. The formula used for calculation was

$$n \geq \frac{Z_{1-\frac{\alpha}{2}}^2 p(1-p)}{d^2}$$

Where p is expected proportion or prevalence of event of interest for the study (0.364)

n is the required sample size

d is the absolute precision (0.03)

$Z_{1-\frac{\alpha}{2}}$ is normal deviate at a level of significance which is 1.96 at 95 % confidence interval

$$n \geq \frac{(1.96)^2 (0.364)(1 - 0.364)}{(0.03)^2}$$

$$n \geq 989 \approx 1000$$

Data sources and measurement

Data were extracted on a structured proforma designed for this study from hospital case records, the trauma registry, operative notes, transfusion records, ICU charts and discharge summaries. Recorded variables comprised age, sex, mechanism of injury and type of trauma; heart rate, systolic and diastolic blood pressure, "Glasgow Coma Scale (GCS)" score,¹³ serum lactate, arterial base excess, hemoglobin and international normalized ratio at admission; damage control surgery, interventional radiology, combined surgery with interventional radiology, CT before haemostasis, massive transfusion, mechanical ventilation, vasopressor support and ICU admission; and units of RBC, FFP and platelets transfused. Each patient was classified by dominant injury complex as having severe traumatic brain injury with haemodynamic instability, severe chest wall or thoracic injury with haemodynamic instability, or severe polytrauma with shock. Injury severity was quantified using the ISS¹⁴ and the Revised Trauma Score (RTS),¹⁵ and the TRISS predicted probability of survival was derived from ISS, RTS, age and mechanism of injury.¹⁶

Variables and definitions

An ISS of 16 or above, which is frequently utilised for analysing individuals at high risk of morbidity and mortality following injury, was considered severe trauma.¹⁴ Haemodynamic instability was characterized as either persistent hypotension with at least two recorded systolic readings below 90 mmHg despite early resuscitation, or systolic blood pressure below 90 mmHg on arrival.¹⁷ Ten or more units of packed red blood cells within the first twenty-four hours were considered a massive transfusion. Definitive haemostasis was defined as control of hemorrhage achieved by operation or by interventional radiology, and the time to definitive haemostasis was measured from hospital arrival to the start of that procedure.

In-hospital mortality, which is defined as death within same admission, was the main outcome. Secondary outcomes included 24-hour mortality, mortality from exsanguination, survival among patients whose "Trauma and Injury Severity Score (TRISS)" predicted probability of survival has been less than 25%, time from arrival to definitive haemostasis, use of computed tomography (CT) before haemostasis, transfusion requirements, including the ratio of fresh frozen plasma (FFP) to red blood cell (RBC), the need for massive transfusion, and length of stay in the "intensive care unit (ICU)".

Bias

All consecutive eligible patients admitted during study period have been included to reduce selection bias. Information bias was reduced by extracting data onto a single predefined proforma with standardized definitions.

Statistical methods

Data was entered into Excel 2019 and analyzed using SPSS version 29 (IBM Corporation, Armonk, NY, USA). Continuous variables was tested for normality utilising the Shapiro-Wilk test. Regularly distributed continuous data are shown as mean with standard deviation, while non-normally distributed variables are shown as median with interquartile range. Categorical variables was presented using frequencies and percentages. To compare survivors and non-survivors, the independent samples t-test, Mann-Whitney U test, and chi-square test were used. A multivariable binary logistic regression model was developed utilizing significant univariate factors, and the AOR with 95% CI is presented. Patients with incomplete records were excluded, not imputed. Two-sided p values under 0.05 have been statistically significant.

RESULTS

A total of 1000 individuals with severe multiple injuries who were hemodynamically unstable were examined. The mean age was 38.4 (SD 15.1) years and 776 (77.6%) were male. Road traffic accidents were the leading

mechanism (635, 63.5%), followed by falls from height (179, 17.9%), assault (148, 14.8%) and other mechanisms (38, 3.8%). Blunt trauma predominated (874, 87.4%). The mean ISS was 30.1 (SD 8.0), the mean RTS 5.9 (SD 1.2) and the mean TRISS predicted probability of survival 57.6% (SD 21.8). Patients were profoundly shocked at admission, with a mean systolic blood pressure of 75.4 (SD 11.4) mmHg, a mean heart rate of 125.5 (SD 6.2) beats per minute and a mean GCS score of 8 (SD 2). Metabolic derangement was marked, with a mean serum lactate of 5.4 (SD 1.8) mmol/L and a mean base excess of -8.8 (SD 3.9) mEq/L. Vasopressors were required by 429 (42.9%) patients (Table 1).

