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Traumatic Brain Injury Risks and Mortality in the Geriatric Patient: A Swedish Perspective

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Abstract

Background: Traumatic brain injury (TBI) is a leading cause of morbidity and mortality in older adults. In high-income settings, falls are the predominant mechanism; however, contemporary Scandinavian data describing injury characteristics and short-term outcomes are limited.

Methods: We performed a retrospective cohort study of patients aged >65 years with TBI treated at Karolinska University Hospital between 2019 and 2023, using the hospital Trauma Registry with supplementary chart review. Patients without Swedish identification numbers, prehospital deaths, and chronic haemorrhages were excluded. We described demographics, injury mechanisms, traumatic intracranial haemorrhage (tICH) subtypes, and 30-day mortality, and evaluated clinical factors associated with death.

Results: The cohort comprised 722 patients; falls accounted for 77.8% of injuries. Overall, 30-day mortality was 18.3%. Advanced age, severe injury, and pre-injury antithrombotic therapy were associated with higher 30-day mortality (p<0.05); the presence of tICH was also associated with increased mortality risk. Subdural haemorrhage was the most frequent tICH subtype (30.6%). Epidural haemorrhage showed the strongest association with mortality (OR 2.8; p=0.035).

Conclusions: Among older adults with TBI treated at a Swedish level I trauma centre, falls were the dominant injury mechanism and short-term mortality was substantial. Mortality was higher in patients of advanced age, with severe injury, receiving antithrombotic therapy, and with tICH, with heterogeneity by haemorrhage subtype. These findings support the importance of fall-prevention strategies and underscore the need for careful management of antithrombotic therapy in older adults at risk for TBI.

Keywords: Traumatic brain injury; Geriatric; Mortality; Trauma register; Intracranial haemorrhages

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Introduction

Traumatic brain injury (TBI), defined as brain injury caused by external forces [1], is a major global health concern and a leading contributor to trauma-related mortality [1,2]. Approximately 10 million people worldwide are hospitalized annually due to TBI [3], including 1 million in the EU [4], and 37,000 in Sweden [5]. TBI also imposes substantial long-term healthcare and economic burdens, with elevated costs persisting up to two years post-injury, and hospital costs for patients >65 years exceeding $2.2 billion in the United States of America [6]. As the population aged ≥60 years continues to grow rapidly [7], understanding TBI in older adults has become increasingly critical. However, geriatric patients differ significantly from younger cohorts because of higher rates of comorbidities and antithrombotic therapy use [1,8]. Furthermore, there are challenges in assessing injury severity using the Glasgow Coma Scale, as it may be difficult to accurately assess [9-11].

In high-income countries, the typical TBI mechanism has shifted from motor vehicle collisions in younger adults to low-energy falls in older adults [12-14]. This trend underscores the importance of studying population-specific risk factors. Traumatic intracranial haemorrhages (tICH)-including epidural (EDH), subdural (SDH), subarachnoid (SAH), and intraparenchymal haemorrhages (IPH)-are responsible for 40% to 50% of trauma-related deaths [4] yet remain understudied in the elderly, with few investigations assessing outcomes by haemorrhage type [5,15-17].

Existing research has predominantly focused on younger adults, with limited evidence on mortality patterns, the impact of comorbidities, antithrombotic use, and differences among tICH subtypes in older patients. Scandinavian data are particularly limited. This study aimed to characterize the demographics of adults aged ≥65 years with TBI in Stockholm, the capital of Sweden, over a five-year period (2019-2023), to determine TBI-related mortality and to evaluate whether mortality varied by tICH subtype. Additionally, the aim was to assess correlations of risk factors-namely the role of antithrombotic therapy-for both TBI occurrence and associated mortality.

Methods

This descriptive, registry-based study analysed 1,215 trauma patients recorded in the Trauma Registry at Karolinska University Hospital (KUH), Solna, between 2019 and 2023. KUH is the primary level I trauma centre in the Stockholm region, with a catchment population of approximately 2.5 to 3 million (the latter including populations from surrounding regions). Eligibility criteria included patients aged ≥65 years who met at least one of the following: trauma team activation, traumatic brain injury (TBI), or interhospital transfer within seven days of injury. Data on the presence of TBI, multi-trauma, intracranial haemorrhage (ICH), and anticoagulation use were obtained by manual review of patients' medical records, as these variables are not captured in the trauma registry. Patients with missing personal numbers and those without skull trauma, or with unclear skull trauma, were excluded. In addition, prehospital deaths, patients with suspected chronic haemorrhage, and patients with coagulation system defects were excluded (Figure 1).

