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RESEARCH PAPER

Traumatic brain injury and mortality in older adults with and without pre-injury dementia

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Abstract

Background: Incidence of traumatic brain injury (TBI) is rising in older adults. Dementia is a common comorbidity and may worsen post-TBI outcomes, but its effects have not been studied.
Objective: To compare all-cause mortality following TBI or non-TBI trauma (NTT) and quantify the impacts of age, deprivation and dementia.
Design: Population-based retrospective cohort study.
Setting: Linked primary and secondary care electronic health records from the Secure Anonymised Information Linkage Databank.
Subjects: Adult residents in Wales aged 18–100 with hospitalised TBI or NTT recorded between January 2000 and December 2022.
Methods: Cox proportional hazard models were used to estimate survival within 1, 6 and 12 months of hospitalised TBI/NTT in those with and without pre-injury dementia diagnosis. Groups were propensity-matched by age, sex and morbidities. Models were stratified by age, sex and deprivation.
Results: A total of 23 428 TBIs ($n = 18\,940$) and 589 169 NTTs ($n = 421\,259$) were identified. TBIs were associated with higher mortality than NTT at all timepoints. Older age was associated with higher mortality after TBI, with 16.9% 1-month mortality in patients aged 65–79, and 31% in patients aged 80–100. In TBI patients, 30-day mortality was significant irrespective of dementia. About 6- and 12-month mortality were higher in those with than without pre-injury dementia diagnosis.
Conclusions: TBI is associated with higher all-cause mortality than NTT, particularly in older age. Among those with TBI, patients with pre-injury dementia had particularly high chronic mortality. There is an urgent need to understand the reasons for poor outcomes in older adult TBI populations, especially those with dementia.

Keywords: traumatic brain injury; trauma; falls; dementia; all-cause mortality; older people

Key Points

- In this population-based cohort study of over 500 000 hospitalised trauma cases, older adults made up a majority of patients.
- Falls were a major cause of traumatic brain injury (TBI) and non-TBI trauma across both sexes.
- Older adults with existing dementia had particularly high mortality risk in the chronic phase following TBI.

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Introduction

Traumatic brain injury (TBI) is a significant cause of death and disability worldwide [1, 2]. In high income countries, TBI incidence and mortality in younger adults is showing relative decreases amid improved public health and road safety guidance [3, 4]. In contrast, incidence has increased in older adults [5, 6]. Older-adult TBI is associated with higher short-term mortality compared to younger patients [7–10]. Chronic mortality rates (6+ months) after TBI remain understudied.

Dementia is a common age-related comorbidity linked to risk of falls [11, 12], an increasingly important cause of TBI [13–15]. Dementia may also increase risk of poor outcomes post-TBI as neurodegenerative processes, such as neuroinflammation, may exacerbate secondary injury effects of TBI [16, 17]. Existing cognitive disturbances may interfere with rehabilitation and subsequent functional recovery, such as independence in activities of daily living [18]. Additionally, people with dementia have increased susceptibility to delirium during hospitalisation, which itself is associated with poor outcomes and could hinder long-term recovery [19, 20]. While extensive research has linked TBI to later dementia risk, the impact of TBI on the vulnerable and growing population of those already living with dementia remains largely unexplored. Also, the impact of pre-existing dementia on longer-term TBI outcomes, rather than early/hospital outcomes, has not been systematically studied.

Routinely collected electronic health records (EHRs) broadly represent population-wide healthcare needs, enabling comprehensive observation of real-world healthcare outcomes. Whilst there are limitations associated with using data collected during care rather than for planned research, the large sample minimises selection bias [21], with higher representation from older or socio-economically deprived individuals who may be disproportionately impacted but typically have limited participation and retention in prospective cohort studies [22–25]. Second, EHRs facilitate longer follow-up, providing a much-needed view of post-TBI outcomes into the chronic phase. Third, the scale of population-based data enables detailed statistical matching, limiting confounding.

We examine all-cause mortality after hospitalised TBI, in older adults with and without existing diagnosed dementia, within the Welsh population [26]. Using primary and secondary care EHRs spanning 23 years, we compare the impacts of TBI and non-TBI trauma (NTT) across propensity-matched groups. We test the hypotheses that TBI is associated with higher mortality at 1, 6 and 12-months post-injury, an effect increased by older age. We then examine the impact of TBI in those with pre-existing dementia diagnosis, hypothesising that those with pre-injury dementia have higher mortality after TBI than those without.

Methods

Study design

This retrospective cohort study uses EHRs from the Secure Anonymised Information Linkage (SAIL) Databank [27] in Wales.