Table 1: Baseline characteristics of the study population (n=1000).

Variables	Frequency (%) /Mean±SD
Age (years)	38.4±15.1
Gender	
Male	776 (77.6)
Female	224 (22.4)
Mechanism of injury	
Road traffic accident	635 (63.5)
Fall from height	179 (17.9)
Assault	148 (14.8)
Other mechanisms	38 (3.8)
Trauma type	
Blunt trauma	874 (87.4)
Penetrating trauma	126 (12.6)
Injury severity score	30.1±8.0
Revised trauma score	5.9±1.2
TRISS probability of survival(%)	57.6±21.8
Systolic blood pressure (mmHg)	75.4±11.4
Diastolic blood pressure(mmHg)	48.8±8.3
Heart rate (beats/min)	125.5±6.2
Glasgow coma scale score	8±2
Serum lactate (mmol/l)	5.4±1.8
Base excess (mEq/l)	-8.8±3.9
Hemoglobin (g/dl)	7.3±2.1
International normalized ratio	1.54±0.47
Patients requiring vasopressors	429 (42.9)

Damage control surgery was the most frequent intervention (536, 53.6%). Interventional radiology was used in 188 (18.8%) patients and combined surgery with interventional radiology in 106 (10.6%). CT before definitive haemostasis was performed in 347 (34.7%) patients, massive transfusion was required in 388 (38.8%), mechanical ventilation in 641 (64.1%) and ICU admission in 821 (82.1%). The median number of RBC units transfused before haemostasis was 5 (IQR 3 to 7) and of FFP units 4 (IQR 3 to 6), and the mean FFP to RBC ratio was 0.84 (SD 0.23) (Table 2).

Table 2: Trauma management, transfusion practices, and clinical outcomes (n=1000)

Intervention	N (%) or Median (IQR) or Mean±SD
Damage control surgery	536 (53.6)
Interventional radiology	188 (18.8)
Combined surgery and interventional radiology	106 (10.6)
Computed tomography before haemostasis	347 (34.7)
Massive transfusion	388 (38.8)
Mechanical ventilation	641 (64.1)
Intensive care unit admission	821 (82.1)
Red blood cell units before haemostasis	5 (3–7)
Fresh frozen plasma units before haemostasis	4 (3–6)
Platelet units before haemostasis	1 (0–2)
Red blood cell units in first 24 hours	9 (7–11)
Fresh frozen plasma units in first 24 hours	6 (4–8)
Fresh frozen plasma to red blood cell ratio, mean (SD)	0.84 ± 0.23
In-hospital mortality	350 (35)
24-hour mortality	114 (11.4)
Mortality due to exsanguination	70 (7)
Survivors with TRISS predicted survival below 25%	7 (0.7)
Intensive care unit length of stay (days)	9.1 ± 5.4
Hospital length of stay (days)	15.0 ± 7.9

In-hospital death occurred in 350 (35.0%) patients and 650 (65.0%) survived. Death within 24 hours occurred in 114 (11.4%) patients, and exsanguination accounted for 70 (7.0%). Seven patients survived despite a TRISS predicted probability of survival below 25%. The mean ICU stay has been 9.1 (SD 5.4) days and mean hospital stay 15.0 (SD 7.9) days. Among the 350 patients who died, severe traumatic brain injury with hemodynamic instability was the commonest injury pattern (157, 45.0%), followed by severe polytrauma with shock (123, 35.0%) and severe chest wall or thoracic injury with hemodynamic instability (70, 20.0%) (Table 2). Representative craniofacial injuries encountered in this cohort are shown in Figure 1.