All analyses were performed using IBM SPSS Statistics (version 29.0.2.0). Statistical significance was defined as $p<0.05$. Baseline characteristics are presented as counts and percentages, with age categorized into four groups. Patients were stratified by 30-day survival status; categorical variables are reported as counts and percentages, and continuous variables as mean (standard deviation, range) or median (interquartile range) for skewed distributions. Between-group differences were assessed using the independent-samples t test for continuous variables, the chi-square test for categorical variables, and the Kruskal–Wallis test for non-normally distributed continuous variables. Odds ratios (ORs) for 30-day mortality were estimated using binary logistic regression with the first category as the reference, and statistical significance was evaluated using the Wald test. ORs were rounded to one decimal place (unless greater precision was required) and p values to three decimals. Multicollinearity among intracranial haemorrhage subtypes was assessed using the variance inflation factor (VIF). Receiver operating

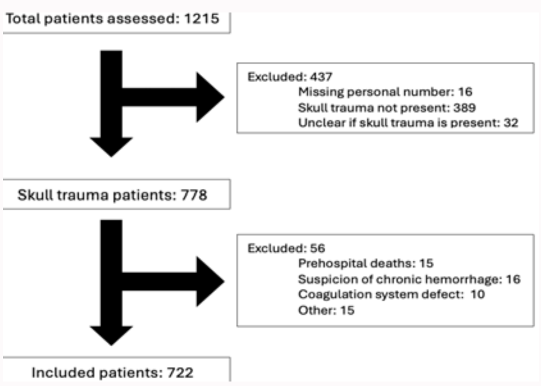

Figure 1: Flow chart illustrating the number of included and excluded patients as well as causes for their exclusion.

characteristic (ROC) curve analyses were performed to evaluate the discriminatory performance of the Glasgow Coma Scale (GCS), the number of intracranial haemorrhage (ICH) subtypes, and the pre-injury American Society of Anaesthesiologists (ASA) score for predicting 30-day mortality.

Results

Population demographics

Detailed baseline characteristics are presented in Table 1. In total, 722 patients were included. Most patients were male (61.5%, n=444) and 38.5% were female (n=278). The largest age stratum comprised patients aged ≥80 years (37.7%, n=272), followed by 75–79 years (25.5%, n=184). The remaining patients were aged 70-74 years (21.1%, n=152) and 65–69 years (15.8%, n=114). The maximum age at injury was 100 years. Polytrauma was present in 40.3% of cases (n=291), whereas 59.7% sustained isolated head injury. At the time of injury, 53.0% of patients were receiving antithrombotic medication (n=383). Information on antithrombotic therapy was missing in 1.5% of patients (n=11). Injury severity was assessed using the prehospital Glasgow Coma Scale (GCS) when available (n=565), and the emergency department GCS when the prehospital score was unavailable (n=118). GCS was undocumented in 6.6% of patients (n=48). Mild TBI (GCS 13-15) was most common (61.8%, n=446),

Table 1: Demographics of the study population (n=722).

Baseline characteristic	*n*	%
Gender		
Male	444	61.5
Female	278	38.5
Age		
65-69	114	15.8
70-74	152	21.1
75-79	184	25.5
80+	272	37.7
Multi-trauma	291	40.3
30-day mortality	134	18.3
Severity		
Mild (GCS 15-13)	446	61.8
Moderate (GCS 9-12)	106	14.7
Severe (GCS <9)	122	16.9
Unknown	48	6.6
Use of antithrombotic therapy	383	53
Pre-injury comorbidity		
ASA I	91	12.6
ASA II	208	28.8
ASA III	392	54.3
ASA IV	31	4.3
Injury mechanism		
Traffic accidents	120	16.6
Penetrating trauma	2	0.3
Blunt force trauma	30	4.2
Low energy fall (same plane)	355	49.2
High energy fall (another plane)	209	28.9

GCS: Glasgow Coma Scale; ASA: American Society of Anaesthesiologists

Table 2: Baseline characteristics stratified by 30-day survival status. Data are presented as n (%) unless otherwise specified. Age is reported as mean (standard deviation, range). p values compare survivors and non-survivors for each characteristic. p<0.05 was considered statistically significant.