Data sources

Datasets used included: Death data from the Annual District Death Extract; hospital admissions from the Patient Episode Dataset for Wales; demographics from the Welsh Demographic Dataset; General Practice (GP) consultations from the Welsh Longitudinal General Practice Dataset. The project protocol and data access were approved by the SAIL Information Governance Review Panel. Patient consent was not required for the use of anonymised EHRs. Individual patient records were linked through an anonymised linking field.

Study population

The study population included individuals with a hospital admission relating to TBI/NTT, aged 18–100 at injury and registered with Welsh GP services from 1 January 2000 to 31 December 2022. Individuals entered the cohort upon TBI/NTT exposure and exited the study after 1, 6 and 12 months or upon death, whichever was sooner.

TBIs were identified from discharge coding using validated international classification of diseases (ICD-10) coding for intracranial injury [28]. NTTs were identified using external injury codes (Chapter XIX: S00-T98) [29], excluding intracranial and superficial injury. Codes were reviewed by a neurologist (L.L.) (Supplementary Appendix 1 and 2).

Individuals aged 40–100 were further categorised by dementia status, capturing the majority of cases along with young-onset dementias occurring between 40 and 64. This created four groups: dementia and TBI (DEM-TBI), dementia with NTT (DEM-NTT), non-dementia with TBI (ND-TBI) and non-dementia with NTT (ND-NTT). Dementia was identified in GP and hospital records using Read V2 and ICD-10 codes (Supplementary Appendix 3). Patients with any dementia diagnosis prior to TBI/NTT were assigned to the dementia group.

Demographic variables included age, sex, deprivation and location. Age was calculated by subtracting week of birth from TBI/NTT date and binned into groups (18–39, 40–64, 65–79, 80–100). Deprivation was the Welsh Index of Multiple Deprivation (WIMD) quintile, a composite ranking of multi-domain deprivation [30]. Location was available as Lower Layer Super Output Areas, encoding small geographic units mapped to rural/urban classifications [31, 32]. Discharge destination classifications are listed in Supplementary Appendix 5.

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Event clustering

TBI/NTT codes were clustered temporally to account for multiple admissions for the same injury event (e.g. the same individual presenting repeatedly for symptoms from the same injury, resulting in the repeated recording of TBI). Diagnoses recorded within 7 days were considered part of the same injury.

Mapping to mechanism

Injury mechanisms were identified using ICD-10 codes for assault, road traffic collision (RTC) or falls (Supplementary Appendix 4). Mechanisms within 7 days of the TBI/NTT were considered associated with the injury.

Statistical analysis

Python 3.11.4 [33] was used with packages pyodbc, numpy and pandas for data processing; scipy, lifelines, statsmodels and pingouin for statistical analysis; tableone for summary statistics; matplotlib for data visualisation.

Matching variables

Matching variables were age, sex and morbidities. Morbidities included psychiatric (anxiety, depression), neurodegenerative non-dementia [Parkinson's disease (PD), motor neuron disease], diabetes, cancer, respiratory (chronic obstructive pulmonary disease), cardiovascular (hypertension, ischemic heart disease, congestive heart failure), liver, renal (chronic kidney disease) and cerebrovascular conditions.

Propensity sampling and matching

Logistic regression was used to calculate propensity scores using the specified matching variables. Age was standardised, and categorical variables (sex, morbidities) were converted into dummy variables. Propensity scores were computed as the predicted log odds (logit) per individual.

The linear sum assignment algorithm was applied to find optimal matches between majority and minority group individuals. Groups were matched by minimising the sum of the absolute differences in their scores, ensuring that matched individuals had similar distributions of the specified variables.

Individuals aged 40–100 were allocated to mutually exclusive subgroups based on dementia and trauma status. The largest (majority) groups (DEM-NTT, ND-TBI, ND-NTT) were matched to the smallest (minority) group (DEM-TBI) in the following ratios: 2:1 ND-TBI, 5:1 DEM-NTT and 8:1 ND-NTT. Compared to an interaction approach, propensity-matching minimises confounding, while explicit stratification generates robust and interpretable results.

Survival analysis

Cox proportional hazards models were applied to estimate survival probability across the matched groups, with the exposure being TBI/NTT. Events were right-censored, with TBI/NTT as the lower limit and censorship occurring upon survival past the follow-up period (1, 6, 12 months). Age, sex and WIMD were used as covariates. The model was then repeated using trauma and dementia as the exposure.

Results

Cohort description

From 4 001 332 adults living in Wales between January 2000 and December 2022 (Figure 1), we identified 23 428 TBI admissions ($n = 18\,940$) and 589 169 NTT admissions ($n = 421\,259$) (Table 1, Figure 2a and b). The most common TBI/NTT diagnoses were traumatic subdural haemorrhage and neck of femur fracture, respectively (Supplementary Appendix 6 and 7).

