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### Fibrinolysis of ST-elevation myocardial infarction in the emergency department: prognosis of elderly patients treated with streptokinase

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#### **Abstract**

The use of fibrinolytic therapy for ST-segment elevation myocardial infarction can lead to fatal complications. Streptokinase, a non-specific fibrinolytic, remains used in developing countries. The safety of this fibrinolytic agent is under study in the elderly population.

Objectives: This study aimed to compare the prognosis of elderly patients (> 75 years old) treated with Streptokinase for ST-segment elevation myocardial infarction versus 75 years old concerning hemorrhagic complications and mortality.

**Methods**: Prospective study (2009-2018). Inclusion of patients treated with Streptokinase for STEMI. Comparison of two groups: elderly patients and patients less than 75 years old.

**Results:** Inclusive of 624 patients. Mean age=59±11 years. Sex-ratio=5.

Patients aged over 75 represented 10% of the study sample. The comparative analysis of the elderly group (n=63) and patients less than 75 years (n=561) found that elderly patients were more frequently male Sex-ratio = 1 vs 7, p<0,001) and had more hypertension (59%) vs (25%) (p<0,001). Atypical clinical presentation was more frequent in the elderly group (30% vs 16%). No difference was found in the two groups concerning fibrinolysis success (57% vs 61%), and intracranial hemorrhage (0 vs 2 patients). In-hospital and one-week mortality were higher in the elderly group (4.8% and 8% vs 0.7% and 1.2%, respectively). Mortality predictors among elderly patients were anterior myocardial infarction (OR=6.37, p=0.044, 95%IC 1.19-34) and cardiogenic shock (OR=38, p<0.001, 95%IC 5.02-287.43). Streptokinase is an effective therapeutic alternative for the elderly with ST-segment elevation myocardial infarction. It causes reperfusion in 57% of cases with no significant increase in major hemorrhagic events.

**Keywords:** Fibrinolysis, STEMI, Elderly, Complications, Outcomes, Emergency

#### Introduction

Older age is an independent prognostic factor of mortality for patients with ST-segment elevation myocardial infarction (STEMI) (1). Elderly adults (aged >75 years) account for up to 25% of the STEMI community population, with mortality being four times as high as that of their younger (2,3).counterparts Primary percutaneous coronary intervention (PCI) is the treatment of choice for those patients. Fibrinolysis is a valuable alternative when mechanical reperfusion is not timely available. However, the value of these therapies in old patients is not well established because elderly patients have been either excluded or rarely enrolled in reperfusion clinical trials (4). The main limit to the use of this strategy was the bleeding risk. In the STREAM study (5), fibrinolysis with a full dose of Tenecteplase, a specific fibrinolytic, was associated with an increased risk of intracranial bleeding in aged patients.

The Streptokinase, a non-specific fibrinolytic agent, is still recommended but not well evaluated in real life on the elderly.

This study aimed to compare the prognosis of elderly patients (aged more than 75 years old) treated with Streptokinase for STEMI versus patients aged less than 75 years old concerning hemorrhagic complications and mortality.

#### Methods

We conducted a prospective observational study over 10 years from January 2009 to December 2018.

Inclusion criteria (all of these criteria): (1)Patients who were admitted to the emergency department for STEMI and treated with Streptokinase as a

reperfusion strategy, (2) STEMI was identified by the onset of typical chest pain lasting more than 20 minutes, unrelieved by nitrate, and associated with typical ST-segment elevation on at least two contiguous leads of a standard 12-lead ECG or a new onset of left bundle branch.

Exclusion criteria: Patients with non-persistent ST-segment elevation, and/or a diagnosis other than STEMI after investigations.

Study protocol: Patients included in the study were divided into two groups: (1) Patients > 75 years old: received 250 mg of salicylate intravenously (IV), 75 mg of Clopidogrel orally, and subcutaneous (SC) dose of 0.75mg/kg of enoxaparin; (2) Patients ≤ 75 years old: received 250 mg of salicylate intravenously, 300 mg of Clopidogrel orally and enoxaparin (30mg IV bolus followed 15 minutes later by 1mg/kg SC).

All patients received 1.5 MU of Streptokinase over 60 minutes IV. An ECG was used to assess the success of thrombolysis at 90 minutes. Angiography was performed as soon as possible. Hemodynamic and hemorrhagic complications were assessed during the first 24 hours.

Statistical analysis: Data are expressed numbers and rates. Descriptive statistics were used illustrate subjects' to demographic characteristics. Comparison of characteristics of the two groups was performed by the  $\chi$ 2 test or Fischer's exact test as appropriate. Continuous variables were expressed as means ± standard deviation, and the subgroups were compared by Student's t-test. Risk factors were evaluated in univariate analysis for the compared data. A pvalue of less than 0.05 was considered statistically significant.

#### **Results**

We included 624 patients with a mean age of 59±11 years and a sex ratio of 5. Sixty-three patients were aged more than 75 years old. The comparative study of the two groups is detailed in Tables 1 and 2.

Table 1: Comparative analysis of demographic and cardiovascular risk factors.

	All N=624	>75 years N =63	≤75 years N =561	P
Age mean ±SD (years)	59±11	79±4	56±9	< 0.001
Male gender n (%)	518(83)	32(51)	488(87)	< 0.001
Current Smoking n (%)	458(74)	24 (38)	434 (77)	<0,001
Hypertension n (%)	180 (29)	37 (59)	143 (25)	<0,001
Diabetes n (%)	173(28)	15 (24)	158 (28)	NS
Known coronary artery disease (%)	45(7)	7 (11)	45(7)	NS
Ischemic stroke n	4	1	3	NS

Table 2: Comparative study of clinical and biological parameters

	All	>75	≤ 75	P-
	patients	years	years	value
	N=624	N=63	N=561	
Mean delay chest pain-	183±144	251±169	175±139	< 0.001
FMC (min) mean±SD				
Atypical presentation	209 (23)	19 (30)	190 (16)	0.006
n (%)				
Heart rate (bpm)	79±21	82±24	79±20	NS
mean±SD				
Systolic blood pressure	$139 \pm 33$	$136 \pm 40$	$139\pm32$	NS
(mmHg) mean±SD				
SpO2 (%)) mean±SD	96±2	94±6	96±2	NS
Killip ≥2 n (%)	56 (13)	12(19)	42(7.5)	0.002
Creatinine (µmol/l)	83±28	101±6	82±20	< 0.001
mean±SD				
Hemoglobin (g/dl)	$14.6 \pm 1.7$	13.7±2	$14.7\pm2$	0.002
mean±SD				

The mean delay between first medical contact and fibrinolysis was 29±20 minutes in the aged group versus 25±19 min (p=0.12).

The fibrinolysis failure rate was similar in the two groups (57% versus 62%; p=0.32). The angiography was performed at the same delay in the two groups. Table 3 details the comparative analysis of complications in the two groups of patients. Univariate analysis identified two factors linked to mortality in elderly patients: an extended anterior wall and cardiogenic shock (Table 4)

Table 3: Comparative study of complications and mortality in the two groups

Hemodynamic complications:           Cardiac arrest n(%)         39 (9)         8(12)         31(5,6)         0,027           Asystole n (%)         6 (3)         3(5)         3 (0,5)         0,016           Ventricular fibrillation n         33         5(8)         28(5)         NS           (%)         (6.5)         (6.5)         (7)         0,06           Cardiogenic shock n (%)         30         6(10)         24(4,3)         0,06           (12)         Acute heart failure n (%)         80(18)         16(25)         64(11)         0,004           High grade AVB n (%)         31(4)         2 (3,1)         29(5)         NS           Hypotension n (%)         109(1)         13(20)         96(17)         NS           Sinus bradycardia n (%)         78         10(16)         68(12)         NS           Hemorrhagic complications:           Cerebral bleeding n         2         0         2         NS           Gingival hemorrhage n (%)         37(2)         4         33(6)         NS           Hematemesis n         11         0         11         NS           Mortality         First day n         7         3         4         0.0004 </th <th></th> <th>All patien ts N=62</th> <th>&gt;75 years N=63</th> <th>≤75 years N=561</th> <th>P-value</th>		All patien ts N=62	>75 years N=63	≤75 years N=561	P-value
Cardiac arrest n(%) 39 (9) 8(12) 31(5,6) 0,027  Asystole n (%) 6 (3) 3(5) 3 (0,5) 0,016  Ventricular fibrillation n 33 5(8) 28(5) NS  (%) (6.5)  Cardiogenic shock n (%) 30 6(10) 24(4,3) 0,06  (12)  Acute heart failure n (%) 80(18) 16(25) 64(11) 0,004  High grade AVB n (%) 31(4) 2 (3,1) 29(5) NS  Hypotension n (%) 109(1 13(20) 96(17) NS  Sinus bradycardia n (%) 78 10(16) 68(12) NS  Hemorrhagic complications:  Cerebral bleeding n 2 0 2 NS  Gingival hemorrhage n (%) 37(2) 4 33(6) NS  Hematemesis n 11 0 11 NS  Mortality  First day n 7 3 4 0.004					
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(%)       (6.5)         Cardiogenic shock n (%)       30       6(10)       24(4,3)       0,06         Acute heart failure n (%)       80(18)       16(25)       64(11)       0,004         High grade AVB n (%)       31(4)       2 (3,1)       29(5)       NS         Hypotension n (%)       109(1)       13(20)       96(17)       NS         Sinus bradycardia n (%)       78       10(16)       68(12)       NS         Hemorrhagic complications:         Cerebral bleeding n       2       0       2       NS         Gingival hemorrhage n (%)       37(2)       4       33(6)       NS         Hematemesis n       11       0       11       NS         Mortality       First day n       7       3       4       0.004	Asystole n (%)	6 (3)	3(5)	3 (0,5)	0,016
Cardiogenic shock n (%) 30 6(10) 24(4,3) 0,06 (12)  Acute heart failure n (%) 80(18) 16(25) 64(11) 0,004  High grade AVB n (%) 31(4) 2 (3,1) 29(5) NS  Hypotension n (%) 109(1 13(20) 96(17) NS  Sinus bradycardia n (%) 78 10(16) 68(12) NS  Hemorrhagic complications: Cerebral bleeding n 2 0 2 NS  Gingival hemorrhage n (%) 37(2) 4 33(6) NS  Hematemesis n 11 0 11 NS  Mortality  First day n 7 3 4 0.004	Ventricular fibrillation n	33	5(8)	28(5)	NS
Acute heart failure n (%) 80(18) 16(25) 64(11) 0,004 High grade AVB n (%) 31(4) 2 (3,1) 29(5) NS Hypotension n (%) 109(1 13(20) 96(17) NS Sinus bradycardia n (%) 78 10(16) 68(12) NS Hemorrhagic complications: Cerebral bleeding n 2 0 2 NS Gingival hemorrhage n (%) 37(2) 4 33(6) NS Hematemesis n 11 0 11 NS Mortality First day n 7 3 4 0.004	(%)	(6.5)			
Acute heart failure n (%) 80(18) 16(25) 64(11) 0,004 High grade AVB n (%) 31(4) 2 (3,1) 29(5) NS Hypotension n (%) 109(1 13(20) 96(17) NS  Sinus bradycardia n (%) 78 10(16) 68(12) NS  Hemorrhagic complications: Cerebral bleeding n 2 0 2 NS Gingival hemorrhage n (%) 37(2) 4 33(6) NS Hematemesis n 11 0 11 NS  Mortality First day n 7 3 4 0.004	Cardiogenic shock n (%)	30	6(10)	24(4,3)	0,06
High grade AVB n (%) 31(4) 2 (3,1) 29(5) NS Hypotension n (%) 109(1 13(20) 96(17) NS  8) Sinus bradycardia n (%) 78 10(16) 68(12) NS  (14)  Hemorrhagic complications: Cerebral bleeding n 2 0 2 NS Gingival hemorrhage n (%) 37(2) 4 33(6) NS Hematemesis n 11 0 11 NS  Mortality First day n 7 3 4 0.004		(12)			
Hypotension n (%) 109(1 13(20) 96(17) NS 8)  Sinus bradycardia n (%) 78 10(16) 68(12) NS (14)  Hemorrhagic complications: Cerebral bleeding n 2 0 2 NS Gingival hemorrhage n (%) 37(2) 4 33(6) NS Hematemesis n 11 0 11 NS Mortality First day n 7 3 4 0.004	Acute heart failure n (%)	80(18)	16(25)	64(11)	0,004
Sinus bradycardia n (%)	High grade AVB n (%)	31(4)	2(3,1)	29(5)	NS
Sinus bradycardia n (%)       78 (14)       10(16)       68(12)       NS         Hemorrhagic complications:         Cerebral bleeding n       2       0       2       NS         Gingival hemorrhage n (%)       37(2)       4       33(6)       NS         Hematemesis n       11       0       11       NS         Mortality         First day n       7       3       4       0.004	Hypotension n (%)	109(1	13(20)	96(17)	NS
(14)         Hemorrhagic complications:         Cerebral bleeding n       2       0       2       NS         Gingival hemorrhage n (%)       37(2)       4       33(6)       NS         Hematemesis n       11       0       11       NS         Mortality         First day n       7       3       4       0.004		8)			
Hemorrhagic complications:           Cerebral bleeding n         2         0         2         NS           Gingival hemorrhage n (%)         37(2)         4         33(6)         NS           Hematemesis n         11         0         11         NS           Mortality           First day n         7         3         4         0.004	Sinus bradycardia n (%)	78	10(16)	68(12)	NS
complications:           Cerebral bleeding n         2         0         2         NS           Gingival hemorrhage n (%)         37(2)         4         33(6)         NS           Hematemesis n         11         0         11         NS           Mortality         First day n         7         3         4         0.004		(14)			
Cerebral bleeding n         2         0         2         NS           Gingival hemorrhage n (%)         37(2)         4         33(6)         NS           Hematemesis n         11         0         11         NS           Mortality           First day n         7         3         4         0.004					
Gingival hemorrhage n (%)       37(2)       4       33(6)       NS         Hematemesis n       11       0       11       NS         Mortality         First day n       7       3       4       0.004					
Hematemesis n         11         0         11         NS           Mortality         7         3         4         0.004	Cerebral bleeding n	2	0	2	NS
Mortality         7         3         4         0.004	Gingival hemorrhage n (%)	37(2)	4	33(6)	NS
First day n 7 3 4 0.004	Hematemesis n	11	0	11	NS
	Mortality				
	First day n	7	3	4	0.004
	First week n	12	5	7	< 0.001