Baseline characteristic	Survivors (>30-days)	Non-survivors (30-day mortality)	P-value
Gender			
Male; n (%)	363 (61,7)	81 (60.4)	**<0.001**
Female; n (%)	225 (38.3)	53 (39.6%)	
Age; Mean (±SD, range)	77.3 (7.6, 66-100)	78.0 (7.2, 66-96)	**<0.001**
65-69; n (%)	105 (17.9)	9 (6.7)	
70-74; n (%)	128 (21.8)	24 (17.9)	
75-79; n (%)	147 (25.0)	37 (24.6)	
80+; n (%)	208 (35.4)	64 (47.8)	
Multi-trauma, n (%)	234 (39.9)	57 (42.9)	
Severity			**<.001**
Mild (GCS 15-13)	412 (70.1)	34 (25.4)	
Moderate (GCS 9-12)	76 (12.9)	30 (22.4)	
Severe (GCS < 9)	66 (11.2)	56 (41.8)	
Undocumented GCS	34 (5.8)	14 (10.4)	
Use of antithrombotic medication	227 (42.7)	81 (60.9)	**<0.001**
Pre-injury comorbidity			**<0.001**
ASA I	80 (13.6)	11 (8.2)	
ASA II	184 (31.3)	24 (18.9)	
ASA III	306 (52.0)	86 (64.2)	
ASA IV	18 (3.1)	13 (9.7)	
Injury mechanism			
Traffic accidents	100 (17)	20 (14.9)	
Penetrating trauma	1 (0.2)	1 (0.7)	
Blunt force trauma	29 (4.9)	1 (0.7)	
Low energy fall (on the same level)	278 (48.8)	68 (50.7)	
High energy fall (from another level)	165 (28.1)	44 (32.8)	

SD Standard Deviation: GSC Glasgow Coma Scale; ASA: American Society of Anaesthesiologists

followed by severe TBI (GCS <9; 16.9%, n=122), whereas moderate TBI (GCS 9-12) was least frequent (14.7%, n=106). Additional baseline variables, including pre-injury American Society of Anesthesiologists (ASA) classification and injury mechanism, are presented in Table 1. Most patients had an ASA class of III or IV (58.6%, n=423). Low-energy falls were the presenting mechanism of injury in 49.2% of patients (n=355), whereas high-energy falls accounted for 28.9% (n=209). Overall, falls comprised 78.1% of injuries (n=564). Traffic accidents accounted for 16.6% of injuries (n = 120). High-energy falls were defined as falls from a height >3 m.

Types of intracranial haemorrhage

More than half of the cohort had no evidence of tICH (57.5%, n=415). Among patients with tICH, similar proportions sustained one (16.3%, n=118) or two (16.1%, n=116) haemorrhage subtypes. Three haemorrhage subtypes were less frequent (9.4%, n=68), and concomitant occurrence of all four subtypes was rare (0.7%, n=5). Across the total cohort, SDH was the most common haemorrhage subtype (30.6%, n=221), followed by SAH (28.4%, n=205) (Figure 2). When stratified by age group, SAH was the most frequent subtype in patients aged 75–79 years. Figure 2 presents subtype distributions stratified by age group and gender.

Mortality

Overall, 30-day mortality was 18.3% (n=134). Among patients who died within 30 days, the median survival was 3 days (IQR 0-11). Median survival differed by TBI severity (GCS categories): 7.5 days (IQR 2.75-13) in mild cases, 3.5 days (IQR 0-15) in moderate cases, and 1.0 day (IQR 0-4) in severe cases (p<0.001). Patients were further classified as survivors and non-survivors based on 30-day outcome (Table 2, Figures 3 and 4). Non-survivors were older than survivors. Injury severity also differed significantly between groups: severe injury accounted for 41.8% of non-survivors compared with 11.2% of survivors (p<0.001).

In univariate logistic regression (Table 3), the strongest associations with 30-day mortality were observed for the presence of all four haemorrhage subtypes (OR: 15.3, 95% CI: 2.5-94.7, p=0.003) and severe injury (GCS<8; OR: 10.3, 95% CI: 6.2-10.2, p<0.001). Multi-trauma was not associated with 30-day mortality (OR: 1.1, 95% CI: 0.8-1.7, p=0.5). Antithrombotic therapy was associated with higher odds of death within 30 days (OR: 2.1, 95% CI: 1.4-3.1, p<0.001). Antithrombotic therapy was not associated with higher odds of any ICH subtype (SAH [OR: 1.025, 95% CI: 0.7-1.4, p=0.882], SDH [OR: 1.3, 95% CI: 0.9-1.8, p=0.103], IPH [OR: 1.1, 95% CI: 0.7-1.6, p=0.703], EDH [OR: 1.1, 95% CI: 0.4-2.6, p=0.9]).