Cohort characteristics are presented in Table 1. There were more TBIs in younger males (18–39); however, numbers were comparable between sexes in older age (Figure 2b). The majority of younger-age NTTs were in males, a pattern reversing in older age (Figure 2a). There were more female NTTs in older (65+) than younger (18–64) individuals [$x^2(1) = 70\,283$, $P < .0001$] (Supplementary Appendix 8 and 30).

Characteristics of the cohorts with and without dementia diagnosis are presented in Supplementary Appendix 9 and 10, 31. The dementia groups had more fall-related injuries (DEM-NTT: 69.5%; DEM-TBI: 70.1%; ND-NTT: 57.4% and ND-TBI: 58.2%).

Mechanism of injury varies with age, with falls common in older groups

Mechanisms of injury varied by age (Figure 2c and d), with falls the most common overall. This pattern was apparent from around age 55 and consistent across sexes, associated with 60% of TBIs in women and 54% in men aged 55–60. In those aged 85–100, this increased to 82% in women and 77% in men. Similar trends were observed for NTT. After matching, mechanisms were similar (Supplementary Appendix 23–26, 32).

TBI, older age and greater deprivation associated with higher mortality across all timepoints

TBI was associated with higher mortality than NTT at all timepoints [1M: hazard ratio = 3.30, 95% CI = (3.17,3.44); 6M: 2.00 (1.94,2.06); 1Y: 1.76 (1.71,1.81)] (Figure 3a–c).

Older age (65+) was associated with higher mortality following TBI and NTT, with greater impact chronically. The oldest group (80–100) was associated with the highest mortality. At 1 month, their mortality risk was 11× that of the youngest group (18–39) [11.5 (10.7,12.5)] (Figure 3a), increasing over time [6M: 23.0 (21.6,24.6); 12M: 26.9 (25.3,28.5)] (Figure 3b and c).

The 65–79 age band was associated with 5–11× the risk of mortality of the youngest group [1M: 5.63 (5.19,6.12);

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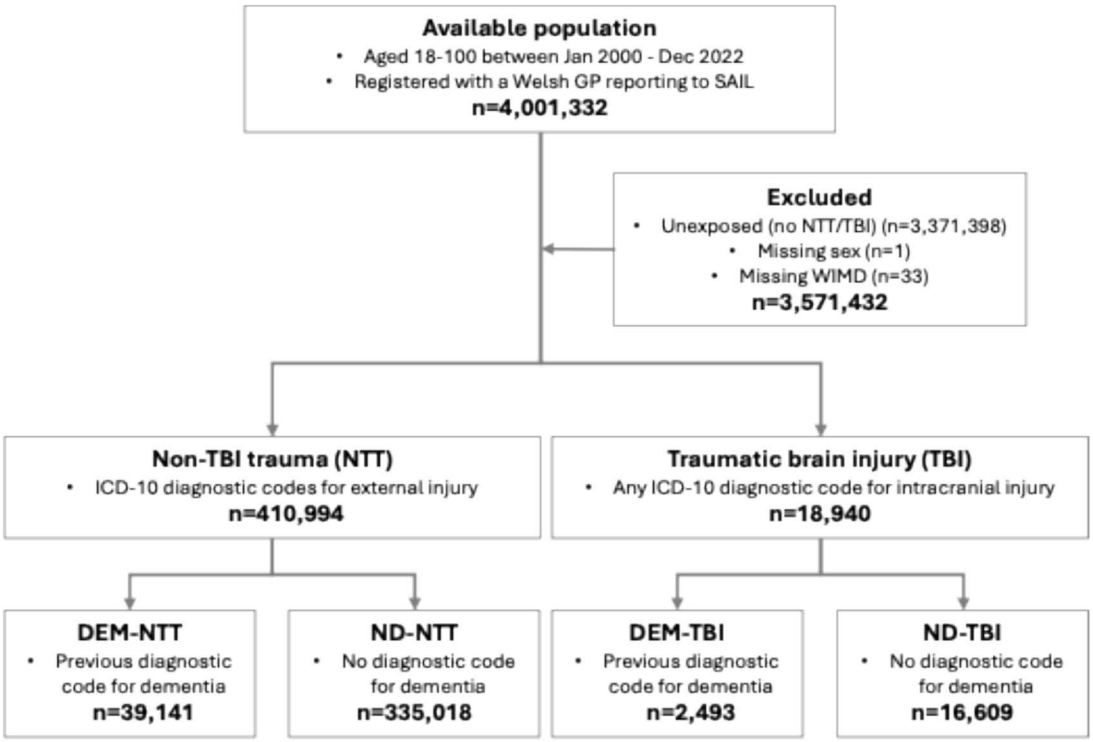


Figure 1. Flow diagram depicting total eligible population throughout the study period and individuals who were included in the subsequent analyses for the effects of injury (TBI vs NTT), followed by the effects of injury and pre-existing dementia (DEM vs ND). As the numbers for people with and without dementia are inclusive of individuals who sustained multiple injuries before and after their diagnosis of dementia, the events represented by each group (DEM-NTT, ND-NTT, DEM-TBI and ND-TBI) are unique, while some individuals are represented in multiple groups.