Table 4: Factors associated with early mortality in elderly patients

	Death N=5	Survivor N=58	P	OR (CI 95%)
Extended anterior wall n (%)	3(60)	9(15)	0,044	6,37 (1,19-34)
Cardiogenic sh1ck n (%)	4(80)	2(3,4)	<0,001	38 (5,02- 287,43)

#### **Discussion**

treated with a full dose of Streptokinase fibrinolysis had the same reperfusion success rate as younger patients without increasing bleeding risk. However, the mortality rate remains higher. This study is a prospective cohort evaluating the management of elderly patients with STEMI in 'real life'. However, it had several limitations: the relatively small sample of elderly group patients and the difficulty in collecting outcomes after the 7<sup>th</sup> day of management.

The study shows that older adults with STEMI

In this study, we used a non-specific fibrinolytic agent. The use of fibrinolysis for the treatment of STEMI in the elderly has been controversial. Thiemann et al (6) concluded after observational study of 7864 patients treated for acute myocardial infarction in the USA that the use of fibrinolytic therapy in patients over 75 may do more harm than good. In the same study, 30day mortality in patients aged 76-86 was significantly higher in those who received fibrinolytic therapy than in those who did not (18.0% vs 13.6%; p=0.003). These results appear to contradict the Fibrinolytic Therapy Trialists' (FTT) overview of 58,600 patients randomized into thrombolytic trials, which concluded that the 35-day mortality rate in the 5788 patients aged over 75 was lower in those who received thrombolytic therapy than in those who did not (24.3% vs 25.3%). The difference was not significant. However, the absolute mortality reduction in patients over 75 was like that of patients under 55 (7). On the other hand, the benefits of fibrinolysis in STEMI patients over 75 years have been proven in many different studies.

The analysis of 3897 elderly patients from the 'Swedish register of information and knowledge about Swedish heart intensive care admission' found that fibrinolytic therapy was associated with a 13% adjusted relative reduction in the composite of mortality and cerebral bleeding complications after 1 year (95% confidence interval, 0.80-0.94; P = .001) (8).

In our series, the elderly patients' fibrinolysis success rate with Streptokinase was like younger patients (57% versus 61.3%) and higher than most previous published studies. The success rate was 43%, 44% and 53% in Chesebro et al (9), Stack et al (10), and Hogg et al (11) studies, respectively. A recent study found a successful fibrinolysis with Streptokinase in 70% of patients. This finding can be explained using modern adjunctive therapy containing dual antiplatelet therapy and low-weight molecular heparin. The benefits of the dual antiplatelet therapy are explained by the fact that Clopidogrel is a potent antiplatelet agent with a synergistic antithrombotic effect with aspirin because platelet activation can still occur through thromboxane A2-independent pathways despite the inhibition of cyclooxygenase by aspirin, leading to the aggregation of platelets and the formation of thrombin (12).

However, the use of those molecules in the elderly population can have limits because of the higher vulnerability related to distinct pharmacokinetic and pharmacodynamic responses of those patients (13). The adaptation of doses, regarding age, weight, and potential renal impairment, should be considered and can prevent side effects.

The most serious side effects of the use of fibrinolytic agents are bleeding, especially

intracerebral bleeding. This complication increases with age (14). The incidence of this complication remains rare, even in elderly populations, and varies from 0.5% in controlled studies and meta-analysis to 3% in prospective analysis (15,16). That can be explained by the exclusion of the elderly from clinical trials in acute myocardial infarction (17).

The use of specific fibrinolytics such as Tenecteplase can reduce the incidence of bleeding complications. In the ASSENT-2 trial, the incidence of this complication was 1.1% with Tenecteplase versus 3% with Streptokinase (18). However, in a most recent study, the use of plasminogen activators in elderly patients versus Streptokinase was an independent predictive factor of intracranial hemorrhage (19). It is feasible that, because increased fibrin specificity is more effective in producing clot lysis, it also increases the risk of intracranial hemorrhage (18). In our study, we didn't observe any cerebral bleeding in elderly patients, which can be explained by the small sample size, the strict use of a written protocol, and the extension of the contraindication of fibrinolysis to all patients with a history of stroke, independent of his delay.

hemodynamic and were dominated by acute heart failure. In our study, acute heart failure occurred in 25% of elderly STEMI patients versus 11% of younger ones. In the literature, the incidence of this complication ranges from 18% to 37 % (20). Cardiogenic shock is the most serious complication because of the high-risk mortality and the decrease in efficacy of fibrinolytic agents. In the SHOCK trial, fibrinolysis success was

The other, more important, complications were

observed in 15% of patients (21). It's explained by the fact that fibrinolytic agents depend on coronary perfusion pressure to achieve patency (22).

Mortality in STEMI patients increases exponentially with age. In the Gusto I trial, it varies from 3% of patients aged less than 65 years to 30% of patients aged more than 85% (4).

The meta-analyses fibrinolytic therapy trialists found that the mortality increases with age and is independent of the reperfusion strategy. In this study, 24.3% of patients aged more than 75 years treated by fibrinolytic agent died in the first month, versus 25,3% in the control group.

The predictive factors of mortality in our study were an extended anterior wall and cardiogenic shock.

The cardiogenic shock was the first cause of mortality found in the literature. The ventricular rupture is more frequent in aged patients, and it's due to anatomical and physiological modifications of the myocardium by age.

The mortality rate in our population was lower than in the literature; it can be explained by the rigorous application of a written protocol.

#### Conclusion

Full-dose Streptokinase is an effective therapeutic alternative for the elderly. It causes reperfusion in 57% of cases with no significant increase in major hemorrhagic events.

In contrast, mortality in this population is higher than in patients under 75 years old patients and it's related to the extent of myocardial infarction and the development of cardiogenic shock.

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# Thrombolysis failure with Streptokinase in ST-elevation myocardial infarction

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#### **Abstract**

In patients with acute ST elevation myocardial infarction (STEMI), coronary reperfusion with primary percutaneous coronary intervention (PCI) or fibrinolysis improve the prognosis. The main reason to prefer PCI, if it can be performed timely within 120 minutes, is his high incidence of reperfusion compared to thrombolysis. About 25-50% of patients treated with streptokinase fail to achieve successful reperfusion and these patients have poor prognosis.

The aim of this study was to determine independent factors related to thrombolysis failure with Streptokinase in STEMI patients using clinical and electrocardiogram criteria.

**Methods:** A prospective observational study conducted over 9 years including patients treated with Streptokinase for STEMI. Thrombolysis failure was defined using electrocardiogram criteria. Multivariate analysis was used to identify factors related to thrombolysis failure.

**Results:** From a total of 510 patients included, 198 (37%) failed fibrinolysis. The mean age of those patients was 60±11 years, 83% of the population were male. The average delay chest pain to first medical contact (FMC) was 224±170 minutes. Door to needle time was less than 30 minutes. The failure was associated with four variables including Female gender (aOR: 2.05, 95% CI 1.27-3.30; P<0.001), the delay from chest pain to FMC >180 min (aOR:2.413, 95% CI 1.67-3.47; p<0.001), Extended anterior infarct (OR: 2.01, 95% CI 1.05-3.84; p=0.033) and cardiogenic Shock (OR: 7.12, 95%CI 1.66-30.48; p=0.008).

**Conclusion:** Streptokinase had a failure rate of 37%. Female gender, a longer delay from pain to first medical contact, extended anterior infarction, and cardiogenic shock were highly predictive of thrombolysis failure.