On univariate comparison, non-survivors more often underwent damage control surgery, interventional radiology, combined surgery with interventional radiology and CT before haemostasis, and more often required massive transfusion, mechanical ventilation, ICU admission and vasopressor support (all

$p<0.001$). Non-survivors were older than survivors (45.2 versus 36.5 years; $p=0.018$) and had a greater injury burden (ISS 37.6 versus 26.8; $p<0.001$), lower RTS (4.3 versus 6.2; $p<0.001$), as well as lower GCS score (7.5 versus 11.1; $p<0.001$). Serum lactate was higher among non-survivors (7.8 versus 4.6 mmol/l; $p<0.001$). Non-survivors also reached definitive haemostasis considerably later (142 versus 78 minutes; $p<0.001$) and received a less balanced FFP to RBC ratio (0.61 versus 0.91; $p=0.002$) (Table 3).

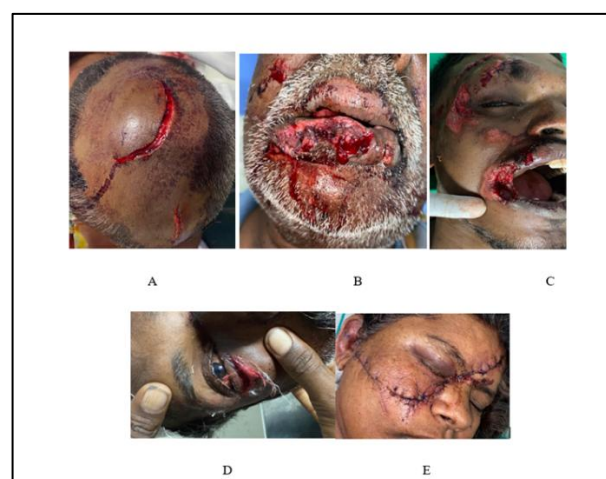


Figure 1: Representative craniofacial injury patterns among haemodynamically unstable patients with severe multiple injuries. (A) Deep scalp laceration sustained in a road traffic accident, a recognized source of significant external hemorrhage; (B) extensive perioral soft tissue laceration with active bleeding; (C) avulsion injury of the lower lip and oral commissure with associated dentoalveolar injury; (D) full thickness periorbital and eyelid laceration and (E) frontal and periorbital lacerations after primary surgical repair.

All images were cropped to the region of clinical interest to remove identifying features. Written informed consent for publication was obtained from each patient or their legally authorized representative.

On multivariable logistic regression, each one-point rise in ISS independently increased the odds of in-hospital death (AOR 1.12; 95% CI 1.05 to 1.20; $p<0.001$). Serum lactate above 6 mmol/L (AOR 3.84; $p=0.004$), time to haemostasis beyond 120 minutes (AOR 2.96; $p=0.017$), GCS score below 8 (AOR 3.12; $p=0.014$), massive transfusion (AOR 1.58; $p=0.041$), mechanical ventilation (AOR 1.91; $p=0.009$) and vasopressor requirement (AOR 2.28; $p=0.001$) have also been independent predictors of death, whereas an FFP to RBC ratio of 1:1 or higher was independently protective (AOR 0.42; 95% CI 0.18 to 0.96; $p=0.039$). Damage control surgery, interventional radiology, combined surgery with interventional radiology and CT before haemostasis were not independently associated with mortality after adjustment (all $p>0.05$) (Table 4).

Table 3: Comparison of survivors and non-survivors.