6M: 9.86 (9.22,10.6); 12M: 11.2 (10.5,11.9)]. The 40–64 age band was associated with 1–3× the risk [1M: 1.78 (1.63,1.96); 6M: 2.47 (2.30,2.67); 12M: 2.86 (2.68,3.07)].

Male sex and greater deprivation were associated with higher mortality. Results were significant at $P < .0001$ (Supplementary Appendix 11–16, 27–29, 33).

All-cause mortality is highest in TBI with dementia at chronic timepoints

We next quantified the impact of pre-injury dementia diagnosis. At 1 month, the TBI groups had the highest mortality irrespective of dementia [ND-TBI: 2.87 (2.65,3.12); DEM-TBI: 2.67 (2.42,2.95)] (Figure 4a).

From 6 months post-injury, mortality was highest in the DEM-TBI group [6M: 2.29 (2.14,2.44); 1Y: 2.15 (2.03,2.28)], followed by ND-TBI [6M: 1.79 (1.69,1.90); 1Y: 1.52 (1.44,1.60)] (Figure 4b and c). The DEM-NTT group was associated with higher mortality than the ND-NTT group at all timepoints [DEM-NTT: 1M: 1.28 (1.19,1.38); 6M: 1.57 (1.50,1.64); 12M: 1.59 (1.53,1.65)].

Age effects remained significant, with a three-fold difference between the oldest group (80–100) relative to 40–64 [1M: 2.94 (2.50,3.46)]; 6M: 3.73 (3.35,4.17); 12M: 3.71 (3.38,4.07)], and a two-fold difference in individuals aged 65–79 [1M: 1.84 (1.55,2.18); 6M: 2.11 (1.88,2.37); 12M: 2.06 (1.86,2.27)].

Male sex was generally associated with higher mortality. Greater deprivation was associated with higher mortality from 6 to 12 months (Supplementary Appendix 17–22, 34).

Discussion

We systematically study the impacts of older age and pre-injury dementia on mortality after hospitalised TBI. We confirm, replicating at scale, findings from the literature: TBI is associated with greater mortality than NTT, and older adults have much higher post-TBI mortality [7–9, 34–37]. In the largest population study to date, we further demonstrate high rates of post-TBI mortality in people with diagnosed comorbid dementia, effects exacerbated by older age and particularly marked at chronic timepoints.

Older adults with dementia may have worse outcomes after TBI for several reasons. The increased mortality in comorbid TBI and dementia was only apparent from 6 months, suggesting that the impact of dementia may not be through acute factors such as increased injury extent. TBI is associated with dementia risk, with shared mechanisms of chronic microglial activation and neuroinflammation that may accelerate existing neurodegenerative pathology

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Table 1. Demographics of study population (please see Methods section 2.2: study population for classifications of region, WIMD, mechanism, discharge destination and comorbidities).