Keywords: Streptokinase, Thrombolysis, Failure, STEMI, Predictors

#### Introduction

Primary percutaneous coronary intervention (PCI) is the recommended reperfusion strategy in patients presenting with a ST-segment elevation myocardial infarction (STEMI) (1). Intravenous thrombolysis is the most widely used reperfusion strategy if timely primary PCI cannot be performed. Streptokinase, a non-specific fibrinolytic still recommended. This strategy prevents approximately 20–30 deaths per 1000 patients treated, with a proportional reduction in mortality of up to 25% in certain subgroups (2). However, it's not always successful. In the angiographic sub-study of the GUSTO-I trial, the 90-minute arterial patency rate (defined by the presence of TIMI grade 2 or 3 flow) was 54% in patients who received Streptokinase, with only 29% of patients having TIMI grade 3 flow (3). The most well-known factor of thrombolysis failure was the delay from the pain onset. De Balder demonstrated in 2001 that the direct effect on tissue perfusion depends on mechanical, rheological, metabolic. and hematological factors(4).

This study aimed to identify predictive factors of thrombolysis failure in patients treated with Streptokinase for STEMI.

#### Methods

#### Study design and setting:

We conducted a prospective observational study for 9 years (January 2009 to December 2016).

#### Study population:

**Inclusion criteria:** Patients who were admitted to the emergency department for STEMI and treated with Streptokinase as a reperfusion strategy.

Exclusion criteria: Presence of contraindications to streptokinase therapy, patients presenting with evolved myocardial infarction, or dying within 90 minutes of streptokinase therapy.

**Data collection:** Data were recorded in real time on a specific form.

#### **Definitions**

<u>STEMI</u> was identified by the onset of typical chest pain lasting more than 20 minutes, unrelieved by nitrate, and associated with typical ST-segment elevation on the standard 12-lead ECG.

<u>Thrombolysis</u> strategy: Streptokinase was administered at the standard dose of 1.5 × 106 units over 60 minutes. All patients received aspirin, clopidogrel, and heparin as recommended in the European Society of Cardiology (1). An ECG was recorded just before starting the Streptokinase infusion, and a second ECG was recorded 90 minutes later.

#### The criteria for thrombolysis failure were:

-Persistent pain

-Less than 50% resolution of the ST-segment elevation in the worst lead and no accelerated idioventricular rhythm 90 minutes after the initiation of thrombolytic treatment.

**Data analysis:** Statistical analysis was carried out with SPSS (version 18.0) statistical software package.

Data were presented as frequency and percent for categorical variables and as mean with standard deviation for quantitative variables.

A univariate analysis comparing the two groups was performed, with the chi-square test with Yates' correction or Fisher's exact test when appropriate, odds ratio (OR) with 95% confidence intervals (CI), and the unpaired t-test.

The logistic regression analysis with thrombolysis failure as the dependent variable was run. The analysis was performed with a binary logistic regression and "enters" method, with an entry criterion of 0.05 and removal criteria of 0.10. Differences were statistically significant with p<0.05 or when the 95% confidence interval (CI) of the odds ratio (OR) excluded the value of 1.

Ethical consideration: The prospective study is approved by the Ethics Committee of Ben Arous

Hospital. Patient consent was obtained for the collection of data and subsequent follow-up.

**Declaration of interests**: All authors declare no competing interests.

#### Results

*General characteristics of the study population:* 

In the local registry, 1300 patients were included. Seventy percent were treated with fibrinolysis (Figure 1). We included 510 patients with a mean

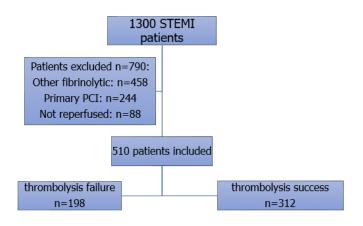


Figure 1: Flowchart of study population

age of 59+/- 11 years, and 83% of the population were male. Principal comorbidities were (%) smoking (76), hypertension (31), diabetes (28), dyslipidemia (12).

Mean parameters at admission were: Systolic blood pressure: 139±34 mmHg; diastolic blood pressure: 87±18 mmHg; Heart rate: 79±21 bpm; Respiratory Rate: 18±4; SpO2: 96±11%.

The mean delay from chest pain to first medical contact was 181 +/-141 minutes.

The initial ECG analysis resulted in the diagnosis of acute inferior myocardial infarction (52%), anterior myocardial infarction (38%), acute inferoposterior myocardial infarction (31%), acute anterolateral myocardial infarction (9%), and acute extended anterior myocardial infarction (28%).

Initial hemodynamic complications were dominated by (%): hypotension (17), bradycardia (12), acute heart failure (11), cardiogenic shock (5), ventricular fibrillation (4.5). Failure was observed in 37% (n=198) of patients.

In-hospital mortality rate among patients treated with Streptokinase was 1%.

#### Comparative study:

The comparative study between patients who failed thrombolysis and patients who succeeded thrombolysis identified Gender, smoking, delay of consultation, and extended anterior wall as related factors to fibrinolysis failure (Table 1).

Cardiogenic shock was more frequent in the group of failed thrombolysis (Table 1). Univariate analysis is represented in Table 2. In multivariate analysis, we identified four independent factors of thrombolysis failure: female gender, active smoking, delay pain to first medical contact >180 minutes, extended anterior infarct, Killip>III, and cardiogenic shock (Table 2).

Table 1: Comparative analysis of the groups

	Failure	Success	p-value
	N=198	N=312	•
Sex ratio	3.3	7.18	0.003
Mean age (years) ±SD	60±11	58±11	0.061
Risk factors: Current smoking n (%) Hypertension n (%) Diabetes n (%) Dyslipidemia n (%)	133(68) 69(35) 65(33) 30(15)	252(81) 87(28) 76(24) 31(10)	<0.001 0.095 0.036 0.076
Median delay pain to FMC (min) ±SD	224±170	153±116	< 0.001
Extended anterior infarct n (%) Clinical criteria	65(33)	66(21)	0.003
Heart rate (bpm) ±SD	80±24	78±40	0.179
Systolic blood pressure (mmHg) ±SD	136±37	140±33	0.278
Killip class ≥3 Door to needle time (min) ±SD	12(6) 27±19	2 26±17	<0.001 0.535
Creatinine (μmol/l) ±SD Glycemia (mmol/l) ±SD	86±39 12±6	81±21 10±5	0.037 0.002
Bradycardia n (%)	17 (9)	45 (14.5)	0.05
Acute heart failure n (%)	12 (6)	14 (4.5)	0.415
Cardiogenic shock n (%)	21 (11)	5 (2)	< 0.001
Cardiac arrest n (%)	10 (5)	17 (5.5)	0.858

Table 2: Predictors of thrombolysis failure

	univariate analysis		Multivariate	analysis
Characteristics	HR (95%) CI	p-value	HR (95%) CI	p-value
	2.13 (1.35-	•	2.054(1.27-	•
Female Gender	3.37)	< 0.001	3.30)	< 0.001
	2.14 (1.46-		1.89 (1.14-	
Current smoking	3.16)	< 0.001	3.11)	0.012
Ü	0.697			
Diabetes	(0,463-			
	1,006)	0.005		
Pain to FMC >180	1.602 (1.32-		2.413(1.67-	
min	1.93)	< 0.001	3.47)	< 0.001
<b>Extended anterior</b>	2,68 (1,59-		2.015(1.05-	
infarct	3,56)	0,02	3.84)	0.033
Villin along >2	2.02 (1.59-		5.19 (1.06-	
Killip class ≥3	2,57)	< 0.001	25.2)	0.009
Creatinine	1.18 (0.90-			
>80µmol/l	1.55)	0.245		
Glycemia	1.05 (0.79-			
>8mmol/l	1.39)	0.71	_	
Cardiogenic	1.837 (1.47-		7.12(1.66-	0.000
shock	2.29)	< 0.001	30.48)	0.008

CI: confidence interval, FMC: first medical contact, Min: minutes.

#### **Discussion**

In this study, we have studied the extent of failed thrombolysis in 510 patients admitted to the emergency department for STEMI and treated with Streptokinase. We have also studied the association of failed fibrinolysis with demographic, clinical, and prognostic variables, and we identified predictive factors of fibrinolysis failure.

The extent of thrombolysis failure was 37%. In several studies, it varies from 15 to 50%. It depends on the criteria used for failed thrombolysis, the drug used, and the inclusion and exclusion criteria used. In Sudhindra Rao et al study (5) failed thrombolysis was observed in 40% of patients using the same criteria as the present study. Purcell IF et al (6), who used 120min ECG post thrombolysis with Streptokinase, and Katyal VK et al (7), who used < 30% at 90min as criteria, observed 34% failed thrombolysis.

Failed fibrinolysis had a higher percentage of females in the present study, and it represents an independent predictive factor of failure with an odds ratio = 2.054. Similar observation was made in GISSI-2(8), Gabriel IB et al (9), and M Sezer et al (10) studies. It can be explained by the high rate of atypical presentation and late consultation among women (11).

Diabetes is one of the important cardiovascular factors, and diabetics have a poor prognosis after STEMI, which can be partly due to abnormal microvascular flow. In the present study, diabetics had a higher trend of failed fibrinolysis. The same results were found by Keshav Singh et al (12).

Smoking was seen in a higher percentage of patients with successful thrombolysis in this study. Similar observation was made by GISSI (8) and Sezer et al. (10), Zahger et al (13) showed that smoking was significantly associated with successful thrombolysis and lower mortality, which can be attributed to the incidence of acute MI in younger age and a lesser atherosclerotic burden, and more thrombus at the site in smokers.

Time from onset of symptoms to first medical contact in this study was significantly higher in the failed group (224±170 min) when compared to the successful group (153±116 min). GISSI-2 (9) showed a significantly higher proportion of successful thrombolysis in patients presenting within 3 hours. We found that presentation after 180 minutes was an independent predictive factor of fibrinolysis failure. In this context, Bonnefoy et al. (14) demonstrated the non-inferiority of thrombolysis compared to primary PCI when performed in the first three hours.

An extended anterior wall myocardial infarction was seen in 33% and 21% of patients with failed and successful thrombolysis, respectively. There was a trend towards a higher occurrence of failed thrombolysis in anterior wall MI. Gabriel IB et al (9) and GISSI 2(10) showed this observation in significant proportions.

The pathophysiology of those findings was explained by the fact that the length of the culprit artery distal to left-system lesions was longer, and a greater percentage of the vessel lay distal to the culprit stenosis in left-system lesions It could be speculated that slower flow in the left system may

be related to more extensive necrosis and increased myocardial edema as a result of the large myocardial mass subtended by the longer arteries(15).

In the present study, cardiogenic shock was identified as an important predictive factor of thrombolysis with an odds ratio of 7.12. In the SHOCK registry trial (16),Successful thrombolysis was obtained only for 26% of patients in cardiogenic shock. One mechanism for failure thrombolysis associated cardiogenic shock may be a greater impairment of coronary blood flow, and thus a lower effect of fibrinolytic agents, compared with patients with better hemodynamics (17).

