

Acute Traumatic Brain Injury: Mortality in the Elderly

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■ OBJECTIVE: Despite recent progress, prognosis for the elderly (defined as aged ≥70 years) afflicted by traumatic brain injury (TBI) is unfavorable and surgical intervention remains controversial. Research during the past decade on the mortality rates or prognostic factors for survival in the elderly is limited.

■ METHODS: We analyzed 97 patients aged ≥70 years who were treated surgically for closed TBI at our neurosurgical unit between January 1, 2003 and December 31, 2012. In addition, we analyzed 22 patients aged ≥70 years who had sustained a closed TBI and on whom no neurosurgical intervention was performed. Outcome in both groups was measured as 30-, 90- and 180-day mortality.

■ RESULTS: Surgically treated patients: median age, 76 years' 30-day overall mortality rate, 36%. Higher mortality was seen with lower level of consciousness, high energy trauma, one pupil fixed and dilated, and more extensive intracranial pathology. Presence of warfarin, more advanced age, or degree of midline shift were not associated with worsened outcome. Patients not treated neurosurgically: median age. 81.5 years; 30-day overall mortality rate, 23%. Mortality for patients with Glasgow coma scale (GCS) 10–15 was 6%, GCS 6–9 67%, and GCS 3–5 100%.

■ CONCLUSIONS: Selected patients aged ≥70 years can benefit from surgical intervention for closed TBI. Level of consciousness, radiologic type of injury, mechanism of injury, and pupil abnormalities should be carefully evaluated. There also seems to exist a group of patients in whom surgical intervention offers little benefit, as mortality rate is low without surgical intervention.

INTRODUCTION

raumatic brain injury (TBI) (16) is a major contributor to death and disability globally and is, according to the Centers for Disease Control and Prevention, a contributing factor in 31% of all injury-related deaths in the United States (5). Acute intracranial hematomas are associated with TBI and estimations suggest that it occurs in 25%-45% of the patients with severe TBI (Glasgow coma scale [GCS] 3-8) and in 3%-12% in moderate TBI (GCS 9-12) (32). Historically, advances in the knowledge of the pathophysiology underlying TBI led to a number of therapeutic approaches that were introduced in the 1970s (e.g., intracranial pressure monitoring) and with incremental degrees of refinement gradually led to better survival (22). A patient category that has not reaped as much benefit from these changes in treatment is the elderly, in this article defined as aged \geq_{70} years. Apart from chronic subdural hematoma, which is a separate entity not included in this article and where the prognosis is much better (2), prognosis for the elderly have long remained unfavorable even as the outcome for younger patients improved (3, 7, 10). Age is now recognized as one of the more important factors independently contributing to the prognosis (9, 10, 18). According to Hukkelhoven et al. (10) and Mushkudiani et al. (18)., mortality rates for severe TBI increases by 37% for every decade we age, with no threshold age clearly distinguishable.

Because of this, specialist neurosurgical care has remained controversial for the older patients who have sustained trauma,

Key words

- aSDHCraniotomy
- Elderly
- Head injury
 TBI
- Trauma
- naum

Abbreviations and Acronyms

aSDH: Acute subdural hematoma CT: Computerized tomography EDH: Epidural hematoma GCS: Glasgow coma scale TBI: Traumatic brain injury TICH: Traumatic intracerebral hematoma tSAH: Traumatic subarachnoid hematoma

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who are therefore less likely to be transferred to neurosurgical units (17). However, recent studies have shown that the outlook may not be so unfavorable. Taussky et al. (26) showed that patients older than 65 years with acute subdural hematoma (aSDH) had a 30-day mortality of 32% and Brazinova et al. (3) showed that patients aged >65 years with severe TBI and GCS 3 or 4 did indeed have a poor but not hopeless prognosis with 80% I-year mortality rate.

An aging population with increasingly longer lives and advanced comorbidities, that is more susceptible to falls and other low energy head trauma, is a significant part of the yearly I million hospital admissions due to TBI in the European Union (7, 11, 14).

The aims of this study were 1) to evaluate the mortality rate for patients \geq_{70} years in a single center neurosurgical units. These patients were treated surgically for TBI with closed head injury and we investigated how previously (1, 4, 7, 9, 23, 27-31) reported clinical and radiologic factors of consequence for mortality were distributed in this population group. 2) To study a population of patients \geq_{70} years with TBI and closed head injury who was referred to the same neurosurgical unit, but where the neurosurgeon deemed surgical intervention not beneficial. The purpose of this report was to compare the mortality rate for this group to the patients who were operated on. To our knowledge, this type of comparison has not been published before.