Variables	Survivors (n=650), N (%)	Non-survivors (n=350), N (%)	P value
Damage control surgery	249 (38.3)	287 (82.0)	<0.001
Interventional radiology	15 (2.3)	173 (49.4)	<0.001
Combined surgery and interventional radiology	10 (1.5)	96 (27.4)	<0.001
Computed tomography before haemostasis	90 (13.8)	257 (73.4)	<0.001
Massive transfusion	125 (19.2)	263 (75.1)	<0.001
Mechanical ventilation	338 (52.0)	303 (86.6)	<0.001
Intensive care unit admission	490 (75.4)	331 (94.6)	<0.001
Vasopressor support	164 (25.2)	265 (75.7)	<0.001
Age (yrs)	36.5 (14.1)	45.2 (16.4)	0.018*
Injury severity score	26.8 (7.2)	37.6 (8.5)	<0.001*
Revised trauma score	6.2 (1.0)	4.3 (1.2)	<0.001*
Glasgow coma scale score	11.1 (3.1)	7.5 (2.8)	<0.001*
Serum lactate (mmol/l)	4.6 (1.7)	7.8 (2.3)	<0.001*
Time to definitive haemostasis (min)	78 (34)	142 (52)	<0.001*
Fresh frozen plasma to red blood cell ratio	0.91 (0.18)	0.61 (0.21)	0.002*

*Statistically significant at the 5% level. SD: standard deviation.

Table 4: Multiple logistic regression analysis for predictors of in-hospital mortality.

Variables	Adjusted OR	95% CI	P value
Injury severity score (per point)	1.12	1.05–1.20	<0.001*
Serum lactate above 6 mmol/l	3.84	1.52–9.70	0.004*
Time to haemostasis beyond 120 min	2.96	1.21–7.25	0.017*
Glasgow Coma Scale score below 8	3.12	1.26–7.71	0.014*
Fresh frozen plasma to red blood cell ratio 1:1 or higher	0.42	0.18–0.96	0.039*
Damage control surgery	0.78	0.49–1.25	0.304
Interventional radiology	0.84	0.46–1.54	0.571
Combined surgery and interventional radiology	0.69	0.31–1.53	0.362
Massive transfusion	1.58	1.02–2.46	0.041*
Mechanical ventilation	1.91	1.18–3.10	0.009*
Vasopressor requirement	2.28	1.42–3.65	0.001*
Computed tomography before haemostasis	1.44	0.90–2.30	0.126

*Statistically significant at 5% level of significance, CI: confidence interval.

DISCUSSION

This study estimated trauma management strategies and their association with clinical outcomes among 1000 hemodynamically unstable patients having several severe injuries. In-hospital mortality has been 35%, with 24-hour mortality of 11.4% and exsanguination accounting for 7.0% of the study population. On multivariable analysis, injury severity (ISS, AOR 1.12 per point), serum lactate >6 mmol/l (AOR 3.84), GCS <8 (AOR 3.12) and time to definitive haemostasis >120 minutes (AOR 2.96) independently predicted death, whereas an FFP:RBC ratio ≥1:1 was protective (AOR 0.42). Massive transfusion, mechanical ventilation and vasopressor requirement also retained significance, while damage control surgery, interventional radiology and pre-

haemostasis CT did not. Survival in this population therefore depends less on which intervention is selected than on how rapidly hemorrhage is controlled and how completely the accompanying coagulopathy is corrected.

Our in-hospital mortality was slightly lower than the 36.4% reported by Otsuka H et al. after implementation of a streamlined trauma management protocol as well as substantially lower than the 64.4% observed before protocol implementation.¹² The similar predictors of mortality identified in both studies suggest that these factors remain consistent across different healthcare settings. Although 24-hour mortality accounted for only one-third of all in-hospital deaths, it indicates that a substantial proportion of fatalities occurred early after admission, emphasizing the importance of rapid

haemorrhage control during the initial hours of trauma care. Consistent with the observations of Sauaia A et al, exsanguination remained the leading potentially preventable cause of death, indicating that early haemorrhagic mortality continues to be a major challenge despite significant advances in trauma resuscitation over the past three decades.² Notably, seven patients survived despite having a TRISS-predicted probability of survival below 25%, suggesting that timely, aggressive, protocol-based management can improve outcomes even in patients with a very poor predicted prognosis. This finding also indicates that TRISS should be used as a prognostic tool to support clinical decision-making rather than as the sole basis for limiting intensive treatment.