#### Conclusion

In STEMI patients seen in Tunisia, Streptokinase had a failure rate of 37%. Female gender, a longer delay from pain onset to first medical contact (more than 3 hours), extended anterior infarction, and cardiogenic shock were highly predictive of thrombolysis failure. Patients with those criteria may benefit from an invasive reperfusion strategy earlier to improve morbidity and mortality or fibrinolysis with specific agents as a part of the pharmaco-invasive strategy.

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# Prognostic impact of prehospital management in traumatic brain injury

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#### **ABSTRACT**

**Background:** Traumatic brain injury remains a major global health concern, with prehospital management playing a decisive role in both survival and neurological recovery. The actions performed at the accident scene can prevent or exacerbate secondary brain injury, particularly through their impact on oxygenation, perfusion, and the quality of immobilization. **Objective:** To assess the prognostic and functional impact of prehospital management among patients with traumatic brain injury admitted to the intensive care unit.

**Methods:** A retrospective, descriptive, and analytical study was conducted over six months (January–June 2025) in the medical intensive care unit. We included all patients over 16 years old admitted for TBI with documented prehospital care. Functional outcomes were categorized using the Glasgow Outcome Scale.

Results: Twenty-seven patients were included (median age 33 years; 74.1% male). Road traffic accidents accounted for 96.3% of cases. The median of prehospital Glasgow Coma Scale score was 10. Prehospital care was provided mainly by the Mobile Emergency and Resuscitation Service (59.5%). Treatment included oxygen therapy (25.9%), intubation (25.9%), and cervical immobilization (100%). The median delay between trauma and hospital arrival was 30 minutes. Mortality reached 18.5%. Pupillary abnormalities at both prehospital and admission stages were significantly associated with mortality (p = 0.02). Longer Intensive care unit and in-hospital stays were significantly associated with poor functional outcomes (p = 0.034 and p = 0.04, respectively). Conclusion: The outcomes of TBI patients depend not only on injury severity but also on the quality and timeliness of prehospital management. Rapid prehospital Mobile Emergency and Resuscitation Service medical team, continuous monitoring, and safe transport significantly reduce mortality and functional disability.

**Keywords:** Traumatic brain injury, prehospital care, prognosis, intensive care, functional outcome, mortality.

#### Introduction

Traumatic brain injury (TBI) remains a major global public health issue (1). Initial management, particularly during the prehospital phase, plays a crucial role in determining both vital and functional prognosis (2–4). The period between the trauma and hospital admission is critical: actions performed at the accident scene can either prevent or worsen secondary brain injuries. Inadequate cerebral oxygenation and perfusion, delayed transfer, or insufficient immobilization can compromise survival and neurological recovery (5).

Prehospital emergency systems differ from one country to another (6). In Tunisia, prehospital management of trauma victims involves several actors: the Mobile Emergency and Resuscitation Service (MERS), the Civil Protection teams, and private ambulances.

The objective of this study was to evaluate the prognostic and functional impact of prehospital management among patients with traumatic brain injury admitted to the intensive care unit.

#### Methods

This was a retrospective, descriptive, and analytical study conducted over six months (January–June 2025) involving patients admitted for traumatic brain injury to the emergency room of Habib Bourguiba University Hospital in Sfax. We included all patients aged over 16 years admitted for TBI, regardless of severity, who had documented prehospital management. We excluded patients secondarily transferred from another hospital and those with incomplete

records. Data were collected from medical files and prehospital care forms. Functional outcomes were categorized as poor (GOS 2–3) or good (GOS 4–5). Data analysis was performed using SPSS software (version 25.0).

#### **Results**

A total of 27 patients were included. The median age was 33 years (range 16-72), with a male predominance (n = 20; 74.1%). Road traffic accidents were the main cause (n = 26; 96.3%). The median prehospital Glasgow Coma Scale (GCS) score was 10; eight patients (29.6%) had a  $GCS \le 8$ . Pupillary examination showed normal pupils in 23 cases (85.2%), unilateral mydriasis in 2 (7.4%), and bilateral mydriasis in 2 (7.4%). Vital distress signs were observed as circulatory distress (n = 2; 7.4%), respiratory distress (n = 7; 25.9%), and neurological distress (n = 22; 81.5%). Vomiting occurred in five patients (18.5%). The median delay between trauma and the arrival of emergency services was 15 minutes (5-30 minutes). Prehospital care was provided by SMUR in 16 patients (59.5%), by Civil Protection in 3 (11.1%), and by type B ambulances in 1 (3.7%).Prehospital interventions included oxygen therapy (n = 7; 25.9%), intubation (n = 7; 25.9%), hemodynamic stabilization by using fluid filling (n=4;14.8%) and catecholamines (n=4;14.8%), spine immobilization in all patients (100%), sedation (n = 7; 25.9%), and continuous monitoring (n = 7; 25.9%). On arrival to the emergency room, the median time from trauma to hospital arrival was 30 minutes (5-60 minutes). The median GCS score was 13 (mean 10.05  $\pm$ 

4.68). Pupils were normal in 22 cases (81.5%), unilaterally dilated in 4 (14.8%), and bilaterally dilated in 1 (3.7%). Vital distress signs were present in 16 patients (59.3%). Associated injuries included polytrauma (n = 21; 77.8%), thoracic trauma (n = 18; 66.7%), abdominal trauma (n = 13; 48.1%), and bone fractures (n = 24; 88.9%). All patients were admitted to the Intensive Care Unit (ICU). Neurosurgery was required in 5 patients (18.5%). The median ICU stay was 18 days (5–72), and the median total in-hospital stay was 21 days (6-72). Complications included infections (n = 21; 77.8%) and seizures (n = 9; 33.3%). Tracheostomy was performed in 12 patients (44.4%). The Glasgow Outcome Scale (GOS) at discharge was: death (GOS 1) = 5(18.5%), vegetative state (GOS 2) = 1 (3.7%), severe disability (GOS 3) = 2(7.4%), moderate disability (GOS 4) = 5 (18.5%), and good recovery (GOS 5) = 14 (51.9%). Table 1 shows that prehospital bilateral mydriasis admission and myosis or normal light reflex at admission were the only factors significantly associated with mortality (p = 0.02 for both). Table 2 shows that ICU stay duration and total hospital stay were the only factors significantly associated with poor functional outcome (GOS 2-3) (p = 0.034 and p =0.04, respectively).

#### **Discussion**

Traumatic brain injury remains one of the leading causes of mortality and acquired disability worldwide. In developing countries, where road traffic accidents are frequent and prehospital resources are limited, prehospital care is a major

Table 1: Factors Associated with Mortality

Median age (years)       36       20       0         Sex (M); n(%)       15 (68.18)       5 (100)       0         Pre-hospital Glasgow Coma Scale (median)       10       10       0         Pupil status (pre-hospital)       18 (81.81)       3 (60)       0         - Unilateral mydriasis; n(%)       2 (9.09)       0       0         - Bilateral mydriasis; n(%)       0       2 (40)       0         Pre-hospital vital distress signs       - Circulatory distress; n(%)       18 (81.81)       5 (100)       0         - Respiratory distress; n(%)       16 (72.72)       4 (80)       0         - Neurological distress; n(%)       17 (77.27)       5 (100)       0         - Vomiting; n(%)       4 (18.18)       1 (20)       0         Median delay between trauma and       4 (18.18)       1 (20)       0	
Sex (M); n(%)       15 (68.18)       5 (100)       0         Pre-hospital Glasgow Coma Scale (median)       10       10       0         Pupil status (pre-hospital)       0       18 (81.81)       3 (60)       0         - Unilateral mydriasis; n(%)       2 (9.09)       0       0         - Bilateral mydriasis; n(%)       0       2 (40)       0         Pre-hospital vital distress signs       0       2 (40)       0         - Circulatory distress; n(%)       18 (81.81)       5 (100)       0         - Respiratory distress; n(%)       16 (72.72)       4 (80)       0         - Neurological distress; n(%)       17 (77.27)       5 (100)       0         - Vomiting; n(%)       4 (18.18)       1 (20)       0         Median delay between trauma and first aid arrival (min)       15       20       0	р
Pre-hospital Glasgow Coma Scale (median)       10       10       0         Pupil status (pre-hospital)       - Myosis or normal light reflex; n(%)       18 (81.81)       3 (60)       0.         - Unilateral mydriasis; n(%)       2 (9.09)       0       0         - Bilateral mydriasis; n(%)       0       2 (40)       0.         Pre-hospital vital distress signs       - Circulatory distress; n(%)       18 (81.81)       5 (100)       0         - Respiratory distress; n(%)       16 (72.72)       4 (80)       0         - Neurological distress; n(%)       17 (77.27)       5 (100)       0         - Vomiting; n(%)       4 (18.18)       1 (20)       0         Median delay between trauma and first aid arrival (min)       15       20       0	.8
(median)       10       10       0         Pupil status (pre-hospital)       18 (81.81)       3 (60)       0.         – Unilateral mydriasis; n(%)       2 (9.09)       0       0         – Bilateral mydriasis; n(%)       0       2 (40)       0.         Pre-hospital vital distress signs         – Circulatory distress; n(%)       18 (81.81)       5 (100)       0         – Respiratory distress; n(%)       16 (72.72)       4 (80)       0         – Neurological distress; n(%)       17 (77.27)       5 (100)       0         – Vomiting; n(%)       4 (18.18)       1 (20)       0         Median delay between trauma and first aid arrival (min)       15       20       0	.2
- Myosis or normal light reflex; n(%)	.6
- Unilateral mydriasis; n(%) 2 (9.09) 0 0 - Bilateral mydriasis; n(%) 0 2 (40) 0.  Pre-hospital vital distress signs - Circulatory distress; n(%) 18 (81.81) 5 (100) 0 - Respiratory distress; n(%) 16 (72.72) 4 (80) 0 - Neurological distress; n(%) 17 (77.27) 5 (100) 0 - Vomiting; n(%) 4 (18.18) 1 (20) 0  Median delay between trauma and first aid arrival (min) 15 20 0	
- Bilateral mydriasis; n(%)       0       2 (40)       0.         Pre-hospital vital distress signs       - Circulatory distress; n(%)       18 (81.81)       5 (100)       0         - Respiratory distress; n(%)       16 (72.72)       4 (80)       0         - Neurological distress; n(%)       17 (77.27)       5 (100)       0         - Vomiting; n(%)       4 (18.18)       1 (20)       0         Median delay between trauma and first aid arrival (min)       15       20       0	80
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- Respiratory distress; n(%)       16 (72.72)       4 (80)       0         - Neurological distress; n(%)       17 (77.27)       5 (100)       0         - Vomiting; n(%)       4 (18.18)       1 (20)       0         Median delay between trauma and first aid arrival (min)       15       20       0	•
- Neurological distress; n(%) 17 (77.27) 5 (100) 0 - Vomiting; n(%) 4 (18.18) 1 (20) 0 Median delay between trauma and first aid arrival (min) 15 20 0	.3
- Vomiting; n(%) 4 (18.18) 1 (20) 0  Median delay between trauma and first aid arrival (min) 15 20 0	.6
Median delay between trauma and first aid arrival (min) 15 20 0	.4
first aid arrival (min)	.6
Pre-hospital medical team	.3
•	
	.9
1	.5
	.6
Pre-hospital resuscitation measures	,
	.6
	.5
	.6 .6
	.5
- Continuous monitoring (monitor	.7
Hospital admission data	
<ul> <li>Median delay between first aid and hospital arrival (min)</li> <li>30</li> <li>30</li> </ul>	.7
admission	.3
Pupil status at admission	
- Myosis or normal light reflex; n(%) 20 (90.9) 2 (40) 0.	02
- Unilateral mydriasis; n(%) 2 (9.09) 2 (40) 0	.1
– Bilateral mydriasis; n(%) 0 1 (20) 0	.2
Vital distress at admission; n(%) 12 (54.54) 4 (80) 0	.3
Associated injuries	
– Polytrauma; n(%) 17 (77.27) 4 (80) 0	.6
- Thoracic trauma; n(%) 15 (68.18) 3 (60) 0	.5
- Abdominal trauma; n(%) 11 (50) 2 (40) 0	.5
- Bone fractures; n(%) 20 (90.9) 4 (80) 0	.4
	.5
Evolution data	
– Median ICU stay (days) 16 10 0	.2
- Median total hospital stay (days) 21 12 0	.1
Complications	
- Infection; n(%) 18 (81.81) 3 (60) 0	.3
- Seizures; n(%) 7 (31.81) 2 (40) 0	.5
- Tracheostomy; n(%) 11 (50) 1 (20) 0	