		Overall	Non-TBI trauma	TBI
Episodes	–	612 597	589 169	23 428
N	–	440 199	421 259	18 940
Age, mean (SD)[a]	–	59.6 (23.8)	59.5 (23.9)	62.6 (22.7)
Sex, *n* (%)	**Female**	304 618 (49.7)	295 490 (50.2)	9128 (39.0)
	Male	307 978 (50.3)	293 678 (49.8)	14 300 (61.0)
Region, *n* (%)	**Rural**	174 584 (30.1)	167 947 (30.1)	6637 (30.0)
	Urban	404 718 (69.9)	389 212 (69.9)	15 506 (70.0)
WIMD quintile (2019), *n* (%)	**1 (most deprived)**	137 485 (22.4)	132 263 (22.5)	5222 (22.3)
	2	132 278 (21.6)	127 367 (21.6)	4911 (21.0)
	3	124 564 (20.3)	119 931 (20.4)	4633 (19.8)
	4	114 468 (18.7)	109 960 (18.7)	4508 (19.2)
	5 (least deprived)	103 752 (16.9)	99 598 (16.9)	4154 (17.7)
Mechanism of injury, *n* (%)	**Assault**	27 975 (4.6)	26 595 (4.5)	1380 (5.9)
	Fall	306 270 (50.0)	293 929 (49.9)	12 341 (52.7)
	Other/unknown	237 769 (38.8)	230 291 (39.1)	7478 (31.9)
	RTC	40 583 (6.6)	38 354 (6.5)	2229 (9.5)
Discharge destination, *n* (%)	**Death**	25 607 (4.2)	22 545 (3.8)	3062 (13.1)
	Leaving medical care	488 253 (79.8)	473 799 (80.6)	14,454 (61.8)
	Ongoing medical care	78 456 (12.8)	73 416 (12.5)	5040 (21.5)
	Other/unknown	729 (0.1)	697 (0.1)	32 (0.1)
	Social/care home	18 497 (3.0)	17 686 (3.0)	811 (3.5)
Comorbidities, *n* (%)	**Psychiatric**	140 732 (23.0)	134 525 (22.8)	6207 (26.5)
	Neurodegenerative (non-dementia)	12 199 (2.0)	11 706 (2.0)	493 (2.1)
	Diabetes	118 403 (19.3)	112 832 (19.2)	5571 (23.8)
	Cancer	179 127 (29.2)	171 504 (29.1)	7623 (32.5)
	Respiratory	68 599 (11.2)	66 152 (11.2)	2447 (10.4)
	Cardiovascular	320 832 (52.4)	307 397 (52.2)	13 435 (57.3)
	Liver	18 741 (3.1)	17 530 (3.0)	1211 (5.2)
	Renal	20 099 (3.3)	19 130 (3.2)	969 (4.1)
	Cerebrovascular	148 661 (24.3)	140 697 (23.9)	7964 (34.0)

[a]Age range reflects eligibility criteria (18–100). For sample characteristics after matching, please see Supplementary Appendix 8.

[38–40]. A TBI may set those with dementia on a trajectory of steeper functional decline, with increasing impact over time. This population also lives with significant multimorbidity and frailty [41, 42]. While our groups were matched by multimorbidity, people with dementia may experience additional challenges in coping with the same conditions [42], such as adhering to medication or rehabilitation routines [43, 44]. Dementia may also delay the identification of symptoms during illness [45], leading to inadequate management of post-TBI problems and modifying re-injury risk.

Poor outcomes such as increased dependence and mortality have been consistently observed with age [46–49]. However, previous studies have focused on acute mortality [7–10]. Here we observed higher mortality in the TBI group upon discharge, highlighting the particular vulnerability of older adults even during the hospitalisation period. Our findings are well-aligned with existing cohort studies reporting 16%–30% acute mortality in ages 65–79 [9, 47, 50, 51] and 30% in ages 80–100 [9, 50]. We extend the literature by examining chronic survival, finding that 6- and 12-month mortality were significantly higher in patients aged 80–100 than 65–79. It is important to consider the extent to which older age and dementia contribute to mortality due to health inequities. Older patients tend to experience longer delays between admission and imaging assessment, and are less likely to be transferred for neurosurgical intervention [52]. Those with cognitive impairment are also less likely to access specialised rehabilitation [53–56]. Further, frail patients may require specialised accommodations like multidisciplinary team involvement and care planning that may not be broadly available [57]. These differences may lead to a self-fulling prophecy in the care of older TBI patients, where the provision of care and potential recovery are limited as they are presumed to do poorly.

We also observed a significant association between old age and TBI occurrence, and that the male preponderance in younger-age TBI is not observed in older groups. Of note, older women also had more NTTs. This aligns with studies demonstrating a doubling of fracture incidence in older women compared to men [58], perhaps reflecting the impact of conditions like osteoporosis. Our results advocate for adequate older adult and female representation in future TBI research, as both are consistently understudied. We showed