Among the determinants examined, injury severity exerted the strongest anatomical effect, with each one-point increase in ISS raising the adjusted odds of in-hospital death by 12% in our study. This finding is accordance with Wong TH et al., who reported a 12–14% increase in odds of mortality with each unit increase in ISS across different prediction models, and with Otsuka H et al., who also identified higher ISS as an independent predictor of poorer survival among hemodynamically unstable patients with severe trauma.^{18,12} Thus these findings emphasize that anatomical injury severity remains a major determinant of mortality, although timely and protocol-based trauma management can substantially improve survival outcomes. The same is largely true of neurological injury: severe traumatic brain injury with hemodynamic instability was the commonest pattern among non-survivors (45%), reflecting the established synergy by which a single hypotensive episode approximately doubles mortality after severe head injury.¹⁹ Most patients in our study sustained blunt trauma, many of whom may also have traumatic brain injury. Therefore, permissive hypotension should be used cautiously, as maintaining adequate cerebral perfusion may be more important than lowering blood pressure to reduce bleeding. In contrast, serum lactate reflects the severity of tissue hypoxia and shock before hospital arrival, even when blood pressure appears relatively preserved because of the body's compensatory mechanisms. Elevated lactate has been related to a four-fold increase in the odds of death in our study, confirming earlier findings that admission lactate is an accurate predictor of mortality in patients with severe trauma.²⁰

The time taken to achieve definitive haemostasis was one of the most important modifiable factors associated with survival. In our study, non-survivors reached haemostasis almost one hour later than survivors (142 vs. 78 minutes), and a delay of more than 120 minutes has been related to a three-fold increase in odds of death. Similar investigations have been reported by Otsuka H et al, who reduced the median time to surgery or interventional radiology from 71.5 to 41.0 minutes after implementing a streamlined trauma management protocol, resulting in a significant reduction in in-hospital mortality (64.4% to 36.4%).¹² Although our patients experienced longer

delays, this may reflect differences in healthcare resources, including limited round-the-clock interventional radiology services, the absence of hybrid emergency rooms, and delays related to specialist availability, operating theatre access, and blood product preparation. Transfusion practices also influenced outcomes. Survivors received a more balanced FFP:RBC ratio than non-survivors (0.91 vs. 0.61), which is consistent with the PROPPR trial, where a 1:1:1 transfusion strategy reduced deaths due to exsanguination (9.2% vs. 14.6%) and improved achievement of haemostasis, although it did not reduce overall mortality.²¹ The stronger association observed in our study may be because patients who died very early had less time to receive plasma transfusions, resulting in lower FFP:RBC ratios.

Although damage control surgery, interventional radiology, and CT before haemostasis have been associated with higher mortality on univariate analysis, these associations didn't remain significant after multivariable adjustment. This is likely because these interventions were mainly performed in patients with the most severe injuries, who were already at a higher risk of death. Therefore, the higher mortality reflects the severity of the patients' condition rather than the harmful effects of the interventions themselves. Similar findings have been reported for whole-body CT, where improved outcomes have been observed when imaging is integrated into an efficient trauma pathway, but delays in definitive haemorrhage control can worsen outcomes. Thus, the timing of CT within the trauma management pathway is more important than the imaging procedure itself.²²

Limitations include the retrospective, single-centre design with its inherent documentation bias and residual confounding despite adjustment. Prehospital time and transfer status were unrecorded, though both influence shock depth and achievable haemostasis time. Causal inference is precluded, particularly for the FFP:RBC ratio. REBOA and hybrid theatre capability were unavailable, and outcomes were confined to the index admission. Findings are therefore most generalisable to tertiary centres in low- and middle-income settings managing blunt, road-traffic-dominant polytrauma without on-site endovascular capability.

CONCLUSION

The severity of injury, the depth of shock, delayed definitive haemostasis, and an uneven transfusion ratio were the main factors influencing mortality in hemodynamically unstable patients having severe multiple injuries. Because injury severity as well as neurological injury are fixed at presentation, the greatest opportunities to improve survival lie in compressing the interval to hemorrhage control and in delivering balanced blood component therapy. This study advances understanding by quantifying these determinants in a large South Indian cohort and by showing that apparent

harm from damage control surgery, interventional radiology and early CT reflects confounding by indication rather than a treatment effect. Prospective multicenter studies incorporating prehospital timelines and functional outcomes are warranted.

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