Mobile Emergency and Resuscitation Service (MERS), intensive care unit(ICU), NA: not applied

Table 2: Factors Associated with Functional Outcome

Variables	GOS 4–5 (n=19)	GOS 2-3 (n=3)	p
Median age (years)	24	21.5	0.4
Sex (M); n(%)	12 (63.15)	3 (100)	0.3
Pre-hospital Glasgow Coma Scale (median)	11	8.5	0.07
Pupil status (pre-hospital)			
<ul><li>Myosis or normal light reflex;</li><li>n(%)</li></ul>	15 (98.74)	3 (100)	0.5
<ul><li>Unilateral mydriasis; n(%)</li></ul>	2 (10.52)	0	0.6
Pre-hospital vital distress signs			
<ul><li>Circulatory distress; n(%)</li></ul>	4 (21.05)	0	0.5
<ul><li>Respiratory distress; n(%)</li></ul>	4 (21.05)	2 (66.66)	0.2
<ul><li>Neurological distress; n(%)</li></ul>	14 (73.68)	3 (100)	0.5
- Vomiting; n(%)	3 (15.78)	1 (33.33)	0.4
Median delay between trauma and first aid (min)	15	15	0.5
Pre-hospital medical team			
– MERS; n(%)	12 (63.15)	1 (33.33)	0.3
<ul><li>Civil protection; n(%)</li></ul>	3 (15.78)	0	0.7
- Type B ambulance; n(%) Pre-hospital resuscitation measures	1 (5.26)	0	0.8
– Oxygen therapy; n(%)	5 (26.31)	1 (33.33)	0.6
– Intubation; n(%)	4 (21.05)	1 (33.33)	0.7
- Catecholamines; n(%)	2 (10.52)	1 (33.33)	0.6
– Fluid filling	2 (10.52)	1 (33.33)	0.6
– Sedation; n(%)	4 (21.05)	1 (33.33)	0.7
- Continuous monitoring (scope, oximeter); n(%)	3 (15.78)	1 (33.33)	0.7
Hospital admission data			
Median delay between first aid and hospital arrival (min)	30	30	0.9
Median Glasgow Coma Scale at admission	13	8	0.5
Pupil status at admission			
<ul><li>Myosis or normal light reflex;</li><li>n(%)</li></ul>	17 (89.47)	3 (100)	0.6
<ul><li>Bilateral mydriasis; n(%)</li></ul>	2 (10.52)	0	0.7
Vital distress at admission; n(%)	9 (47.36)	3 (100)	0.1
Associated injuries			
– Polytrauma; n(%)	14 (73.68)	3 (100)	0.4
- Thoracic trauma; n(%)	15 (98.74)	3 (100)	0.3
<ul><li>Abdominal trauma; n(%)</li></ul>	9 (47.36)	2 (66.66)	0.5
<ul><li>Bone fractures; n(%)</li></ul>	17 (89.47)	3 (100)	0.7
<ul><li>Neurosurgical procedure; n(%)</li></ul>	3 (15.78)	0	0.6
Evolution data			
- Median ICU stay (days)	13	40	0.034
<ul><li>Median hospital stay (days)</li></ul>	17	41.5	0.04
Complications			
- Infection; n(%)	15 (98.74)	3	0.5
– Seizures; n(%)	6 (31.58)	1	0.7
- Tracheostomy; n(%)  Mobile Emergency and Resuscitation Service (M.)	8 (42.1) IERS), intensive o	3 care unit (ICU), N	0.1 VA: not

determinant of both vital and functional outcomes (5,7). In our series, most patients were young men with a median age of 33 years, consistent with international literature (8–10). The delay between the accident and the arrival of emergency services is a critical prognostic factor (11). In low- and middle-income countries, the average prehospital delay is around 217 minutes—much longer than in high-income settings (12). Moderate (10–60 min) and prolonged (≥61 min) delays are associated with higher mortality (OR  $\approx$  1.3) and increased 24-h mortality (OR  $\approx$  3.4–3.8) (13). Rogers et al. emphasized the "golden hour" of trauma care: every minute lost increases the risk of secondary neurological deterioration (14). Longer times from injury to hospital admission are also linked to poorer functional outcomes (15). Patients managed by a medical MERS team had significantly higher survival and neurological recovery than those managed by non-medical teams (16). The advantage of such teams lies in early intubation and assisted ventilation, stable hemodynamic management, continuous monitoring, and coordination with the receiving hospital. These findings are consistent with studies by Baxt & Moody (17) and Sampalis et al. (18), which showed significantly lower mortality in severe TBI patients receiving full prehospital resuscitation. Transport by (MERS) reduces 30-day mortality compared to transport by Civil Protection (16). Management by paramedics is associated with higher mortality and severe disability (GOS  $\leq$  3) (16,19). Initial injury severity, particularly a low GCS score, remains a robust predictor of mortality and sequelae (20), as observed in our study. Pupillary abnormalities (unilateral or bilateral mydriasis) are major neurological signs of poor prognosis (20); they were more frequent among deceased patients in our series. Early oxygenation is one of the most critical interventions (3,21). A systematic review by Shafique et al. showed that prehospital intubation reduces morbidity and mortality compared with non-intubated TBI patients (22).

Thirawattanasoot et al. found that a prehospital  $SpO_2 \ge 94\%$  correlates with better outcomes in hypotensive TBI patients (23). Maintaining adequate cerebral perfusion depends on prompt of hypotension correction through fluid resuscitation and, if necessary, vasoactive drugs. Early stabilization limits secondary ischemic damage (3). A systematic review of moderate-tosevere TBI demonstrated that hypotension is significantly associated with higher mortality (24). A multicenter cohort study confirmed that prehospital hypotension, hypoxia, or hypocapnia each increases the risk of death or disability after TBI (25). Lack of immobilization exposes patients to potential spinal cord injuries, often with devastating consequences (26).Pain and agitation increase intracranial pressure; judicious use of analgesics and sedatives under monitoring improves hemodynamic neurological stability (27,28). Prolonged ICU or hospital stays after TBI are often associated with worse outcomes, more complications, and higher functional disability at discharge (29). In our study, longer ICU and hospital stays were significantly associated with poor functional prognosis, in agreement with the literature.

Limitations: This study has several limitations. It was retrospective and relied on the quality of recorded data. The sample size was relatively small, and no long-term follow-up was performed (GOS was assessed only at discharge). Nevertheless, this study provides a realistic overview of current conditions and highlights the crucial importance of the prehospital chain in post-traumatic prognosis.

#### Conclusion

The prognosis of patients with traumatic brain injury depends not only on the initial severity of the injury but also on the speed and quality of prehospital management. Early Medical MERS team intervention, safe transport, and continuous monitoring are associated with a significant decrease in mortality and neurological sequelae.

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# Clinical and Toxicological Predictors of Intensive Care Unit Admission in Acute Poisoning: A Narrative Review of Recent Evidence (2024–2025)

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#### **Abstract**

Acute poisoning is a major cause of morbidity, mortality, and hospitalization in intensive care units. Accurate risk stratification is essential for guiding triage and optimizing outcomes.

This is a narrative review on clinical and toxicological predictors of admission in intensive care units. Databases searched included PubMed, Embase, and Scopus using the following keywords: "acute poisoning," "ICU admission," "predictors," and "toxins." Studies reporting adult or pediatric cohorts were included.

The major clinical predictors include coma (GCS ≤8), hemodynamic instability, respiratory distress, severe metabolic acidosis (pH <7.25), elevated lactate, and electrolyte disturbances. High-risk toxins—organophosphorus pesticides, paraquat, cardiotoxic drugs, and psychotropic agents—are strongly associated with critical illness. Validated scoring systems, especially the new Poisoning Mortality Score (new-PMS), outperform classical tools. Emerging biomarkers such as urinary 8-oxoGuo show promising prognostic value.

In summary, multimodal approaches integrating clinical assessment, toxin-specific risk factors, validated scoring systems, and emerging biomarkers optimize ICU triage in acute poisoning. External validation and integration into emergency workflows are needed.

**Keywords:** Acute poisoning, ICU admission, clinical predictors, toxicological predictors, scoring systems, biomarkers

Acute poisoning continues to impose a substantial global burden, resulting in hundreds of thousands of hospitalizations and significant mortality annually (1,2). ICU admission decisions are critical in toxicology, balancing timely critical care with limited resources. Historically, ICU triage relied on physician judgment, which is variable.

Recent studies highlight the utility of validated scoring systems and novel biomarkers to complete bedside assessment (1,3,5). Consistent bedside predictors include GCS ≤8, refractory hypotension, hypoxemia (PaO₂/FiO₂ <300), and severe metabolic acidosis (pH <7.25) (4,6). Novel scoring systems such as new-PMS, REMS, and NEWS2 outperform traditional PSS in predicting adverse outcomes (1,3,6).

This review summarises recent evidence on clinical and toxicological predictors, scoring systems, biomarkers, and practical integration into emergency and critical care practice.

A structured narrative search was conducted across PubMed, Embase, and Scopus for articles

published between January 2024 and September 2025. Keywords included: "acute poisoning," "ICU admission," "toxins," "scoring system," and "biomarkers." Studies reporting predictors of ICU admission in adult or pediatric populations were included. Reviews, case reports, and non-English articles were excluded. Two independent reviewers screened articles, resolving disagreements by consensus. Relevant studies were synthesized qualitatively.