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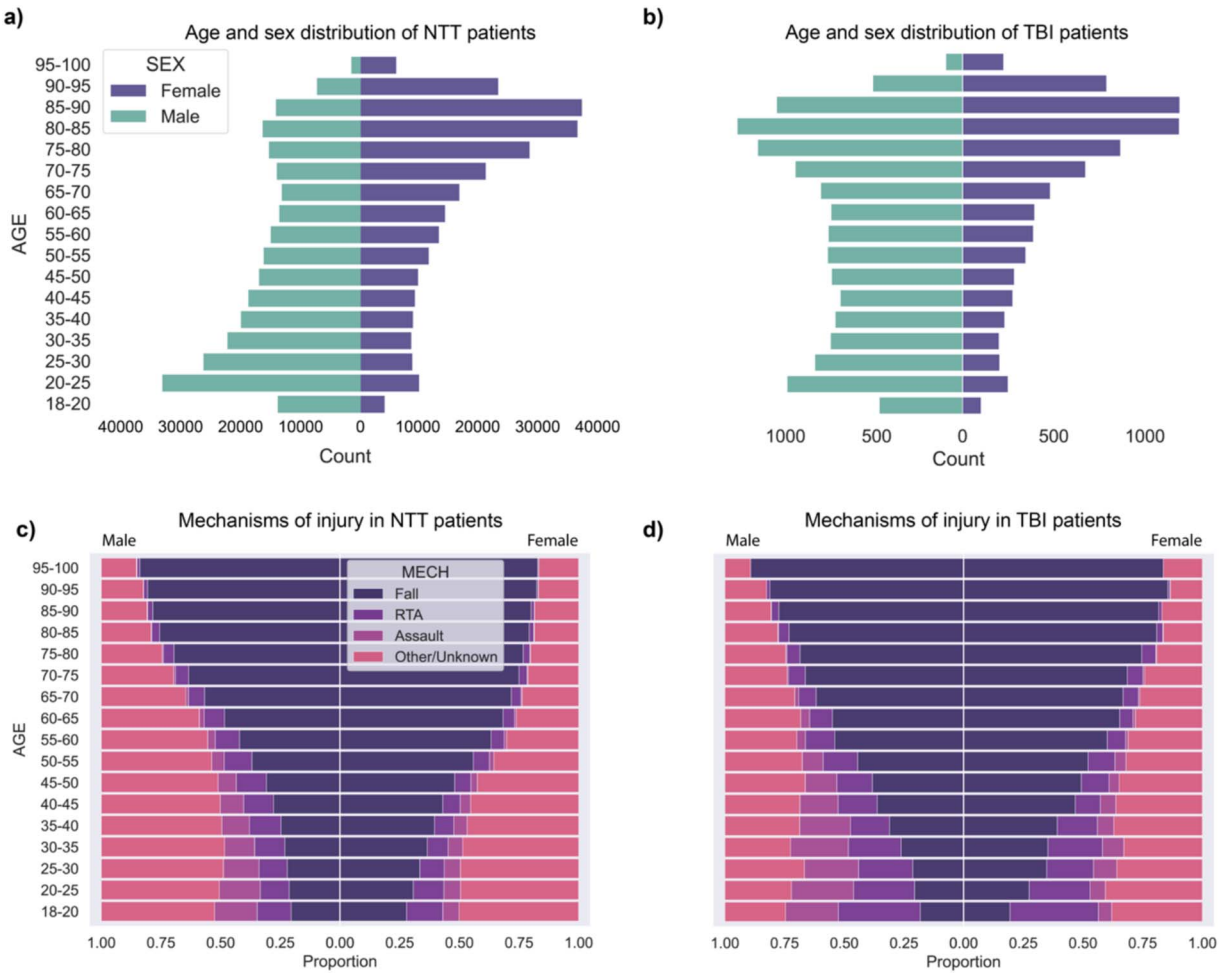


Figure 2. Demographics and injury mechanisms of NTT and TBI patients examined in this study. Males and females are mirrored across all plots, with males on the left and females on the right. (a and b) Counts of (a) NTT and (b) TBI patients. Y-axis is shared across both plots and depicts 5-year age bands, with the youngest group (18–20) at the bottom and the oldest group (95–100) at the top. X-axis depicts counts and differs across the NTT and TBI groups. (cand d) Mechanisms associated with (c) NTT and (d) TBI, expressed in proportions per sex and age band. Both axes are shared across the plots: Y-axis again depicts 5-year age bands, with the youngest at the bottom; X-axis depicts proportions of episodes associated with the following mechanisms (From centre): Falls, RTCs, assault and all other or unknown causes.

that a majority of TBIs in those aged ≥85 were due to falls. Low-energy falls are driving increased older-age TBI [59, 60]. This has implications for clinical care, as presentation with 'mild' TBI in older adults (e.g. 14–15 on the Glasgow Coma Scale [61]) should still be treated with suspicion for significant head injury [15]. Older age is now recognised as a risk factor for significant brain injury within imaging guidelines [62, 63], independently of mechanism or clinical presentation. Emerging blood-based biomarkers reflecting injury extent, e.g. glial fibrillary acidic protein [64], show promise for identifying patients likely to have significant injury, although further work is needed to establish whether current normative ranges are valid in older adults [65].

This finding also has implications for TBI prevention. Effective measures in younger patients, e.g. encouraging helmet usage for RTC-related injuries, are unlikely to substantially reduce the burden of older-age TBI. Trauma in older frail patients, or 'silver trauma', is increasingly recognised as a unique clinical condition requiring specialised treatment [66]. Amid improvements in care and life expectancy, older adults are living with greater polypharmacy and multimorbidity, both linked to frailty and fall risk [67–69]. Antidepressants, sedatives and anticholinergic medications are specifically identified as contributors [70–72]. Long-term anticoagulant use is associated with post-traumatic intracranial haemorrhage [73] and increases mortality risk [74]. Future work should investigate the impact of specific medications and explore medication rationalisation to reduce TBI burden. Falls prevention strategies including exercise programmes [75], delirium prevention [76] and comprehensive geriatric assessment should also be prioritised [77, 78].