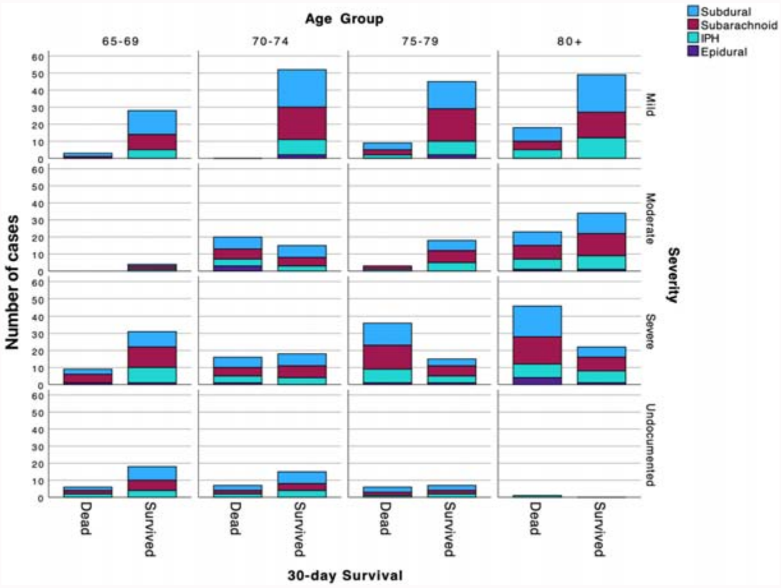

Figure 2: Distribution of traumatic intracranial haemorrhage subtypes stratified by age group and severity.

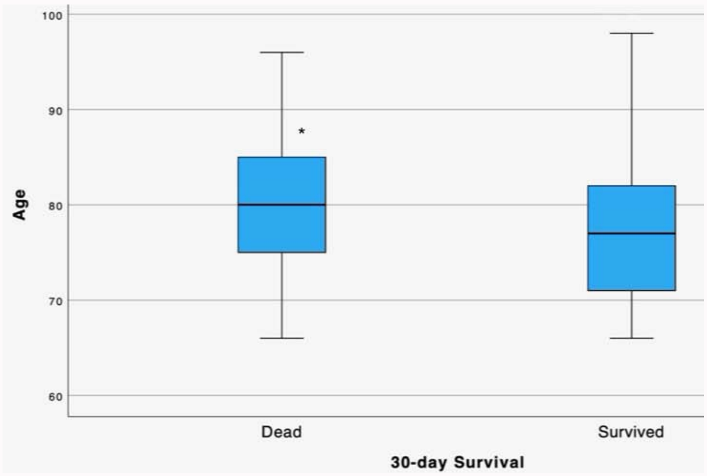

Figure 3: Boxplot illustrating the age distribution of included patients grouped by survival status. *P<0.001.

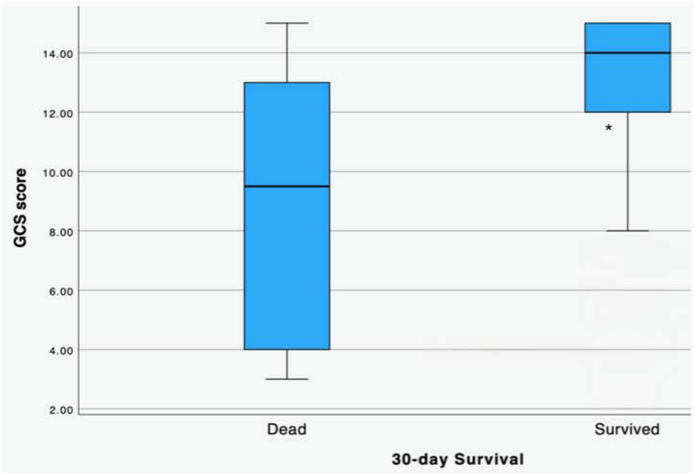

Figure 4: Boxplot illustrating the injury severity of included patients grouped by survival status. Cases where GCS was undocumented were excluded from the analysis (n=48). *P<0.001.

Table 3: Binary logistic regression for 30-day mortality. Univariate analysis predicting the primary outcome of 30-day mortality. The first group in each category serves as the reference. p<0.05 was considered statistically significant.