#### Clinical Predictors of ICU Admission

Neurological impairment: GCS ≤8 indicates airway compromise and the need for mechanical ventilation (1,2,4). Pediatric new-PMS incorporates altered mental status, achieving ~85% predictive accuracy (2). Seizures, especially status epilepticus, increase ICU risk (6).

Cardiovascular instability: Refractory hypotension and need for vasopressors are strongly associated with poor outcomes, particularly in organophosphorus and paraquat poisoning (4,5,9).

Table 1. Clinical Predictors (	CU Admission in Acute Poiso	ning
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Predictor	Definition / Threshold	Impact on ICU Admission	References
Neurological impairment	GCS ≤8; altered mental status	Strong predictor; indicates airway protection and ventilation	(1,2,4)
Seizures	Status epilepticus	Increases ICU risk and length of stay	(6)
Hypotension	Systolic BP <90 mmHg, refractory to fluids	Associated with poor prognosis; need for vasopressors	(4,5,9)
Cardiac arrhythmias/shock	From beta-blockers, CCBs, TCA	Requires ICU monitoring; high mortality	(10,12)
Respiratory compromise	PaO <sub>2</sub> /FiO <sub>2</sub> <300, ARDS, mechanical ventilation	Strong predictor of ICU admission	(9,11)
Metabolic disturbances	pH <7.25, lactate >4 mmol/L, electrolyte disturbances	Independent predictor of ICU need and mortality	(5,12)

Arrhythmias and cardiogenic shock from betablockers, calcium channel blockers, or tricyclic antidepressants often require ICU monitoring (10,12).

Respiratory distress: PaO<sub>2</sub>/FiO<sub>2</sub> <300, ARDS, or mechanical ventilation predicts ICU admission (9,11). Paraquat ingestion carries high mortality with hypoxemia and pulmonary fibrosis (5,11).

Metabolic disturbances: Severe acidosis (pH <7.25), elevated lactate, and electrolyte disturbances are independent predictors of ICU need and mortality (5,12).

The main clinical predictors of ICU admission in acute poisoning are summarized in Table 1.

**Toxicological Predictors** 

High-risk toxins leading to ICU admission:

- **Organophosphorus pesticides**: cholinergic crisis, seizures, respiratory failure (4,9,10).
- **Paraquat**: pulmonary fibrosis, ARDS, multiorgan failure; early renal dysfunction and hypoxemia predict poor outcome (5,11).
- Cardiotoxic drugs (beta-blockers, CCBs): refractory shock, conduction abnormalities (10).
- Psychotrope (TCA, others): seizures, coma, arrhythmias (12).

The most common high-risk toxins and their associated ICU complications are presented in Table 2.

Prediction Models and Scoring Systems

New-PMS is robust for adults and children, achieving an AUC 0.947 in adults (1) and ~85% predictive accuracy in pediatric cohorts (2). REMS, NEWS2, and toxin-specific scores (e.g., aconite, sedative-hypnotics) also show good discriminative ability (6–9).

An overview of validated prediction models and scoring systems is shown in Table 3.

#### **Novel Biomarkers**

Urinary 8-oxo-7,8-dihydroguanosine (8-oxoGuo) correlates with multi-organ dysfunction, ICU length of stay, and mortality (6). Machine learning models integrating biomarkers demonstrate high predictive accuracy in patients requiring hemodialysis (14).

Epidemiological and Regional Considerations

Poisoning patterns vary by geography: pesticides are the most common in rural regions; pharmaceuticals and recreational drugs in urban areas (4,15). Regional toxicological profiles influence model applicability. Emerging psychoactive substances require continuous model updates (7).

Table 2. High-Risk Toxins Associated with ICU Admission

Toxin / Class	Typical ICU Complications	Key Prognostic Indicators	References
Organophosphorus pesticides	Cholinergic crisis, seizures, respiratory failure	Early recognition; airway support; hypotension	(4,9,10)
Paraquat	Pulmonary fibrosis, ARDS, multi-organ failure	Hypoxemia, renal dysfunction	(5,11)
Cardiotoxic drugs (beta- blockers, CCBs)	Refractory shock, arrhythmias, conduction abnormalities	Hemodynamic instability, need for pacing or ECMO	(10)
Psychotropic medications (TCA, others)	Seizures, coma, arrhythmias	GCS ≤8, cardiovascular instability	(12)

Table 3. Prediction Models and Scoring Systems

Model / Score	Population	Key Predictors	Performance / AUC	Notes / References
New-Poisoning Mortality Score (new-PMS)	Adults & Pediatrics	GCS, hypotension, respiratory failure, metabolic derangements	0.947 (adults)	Outperforms MEWS & PSS; validated in multiple cohorts (1,2)
Pediatric new- PMS	Pediatric	Altered mental status, seizures, metabolic derangements	~85% accuracy	Adapted from adult new-PMS; superior to PSS (2)
REMS	Adult	GCS, BP, HR, SpO <sub>2</sub>	AUC ~0.85	Useful in general ICU risk stratification (6,8)
NEWS2	Adult	Vital signs, oxygen requirements	Highest accuracy in predicting ICU admission	Simple bedside tool, validated prospectively (6,8)
Toxin-specific scores	Adult / Pediatric	Toxin-specific features (e.g., aconite, sedative-hypnotics)	Variable	Supports precision risk stratification (7,9)

#### Implementation and Future Directions

Challenges include reliance on laboratory parameters not available at triage and limited integration into electronic health records. Machine learning approaches show promise for real-time triage (10,14). Future priorities: external validation, rapid point-of-care biomarkers, AI-assisted triage tools.

#### **Conclusions**

ICU admission in acute poisoning is predicted by the presence of coma, hemodynamic instability, respiratory failure, and severe metabolic acidosis. High-risk toxins—organophosphorus pesticides, paraquat, cardiotoxic drugs, psychotropics—remain strongly linked to critical illness. Validated scores, especially new-PMS, show high predictive accuracy. Emerging biomarkers such as urinary 8-oxoGuo may refine prognostication. Multimodal approaches integrating bedside assessment, toxin-specific risk factors, validated scores, and emerging technologies optimize ICU triage.

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**Ethical Statement:** This narrative review was conducted in accordance with the principles of the *Declaration of Helsinki* (as revised in 2013). As this study did not involve human participants or identifiable personal data, formal ethical approval and informed consent were not required.

**Competing Interests:** The authors declare no competing interests.

**Funding:** The authors declare that no specific grant from any funding agency in the public, commercial, or not-for-profit sectors was received for this work.

#### **Authors' Contributions**

• **Dr Neila Maaroufi:** conceptualization, patient management, manuscript drafting, figure preparation.

- **Dr Sirine Neji:** literature review, discussion drafting, manuscript editing.
- **Dr Ihsen Abdeslem:** supervision, critical review, final approval of the manuscript.
- **Dr Dorsaf Moualhi:** discussion drafting, manuscript editing.
- **Dr Leila Jedidi:** supervision, final approval of the manuscript.

### Imaging features of breast tuberculosis

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#### **Abstract**

Background: Tuberculosis of the breast is a rare disease, as its incidence remains low even in countries where tuberculosis's incidence is high. It is often overlooked and misdiagnosed as bacterial mastitis. This work aimed to describe the different mammographic and sonographic findings of breast tuberculosis and to outline some imaging features of breast magnetic resonance imaging.

Methods: We conducted a prospective study including 13 patients with breast tuberculosis between 2020 and 2024.

Results: Mammography was abnormal in 10 investigated patients. The main result was an asymmetric density that was diffused in two cases and local in the other cases. Two cases demonstrated multiple mass lesions. Ultrasound was performed in all patients. Six patients presented with a well or poorly defined mass lesion. Four patients presented with ductal dilatation with echogenic components. Two cases have fistulized to the skin surface. Breast magnetic resonance imaging was performed in three patients. One patient had a bilobed mass enhancement on T2 intermediate intensity. The second patient had multiple confluent mass enhancements with irregular margins.

Conclusions: Breast tuberculosis presents with a wide spectrum of imaging features that can closely mimic both inflammatory and malignant breast conditions. Mammography and ultrasound remain key diagnostic tools, while MRI can provide additional information in complex cases. In the emergency setting, ultrasound should be emphasized as the first line modality and raising physician awareness of this pathway is crucial for timely diagnosis and management.

**Keywords:** Breast Tuberculosis; Mammography; Ultrasonography mammary; Magnetic Resonance Imaging

#### Introduction

Tuberculosis (TB) of the breast is a rare disease, as its incidence remains low even in countries where tuberculosis's incidence is high (1). It accounts for < 0.1% of all known breast diseases globally (2). It is a rare manifestation due in part to breast tissue being remarkably resistant to tuberculosis, because it provides an infertile environment for the survival and multiplication of Tuberculosis bacilli (3). Risk factors include females of reproductive age, multiparity, and Human Immunodeficiency Virus (HIV) coinfection (4,5). TB is often overlooked and misdiagnosed as bacterial mastitis. Therefore, the purpose of this work was to study the different mammographic and sonographic findings of breast TB and to outline some imaging features of breast magnetic resonance imaging (MRI).

#### Methods

#### Patient population

In this prospective study, 13 patients with breast TB were included between 2020 and 2024. Final diagnosis was confirmed by histology proof in 10 cases (obtained on micro biopsy (n=7) or on operative specimen (n=3)), and by proofing treatment in the remaining 3 cases. Exclusion criteria were isolated axillary nodes localization.

#### • Imaging data acquisition and analysis

Ultrasound was performed in all patients, 10/13 patients underwent mammography, and three patients underwent breast MRI. The MRI protocol consisted of transversal Dynamic Contrast Enhanced (DCE) and axial T1-weighted and T2-weighted sequences. Imaging findings were

assessed by both junior and senior radiologists using the 2013 Breast Imaging-Reporting and Data System (BI-RADS) lexicon established by the American College of Radiology.

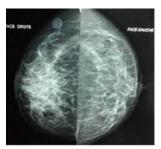
#### **Results**

Demographic data and clinical results

The mean age in our patients was 37±12 years. Of the 13 cases (all female), nine presented a palpable mass/breast nodularity, two patients presented with pain/swelling, and the remaining patients presented with nipple discharge. One patient was pregnant at the time of the first presentation. All patients were Tunisian.

#### *Imaging results*

Mammography was abnormal in 10/10 investigated patients. All of them had an asymmetric density that was disseminated in two cases (Fig.1) and local in the other cases. Two patients had multiple mass lesions, two had skin thickening, and only one showed microcalcifications without suspect grouping.





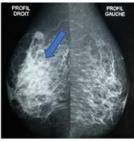


Figure 1: Standard mammogram of face, oblique and craniocaudal view of the right and left breast showing an asymmetric density in the upper quadrants of the right breast (arrow)

Ultrasound was performed in all patients. Six patients had a well or poorly defined mass lesion (Fig.2).