We note several limitations to our study. Like others using EHRs, our findings depend on their data quality, population characteristics and geographical coverage. To assess

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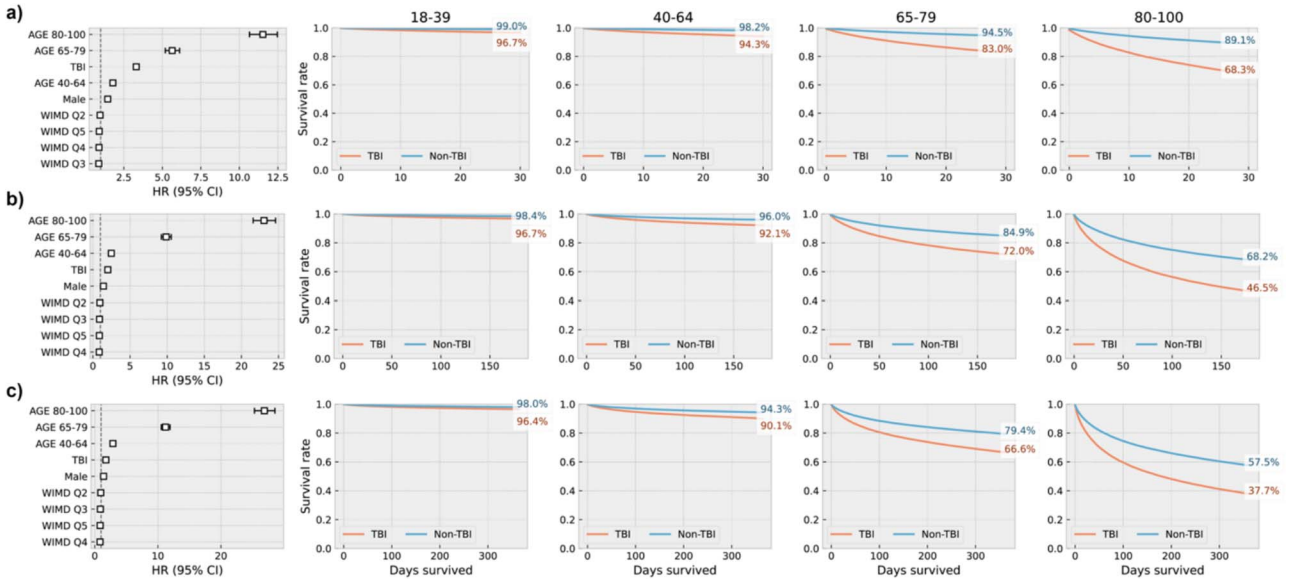


Figure 3. Survival probability at (a) 1 month, (b) 6 months and (c) 12 months across matched groups. Panels are organised as follows (left to right): hazard ratios for impact of examined factors on survival probability; survival curves across clinical groups in individuals aged 18–39; 40–64; 65–79 and 80–100. NB: comparisons for HRs as follows: age bands (80–100, 65–79 and 40–64, compared to 18–39); clinical group (NTT compared to TBI); sex (males compared to females) and deprivation [WIMD, with quintiles 2–5 compared to the most deprived quintile (Q1)].