METHODS

We retrospectively included all patients \geq_{70} years of age who had sustained a closed TBI with either aSDH, epidural hematoma (EDH), traumatic intracerebral hematoma (TICH), traumatic subarachnoid hematoma (tSAH), or a combination thereof, who received surgical treatment at the Department of Neurosurgery, Skane University Hospital Lund, Lund, Sweden. Patients with penetrating head injury and chronic subdural hematomas were excluded. Skane University Hospital Lund is a tertiary care teaching hospital and is the sole provider of neurosurgical care in the south Swedish region with a population of approximately 1,700,000 people. Patients who were treated between January 1, 2003 and December 31, 2012 were included.

We identified eligible patients by matching patients' age with surgical procedure performed according to Nordic Medico-Statistical Committee (NOMESCO) Classification of Surgical Procedure (19) from the records in the operation ward. We included AADo5 (evacuation of acute subdural hematoma), AADoo (evacuation of epidural hematoma), AAD15 (evacuation of traumatic intracerebral hematoma), AAD99 (other operation for head injury), and AAA20 (insertion of intraventricular pressure monitoring device). After identification, the patients' hospital records in Lund were scrutinized to exclude any patients' where the cause of surgery was not TBI. The hospital records were then studied for clinical and radiologic variables that have proved to be of relevance in mortality (1, 4, 7, 9, 23, 27-31): gender, age, type of injury on computerized tomography (CT) scan, mechanism of injury, presurgery pupil abnormalities, use of warfarin, presurgery reaction level scale (25), which was converted to GCS according to a previously reported algorithm (33), and the following comorbidities: cardiovascular disease, atrial fibrillation, dementia, cancer, diabetes, and alcohol abuse. Level of consciousness was grouped into GCS 3-5, GCS

6–9, and GSC 10–15. Mechanism of injury was divided into "low energy" for a fall in the same level and "high energy" for all other mechanisms of injury. In all cases, a presurgery CT scan was performed and analyzed by a neuroradiologist or a radiologist. Type of injury was divided into aSDH for patients with isolated aSDH, aSDH+ for patients with aSDH plus either TICH, tSAH, EDH, or a combination thereof, and Other that includes TICH, tSAH, and/or EDH. Degree of midline shift at the septum pellucidum was also noted from the same examination. In some cases where the latter information was absent, it was measured by the investigators. Anticoagulant effect was reversed using human prothrombin complex (Ocplex: Octapharma, Stockholm, Sweden) presurgically. Survival status 30-, 90-, and 180-days after surgery for baseline TBI was obtained from the National Census office by using national personal identification numbers.

Patients treated nonsurgically had the following inclusion criteria: aged \geq_{70} years with a closed TBI with one or more intracranial hematomas. Consultations from outside hospitals were done with transfer of CT scans, other scans of interest, clinical and laboratory status. The neurosurgeon who deemed surgical intervention unneeded was instructed to fill out a questionnaire describing the clinical situation and why the patient was not deemed eligible for neurosurgical intervention. The questionnaire contained information on the patients' age, CT-verified injury, the mechanism of injury, level of consciousness, other injuries, comorbidities, and use of warfarin. We collected this data from January I, 2013 to April 30, 2013.

RESULTS

Neurosurgically Treated Patients

Demographics. We collected data on 97 patients aged \geq 70 years with TBI who were treated surgically. Median age was 76 years (range, 70–100 years) (Table 1).

Seventy-one patients (73%) had a low energy mechanism of injury. All of the treated patients with high energy mechanism were aged between 70 and 79 years, comprising 26% of treated patients. Of the patients, 90% had I or more comorbidities and 63% had cardiovascular disease. Furthermore, 11% had diabetes, 6% had dementia, 4% had cancer, and 6% had alcohol abuse. Twenty-nine patients (30%) were on warfarin.

Fifty-two patients (53%) presented with aSDH as single injury on the CT scan; 33 (34%) and 12 (12%) presented with aSDH+ or Other, respectively. See **Tables 2** and **3** for distribution of radiologic injuries in these groups and **Figure 1** for distribution of radiologic injuries by age group.

Forty-seven patients (66%) with a low energy mechanism of injury had aSDH as single injury, 17 (24%) had aSDH+, and 7 (10%)

Table 1. Demographics		
Demographics	Surgically Treated	Not Surgically Treated
Median age (years)	76	81.5
Warfarin (%)	29	27
Fall in the same level (%)	73	86

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