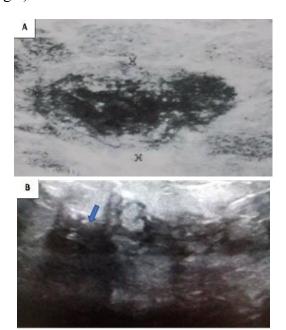


Figure 2: Ultrasound image showing a well (A) or poorly defined (B) mass (arrow)

The disseminated form was seen in two patients having multiple collections, mostly containing debris. Four patients presented with ductal dilatation with echogenic components (Fig.3).

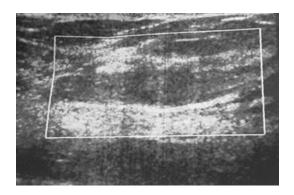


Figure 3: Ultrasound image of ductal dilatation with an echogenic component

Two cases have fistulized to the skin surface (Fig.4). There were no patients presented with abnormal axillary lymph nodes (with cortical

thickness>5mm or replacement of the fatty hilum of the node).

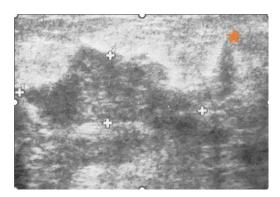


Figure 4: Ultrasound image showing a hypoechoic mass that has fistulized to the skin surface (Star)

MRI was performed on three patients. One patient presented a bilobed mass enhancement (Fig.5) with intermediate intense T2, surrounded by a hypointense, regular and fine halo, hypointense T1, and an early and heterogeneous enhancement (curve type 2).

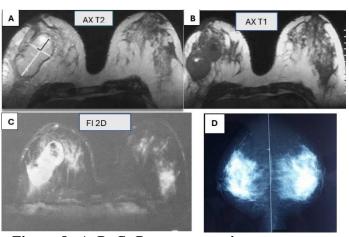


Figure 5: A, B, C: Breast magnetic resonance imaging showing a bilobed mass enhancement of the external quadrants of the right breast. D: Standard mammogram showing asymmetric density of the right breast corresponding to the mass already shown on A B C

The second patient had multiple confluent mass enhancements with irregular margins. One of these masses presented a rim enhancement pattern, classified at ACR5 (Fig.6,7). The remaining patient was identified as non-mass enhancement (NME).

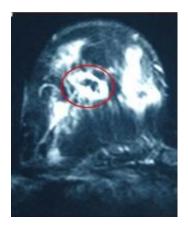


Figure 6: Breast magnetic resonance imaging showing multiple mass enhancements; a mass (encircled) with a rim enhancement

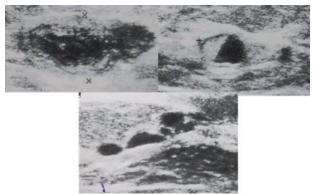


Figure 7: Ultrasound images of multiple and different masses

#### **Discussion**

Imaging findings in our series demonstrated a wide spectrum of appearances across mammography, ultrasound, and MRI, underscoring the diagnostic challenge posed by these entities. Mammography was abnormal in all investigated patients, most frequently showing asymmetric densities and, less commonly, microcalcifications. These findings, although

non-specific, require further evaluation due to overlap with malignancy features (6).

Ultrasound, the most accessible and widely used modality in inflammatory breast disease, revealed diverse patterns including both well-defined and poorly defined masses, ductal dilatation with echogenic contents, and complex collections, of had some which fistulized. These characteristics are often associated with granulomatous mastitis, abscesses, or periductal mastitis (7). The absence of suspicious axillary lymphadenopathy in all patients supports a benign inflammatory etiology in most cases, although clinical correlation and histopathology remain essential.

MRI, although limited to three patients, added valuable diagnostic information in complex or equivocal cases. The imaging patterns ranged from bilobed enhancing masses to non-mass enhancement (NME), with one case classified ACR5 due to rim enhancement and irregular margins. Such features can mimic malignancy, reinforcing the importance of histopathologic confirmation to avoid overtreatment (8).

In the emergency setting, ultrasound represents the imaging modality of choice, given its rapid availability, non-invasiveness, and ability to distinguish between solid and cystic lesions, evaluate abscess formation, and guide drainage if needed. It is particularly useful for patients presenting with acute painful swelling, suspected abscess, or fistulization, where timely diagnosis and intervention are critical. Mammography has little role in the acute phase due to limited

sensitivity in the presence of pain, edema, and dense parenchyma, and is usually deferred once the acute process subsides. MRI, while highly sensitive for characterizing indeterminate lesions and assessing disease extent, is not routinely indicated in emergencies due to limited accessibility, higher cost, and the need for patient stability. Thus, in urgent clinical scenarios, ultrasound not only provides essential diagnostic information but also facilitates immediate therapeutic procedures such as aspiration or biopsy.

For this reason, raising awareness among emergency physicians about the appropriate imaging pathway is essential. Familiarity with the ultrasound-first approach helps ensure rapid triage and timely drainage or biopsy, prevents misdiagnosis as malignancy, and reduces delays in initiating anti-tuberculous therapy. Such awareness ultimately improves patient outcomes and limits complications related to delayed or inappropriate management.

The commonest presentation of breast TB is a firm, painful or painless unilateral breast lump with or without ulceration suggestive of carcinoma on physical examination (2). Tuberculous mastitis might mimic breast cancer both clinically and radiologically (2). McKeown et al. (9) first described the five main types of infection within the breast: nodular mastitis (the major form in our study), disseminated mastitis, sclerosing mastitis, mastitis obliterans, and acute miliary mastitis (extremely rare). Nodular caseous often appearance TB mimics the

fibroadenoma or collection on ultrasound, which demonstrates no vascularity. Later in the disease, the lesions begin to develop a fistula to the nipple areolar complex or skin, thus appearing irregular and mimicking carcinomas with ill-defined margins (10).

In disseminated mastitis, multiple small anechoic fluid collections are scattered through the segment of the breast. Background vascularity is usually normal, although hyper-vascularity like bacterial mastitis can also be observed. Fistula and skin thickening, from the zones of abnormality, can develop particularly later in the disease. A single quadrant or multiple quadrants of the breast can be affected (11).

Tuberculous mastitis obliterans is characterized by duct infection, which occludes the ducts, resembling a cystic mastitis. Lesions present as well or poorly defined hypoechoic and anechoic collections, most containing debris, which mimic the appearance of classical bacterial mastitis and may be associated with increased vascularity. The imaging features of sclerosing mastitis mimic inflammatory carcinoma with ill-defined textural change (10).

As the diagnosis of breast TB is difficult, we propose a diagnostic pathway to increase awareness of this pathology, to ensure timely diagnosis, enable prompt diagnosis, and reduce complications' rates:

• TB should be considered in all patients presenting with an abscess or atypical mass.

• Ultrasound followed by core biopsy and tuberculosis culture is advocated to establish the diagnosis.

#### Conclusion

Breast tuberculosis remains a rare and often misdiagnosed condition, particularly in regions where TB is endemic. Its nonspecific clinical and imaging features frequently mimic more common entities such as bacterial mastitis or malignancy. A high index of suspicion, supported by appropriate imaging—especially ultrasound and MRI—along with histopathological confirmation, is essential for accurate diagnosis and timely management. In the emergency setting, awareness of the imaging pathway—where ultrasound serves as the first-line modality—facilitates rapid triage and intervention, prevents misclassification as malignancy, and reduces unnecessary procedures as well. Raising awareness among emergency physicians is therefore crucial to improving patient outcomes and avoiding diagnostic delays.

#### Acknowledgments: None

Authors' contributions: (I) Conception and design: WF, FH, MK, (II) Administrative support: WF, FH, ED; (III) Provision of study materials or patients: WF, FH, AK; (IV) Collection and assembly of data: FH, ED, ZM; (V) Data analysis and interpretation: WF, FH, MK, AK, ED; ZM (VI) Manuscript writing: WF, FH; (VII) Final approval and revision of manuscript: All authors.

**Conflict of interest statement**: The authors have no conflict of interest to declare.

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# Penetrating Abdominal Trauma: Initial Assessment and Diagnostic Challenge: A Case Report

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#### **Abstract**

**Background:** Penetrating abdominopelvic trauma usually stems from stab wounds or gunshots. Workplace impalement by foreign objects is rare and presents a distinct diagnostic and therapeutic challenge. We report an exceptional case of trans-abdominal impalement by a metallic rod with an unusual trajectory yet minimal internal damage.

Case presentation: A 30-year-old male industrial worker was referred after a workplace accident in which a metal rod penetrated his lower back, traversing the posterior lumbar region, retroperitoneum, and peritoneal cavity. Upon arrival, he was hemodynamically stable, fully conscious (GCS 15/15), with a normal FAST. Contrast-enhanced abdominopelvic CT demonstrated the metallic rod's oblique vertical course through paravertebral muscles, psoas, retroperitoneum, and peritoneal cavity anterior to the descending colon, with signs of jejunal perforation (mesenteric air and fat stranding) but no major vascular, visceral, or skeletal injury. The rod was surgically removed; the perforated jejunal segment was resected, and a jejunojejunal anastomosis was performed. Postoperative recovery was uneventful, and the patient was discharged on day 3 in a stable condition.

Conclusions: This case highlights two key points: the first one is that even when a foreign object follows a long, high-risk trajectory, internal damage may be surprisingly limited depending on dynamics and anatomy. The second is that a negative FAST examination in a hemodynamically stable patient does not exclude significant intra-abdominal injury; timely cross-sectional imaging or surgical exploration remains essential. Awareness of atypical impalement injury patterns is crucial for accurate diagnosis and optimal outcomes.

Keywords: Abdominal Trauma; Outcomes; Emergency; Management

#### Introduction

Penetrating abdomino-pelvic trauma accounts for a significant proportion of emergency surgical admissions. Although most such injuries are due to stab wounds or gunshots, impalement by foreign objects, especially in the workplace, is rare and poses unique diagnostic and therapeutic challenges. These injuries often involve multiple organ systems and require a multidisciplinary approach.

We report a rare case of a transabdominal impalement by a metal rod with an atypical trajectory, resulting in minimal internal damage.

#### **Case Presentation**

A 30-year-old male with no medical history was referred to the emergency department by the prehospital team following a workplace accident. The patient had sustained a penetrating abdominal trauma caused by a metal rod that had entered through the lower back (**Figure 1**).



Figure 1: Clinical view demonstrating the in-situ metallic rod

Upon arrival, He was admitted without delay to the resuscitation room. The patient was in the prone position. A systematic ABCDE approach was performed; the airway was patent, with no signs of obstruction. Breathing was regular, with a respiratory rate of 16 breaths per minute. Oxygen saturation (SpO<sub>2</sub>) was 98% on room air. Auscultation revealed normal, symmetrical breath sounds without wheezing or crackles. Blood pressure was 120/70 mmHg, and heart rate was 110 beats per minute; capillary refill time was less than 3 seconds; peripheral pulses were present, symmetrical, and there were no signs of peripheral hypoperfusion or shock. Neurologically, he was fully conscious, alert, and cooperative; Glasgow Coma Scale (GCS) was 15/15, with no focal neurological deficits. On rectal examination, anal sphincter tone was preserved, indicating intact sacral neurologic function.