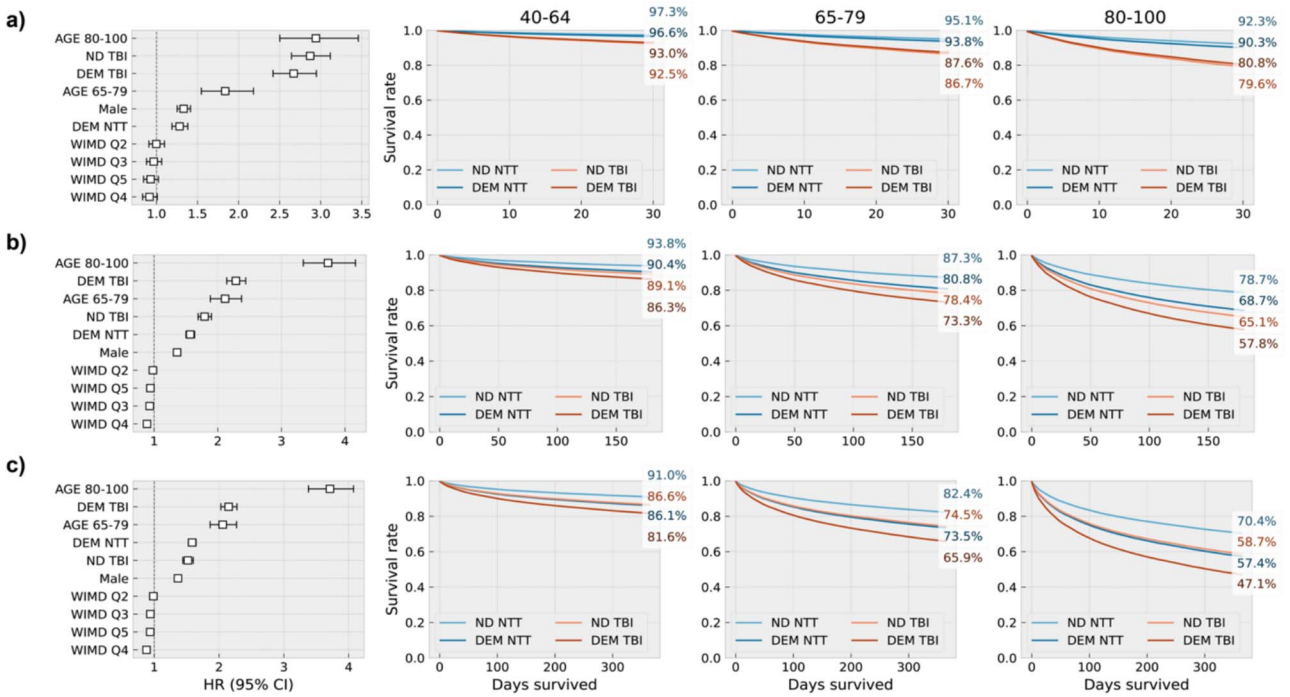


Figure 4. Cox regression results and survival curves at (a) 1 month, (b) 6 months and (c) 12 months post-injury across matched samples. Panels are organised as follows (left to right): hazard ratios for impact of examined factors on survival probability, survival curves across clinical groups in individuals aged 40–64, 65–79 and 80–100. NB: Comparisons for HRs as follows: age bands (80–100 and 65–79, compared to 40–64); clinical group (Dementia and TBI, dementia and NTT, non-dementia and TBI, all compared to non-dementia and NTT (ND-NTT); sex (males compared to females), and deprivation quintiles [WIMD, quintiles 1–5, with quintiles 2–5 compared to the most deprived quintile (Q1)].

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the replicability and generalisability of our findings, they should be reproduced using other databases with different demographic/socio-economic distributions and healthcare systems. In particular, we lacked sufficient ethnicity data to include this analysis. Next, we acknowledge the challenges in identifying TBI using EHRs [79]. While our narrow definition using intracranial injury has been validated [28], it likely captures severe cases and underestimates the true burden of TBI. For this reason, we have not presented prevalence estimates. We however note that our data aligns with expected patterns from studies investigating the increasing burden of older-age and fall-related TBI. Further, not all individuals with dementia are formally diagnosed. This may introduce bias, as diagnosed and undiagnosed individuals may have different patterns of healthcare engagement. We also lacked a severity classification for TBI or dementia, which may have independently impacted survival. Furthermore, we did not examine the impact of delirium or frailty, which are common in dementia but difficult to capture in EHRs. Given the complex interplay between frailty and dementia, assessment and inclusion of frailty measurements should be a priority for future EHRs and research in this area. Future studies should also consider the impact of age-associated factors such as dementia types. For example, older cohorts with dementia may have different dementia subtypes and clinical trajectories than younger cohorts. Next, due to changing diagnostic coding practices, we could not account for prior TBIs, focusing on spell- rather than patient-level outcomes. Additionally, we clustered hospital admissions to identify single injury events (affecting 8% of cases). However, it is possible that multiple admissions reflect underlying frailty not fully captured in our model. We also did not evaluate the isolated impact of TBI, despite the high proportion of polytrauma within this population. Despite these limitations, our study design addresses several potential biases: the inclusion of an NTT group controls for trauma burden, and individual-level matching for key confounders such as multimorbidity at the point of injury strengthens our ability to attribute our findings to the specific impact of dementia.

In this population-based study of older adults in Wales, we observed substantial all-cause mortality following hospitalised TBI within the last 20 years, particularly in people with pre-injury dementia. Further research is needed to understand contributors to this disproportionate mortality, such as biological vulnerabilities or differences in care pathways for older and comorbid patients with TBI.

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Declaration of Conflicts of Interest: None declared.

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Data Availability Statement: The SAIL Databank has an established process for applications to work with the SAIL data sources (https://saildatabank.com/).

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