Baseline characteristic	Odds ratio	95% CI	P-value
Gender			
Female	Reference		
Male	1.1	0.7–1.6	0.782
Age group			
65-69	Reference		
70-74	2.2	0.975–4.9	0.058
75-79	2.9	1.4–7.5	**<.001**
80+	3.6	1.7–7.5	**<.001**
Multi-trauma	1.1	0.8–1.7	0.5
Severity			
Mild (GCS 15-13)	Reference		
Moderate (GCS 9-12)	4.8	2.8–8.3	**<.001**
Severe (GCS < 9)	10.3	6.2–17.0	**<.001**
Unknown	5	2.4–10.2	**<.001**
Use of antithrombotic therapy	2.1	1.4–3.1	**<.001**
Pre-injury comorbidity			
ASA I	Reference		
ASA II	0.9	0.4–2.0	0.9
ASA III	2	1.0-4.0	**<.05**
ASA IV	5.3	2.0–13.6	**<.001**
Type of haemorrhage*			
Subdural	2.6	1.7–4.1	**<.001**
Subarachnoid	2	1.3–3.3	**<.05**
Intraparenchymal	1.4	0.8–2.2	0.2
Epidural	2.8	1.1–7.5	**<.05**
Number of haemorrhages			
0	Reference		
1	3.2	1.8–5.5	**<.001**
2	4.4	2.6–7.4	**<.001**
3	8.6	4.8-15.4	**<.001**
4	15.3	2.5–94.7	**<.05**

*Absence of intracranial haemorrhage serves as the reference for this group. GCS: Glasgow Coma Scale: ASA: American Society of Anaesthesiologists.

The presence of ≥1 tICH subtype was associated with increased odds of 30-day mortality (OR: 3.2, 95% CI: 1.8-5.5, p<0.001), with progressively higher odds as the number of haemorrhage subtypes increased (Table 3). Among individual subtypes, EDH showed the strongest association with mortality (OR: 2.8, 95% CI: 1.1-7.5, p=0.035), followed by SDH (OR: 2.6, 95% CI: 1.7-4.1, p<0.001) and SAH (OR: 2.0, 95% CI: 1.3-3.3, p=0.003). IPH was not associated with a statistically significant difference in mortality (OR: 1.4, 95% CI: 0.8-2.2, p=0.2). Multicollinearity was low (VIF: SDH, 1.3; SAH, 1.5; IPH, 1.3; EDH, 1.1).

Receiver operating characteristic (ROC) curve analysis showed that the GCS score demonstrated good discriminatory ability, with an area under the curve (AUC) of 0.769. The number of haemorrhage types showed poor discrimination (AUC 0.280), and the pre-injury ASA score also showed poor discrimination (AUC 0.398) (Figure 5). Pairwise comparison of the ROC curves demonstrated statistically significant differences between all predictors. The AUC of the GCS score was significantly higher than that of the number of haemorrhage types (AUC difference 0.489, p<0.001) and the pre-injury ASA score (AUC difference 0.371, p<0.001). The number of haemorrhage types also differed significantly from the pre-injury ASA score (AUC difference 0.118, p=0.003).

Discussion

This study examined demographic characteristics and factors associated with 30-day mortality among older adults with traumatic brain injury (TBI), in Stockholm, Sweden, with a particular focus on traumatic intracranial haemorrhage subtypes. Falls were the predominant mechanism of injury, accounting for 77.8% of trauma cases, as well as 83.5% of the 30-day mortality (n=112), consistent with prior reports from Laic et al., Haring et al., Scotti et al., and van der Vlegel et al. [8,9,11,14]. In contrast, falls are less common in younger TBI populations, representing 27.6% of injuries among adults aged