The metal rod had penetrated dorsally near the lumbar spine and followed an oblique intraabdominal course, ending at the level of the left iliac crest (Figure2).



Figure 2: Plain abdominal X-Ray showing the trajectory of the metallic rod.

We performed a focused abdominal sonography for trauma (FAST), which was negative.

Laboratory tests showed normal coagulation (PT 87%), platelets 280,000/mm³, hemoglobin 14.5 g/dL, creatinine 71 μmol/L, and mild hypokalemia (K<sup>+</sup> 3.2 mmol/L).

Given the patient's hemodynamic stability, we decided perform a contrast-enhanced abdominopelvic CT scan. It revealed a posterior penetrating injury caused by a linear metallic object. The entry point was in the left paravertebral lumbar zone. The trajectory followed an oblique vertical course, traversing the paravertebral muscles adjacent to the left transverse process of L3, passing through the psoas muscle, into the retroperitoneal space along the outer border of the psoas, and finally breaching into the peritoneal cavity anterior to the descending colon and superior to the sigmoid colon. (Figure3)





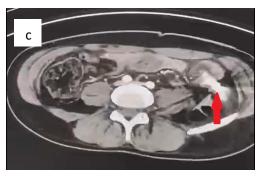


Figure 3(a) (b) (c): Trajectory of the metallic rod on the abdominopelvic CT sections

At the site of intraperitoneal entry, the object lay in proximity to the distal mesenteric vessels of small dimension and loops of the jejunum. Air bubbles were observed within the mesenteric fat, strongly suggesting perforation of a hollow viscus—most likely jejunal, given the extent of pneumoperitoneum. In addition, mesenteric fat stranding was noted in the left iliac fossa, with a thin layer of free intra-peritoneal fluid.

Antibiotics were administered, and the surgery was decided. The perforated intestinal segment was resected, the peritoneal cavity was irrigated, and a jejunojejunal anastomosis was performed. The patient was then transferred to the surgical intensive care unit. The postoperative course was uneventful, and the patient was discharged on postoperative day 3 in stable condition.

#### **Discussion**

Penetrating abdominopelvic trauma can be a complex challenge for the clinician. It commonly requires concurrent resuscitation and urgent decision-making. Although trauma is the leading cause of death worldwide, penetrating trauma is less frequent than blunt trauma and represents less than 15% of all trauma presentations (1). Studies report that penetrating abdominal trauma disproportionately affects young adult males (2).

Penetrating abdominal trauma most commonly arises from stab wounds or gunshot injuries, typically in the context of interpersonal violence. Most series report that gunshot wounds account for a substantial portion of penetrating abdominal trauma ( $\approx 64\%$ ), and stab wounds make up around 31% (3).

Accidental penetrating abdominal injuries (for example, work-related incidents or falls) remain rare compared with violent causes (2).

The most frequently affected sites in intraabdominal stab wounds are the great vessels, the diaphragm, the mesentery, the spleen, the liver, the kidneys, the pancreas, the gallbladder, and the adrenal glands. The left upper quadrant is the most common location of the wounds, followed by the left iliac fossa, the right iliac fossa, and the right iliac fossa. Stab wounds to the posterior abdomen and the flank carry an increased risk of injury to retroperitoneal structures (4). Stab wounds are penetrating in only 45–76 % of cases, and among those, just 35–61 % are perforating (5).

The severity of presentation from penetrating abdominal trauma ranges from the stable patient with pain to the hemodynamically compromised patient with active hemorrhage. Upper abdominal wounds pose a specific threat as they can cross the diaphragm into the chest (6). Penetrating injuries to the anterior abdomen can damage solid organs and/ or cause peritonitis from hollow viscus injuries. Back and flank injuries pose a risk of injury to the retroperitoneal organs without peritonitis (7). The severity is mainly related to initial bleeding and to the high risk of infection due to associated lesions, which occur more frequently than in blunt trauma. The mortality of PPT exceeds 30% (8).

Emergency management of abdomino-pelvic trauma should be performed in a level 1 trauma center. The initial clinical presentation is often dominated by hemorrhagic shock in a

hemodynamically unstable patient. Management must quickly obtain hemostasis by either surgical or radiological means.

Unstable patients with evidence of active hemorrhage should be explored surgically before time-consuming investigations are undertaken. However, simple blood tests, including crossmatching, should be undertaken for all patients regardless of status (9).

Meanwhile, Focused Abdominal Sonography For Trauma (FAST) may serve as a rapid bedside tool to detect free fluid or air within the peritoneal or pericardial cavities; nevertheless, its operator-dependence and limited sensitivity mean that a negative eFAST does not reliably exclude intra-abdominal injuries (5).

In hemodynamically stable patients presenting to the emergency department, the gold standard imaging is the contrast-enhanced CT scan. Its diagnostic accuracy in detecting significant intra-abdominal injury is high, with a sensitivity up to approximately 96% and a specificity exceeding 94% (10) (11).

According to Kyle et al. The management of penetrating abdominal trauma can be divided into: damage control surgery, definitive surgical management, and selective non-operative management(9). In our case, despite the long and high-risk trajectory of the metallic rod, the patient had a single jejunal perforation without significant vascular, visceral, or skeletal injury. The lumbar entry point in our patient, along with the oblique intraperitoneal trajectory, exposed several critical structures, including the left

kidney, ureter, descending colon, and iliac vessels. The absence of injury to these organs is therefore unexpected and noteworthy.

Such cases highlight the unpredictable nature of impalement trauma, in which even extensive penetration may cause limited tissue disruption depending on the velocity, direction, and rotational stability of the foreign body.

#### Conclusion

In penetrating abdominal trauma, hemodynamic stability and negative findings on FAST do not rule out serious intra-abdominal injury. Integrating mechanism, wound trajectory, and contrast CT imaging, with vigilant reassessment, is essential to timely diagnosis and intervention. This case illustrates that prompt surgical action, guided by imaging despite a stable presentation, can lead to a good outcome.

#### Consent

Oral consent for publication was obtained from the patient. All information has been anonymized to protect patient privacy.

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# Penetrating chest trauma with glass: Beware of occult cardiac injuries! A case report

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#### Abstract

Penetrating chest trauma involving the heart is a life-threatening emergency requiring rapid diagnosis and immediate surgical intervention. While knives or firearms most commonly cause cardiac injuries, atypical foreign bodies such as glass fragments pose similar critical risks but remain rare. This case report describes a 31-year-old male who was assaulted with a broken beer bottle, resulting in a 3 cm penetrating wound in the left anterior axillary line. Initial findings included a left hemopneumothorax and a 4 cm glass fragment lodged near the right ventricle, without pericardial effusion in E-FAT and CT scan. The patient remained hemodynamically stable, and the surgery was urgently performed. A thoracotomy was performed with the extraction of the glass fragment and repair of a non-transfixing right ventricular myocardial wound. Postoperative recovery was favorable. This case highlights the importance of cautious evaluation in penetrating chest trauma, even in the absence of pericardial effusion, and the critical role of prompt surgical management to ensure survival.

Key words: Penetrating chest trauma, Cardiac injury, Emergency, Early management

#### Introduction

Penetrating chest trauma is a life-threatening emergency, particularly when it involves the heart. The major reported causes of cardiac wounds are knives or firearms, and the survival of these injuries depends mainly on rapid diagnosis and the availability of immediate surgery (1).

We report the case of a myocardial wound caused by a glass fragment following an assault. Injuries caused by atypical foreign bodies, such as glass fragments, remain rare but pose the same hemodynamic and prognostic risks.

#### Case presentation

A 31-year-old man was admitted to the emergency room after being attacked with a broken beer bottle. The initial clinical assessment documented clear upper airways, polypnea (22 cycles/min), and normal oximetry (98% with 8 liters of oxygen via mask).

Pulmonary auscultation revealed left basilar dullness, without subcutaneous emphysema. There was a 3 cm penetrating wound in the left anterior axillary line. The blood pressure was 100/60 mmHg, and the heart rate was 88 bpm and regular. Glasgow Coma Scale score was 15/15. The general examination revealed no other associated injuries. The interview did not reveal any medical history.

Within the first management, E-FAST revealed a left pleural effusion. There was no associated pericardial or peritoneal effusion. A chest and abdominal CT scan with contrast injection was performed urgently. It confirmed a left hemopneumothorax and identified a 4 cm glass fragment incarcerated in the angula, in contact with the right ventricle (Figures 1 and Video 1).

The patient was immediately transferred to the operating room. A left posterolateral thoracotomy was performed. The glass fragment was carefully extracted with resection of the lingula. Intraoperative exploration revealed a non-transfixing myocardial wound of the right ventricle, which was sutured with two patched U-shaped stitches. The postoperative outcome was favorable.

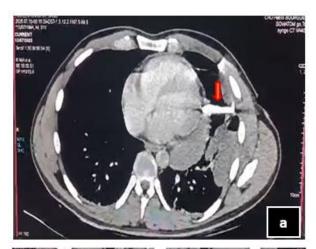




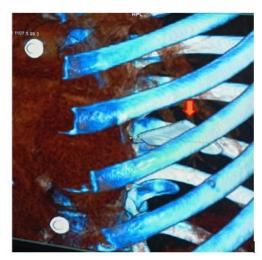
Figure 1: Transverse (a) and coronal (b) CT scans showing a 4 cm glass fragment lodged in the angula, in close contact with the right ventricle.

#### **Discussion**

Penetrating chest wounds are a life-threatening emergency due to the risk of cardiac or vascular injury. Their physiopathology is based on the direct effect of the weapon or sharp object, which can cause massive hemorrhage, tamponade, or compressive pneumothorax (1, 2).

Penetrating chest trauma accounts for a significant proportion of penetrating wounds, although its frequency varies according to geographical and socioeconomic contexts. In

the United Kingdom, a study has shown that this type of trauma accounts for 12% of all penetrating trauma cases treated in emergency departments (3).



Video 1: 3D reconstruction of the embedded glass fragment



Scan QR Code

Initial management is based on the application of Advanced Trauma Life Support principles, including securing the airway, oxygenation, hemodynamic control, and rapid additional examinations (FAST, CT scan) (4, 5). In this context, the absence of pericardial effusion on E-FAST and CT scan, particularly in stable patients, should be interpreted with great caution. Hence, the importance of close monitoring and repeating the E-FAST to look for effusion that may develop later.

Thoracotomy remains the standard surgical technique for cardiac wounds. The surgical method depends on the location of the injury and the patient's condition (anterolateral

thoracotomy in extreme emergencies, and posterolateral thoracotomy in patients with stable parameters) (6).

The outcomes are mainly correlated with initial hemodynamic stability, delay to diagnosis, and the availability of an experienced surgical team (7, 8, 9). In our observation, the absence of tamponade and rapid access to the operating room allowed for a favorable outcome.

In summary, this case highlights the importance of cautious evaluation in penetrating chest trauma, even in the absence of pericardial effusion, and the significant role of early, prompt surgical management to ensure survival.

#### References

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