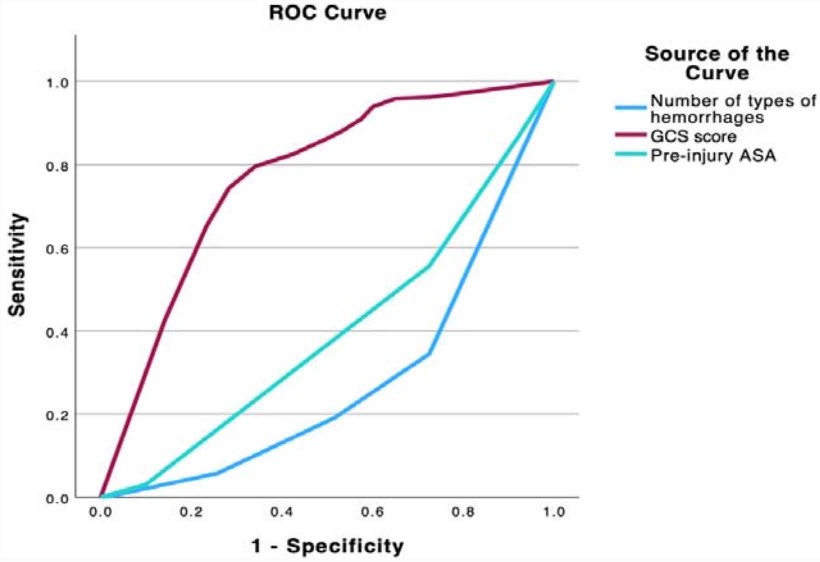

Figure 5: Receiver operating characteristic (ROC) analysis. Cases where GCS was undocumented were excluded from the analysis (n=48). GCS Glasgow Coma Scale, ASA American Society of Anaesthesiologists.

18-40 years [18] and 13% in an international adult cohort [19]. The majority of TBI studies have been conducted on younger trauma patients, with largely different mechanisms of injury. Our findings reinforce the importance of targeted fall-prevention strategies in older adults to potentially decrease the significant incidence of TBI and the associated mortality.

With respect to tICH, an increasing number of haemorrhage subtypes was associated with higher odds of 30-day mortality. Epidural haemorrhage (EDH) demonstrated the strongest association with death, which may reflect its relative rarity in older adults due to firmer dural adherence, and the greater force typically required to produce EDH [5,11]. Subdural haemorrhage (SDH) was the most frequent subtype, followed by subarachnoid haemorrhage (SAH), intraparenchymal haemorrhage (IPH), and EDH, consistent with previous studies by Powers et al. [3] and Skaansar et al. [13].

The 30-day mortality rate was 18.3% (n=134), comparable to previously reported rates of 19% (14), 16.6% (20), and 17.4% [13], but higher than the mortality reported by Scotti, et al., and Harring, et al. (6.2% and 11.3% respectively) [8,9]. This variation may reflect differences in case mix, trauma centre levels, and the mortality timepoint assessed. Pre-injury antithrombotic therapy was associated with increased odds of 30-day mortality, consistent with Scotti et al. [8] and a recent meta-analysis by Ma, et al. [1]. Antithrombotic therapy was not associated with higher odds of individual haemorrhage subtypes in our cohort; nevertheless, prior work has reported low rates of delayed intracranial haemorrhage (DIH) [21]. Together, these findings suggest that the excess mortality associated with antithrombotic therapy may not be fully explained by haemorrhage subtype distribution.

Prehospital and emergency department GCS scores were strongly associated with 30-day mortality, consistent with the findings by Ma et al. [1]. In our cohort, severe injury (GCS<8) was associated with approximately ten-fold higher odds of death compared with mild injury. This pattern was supported by the ROC analysis, in which GCS demonstrated good discriminatory performance for 30-day mortality.

Contrary to our hypothesis, polytrauma was not associated with increased 30-day mortality. This finding may relate to the exclusion of prehospital deaths and differences in injury patterns (e.g., fewer severe traffic-related injuries) or to varying definitions of polytrauma across studies [1]. We defined older adults as aged ≥65 years, consistent with common practice in TBI research [8,9,11,14,19,20].

This study has several limitations. Firstly, it is a retrospective, single-centre analysis conducted in a Swedish population, which may limit the generalizability of the findings to other healthcare settings or regions with different trauma systems. Nonetheless, the study provides contemporary data on the epidemiology and short-term outcomes of TBI in older adults in Stockholm. Further research is warranted to clarify the mechanisms underpinning morbidity and mortality in older trauma populations.

Conclusion

This study characterized the demographics of older adults with traumatic brain injury (TBI) in Stockholm, Sweden, and identified key factors associated with mortality. Consistent with prior research, low-energy mechanisms, particularly falls, were the predominant cause of TBI, and the associated mortality, in this population. Antithrombotic therapy and injury severity based on the GCS score emerged as significant mortality risk factors. These findings highlight the importance of further research on fall-prevention interventions and careful evaluation of antithrombotic management in older patients, including standardized anticoagulation reversal strategies. These results provide a valuable foundation for future large, prospective studies.

Statements and Declarations

The authors declare that they have no conflict of interest.

Ethical Approval

The study was approved by the Swedish Ethical Review Board in Stockholm, reference number Dnr 2024-03805-01.

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