Intracranial acute traumatic subdural hematoma

Acute traumatic subdural hematoma is the most common type of traumatic intracranial hematoma, occurring in 24% of patients who present comatose.

Classification

Delayed Acute Subdural Hematoma.

Infantile acute subdural hematoma.

Interhemispheric acute subdural hematoma.

Epidemiology

It is the most common type of intracranial mass lesion.

Acute subdural hematoma (ASD) is seen in 12% to 29% of severe traumatic brain injury (TBI)

Two-thirds of traumatic brain injury (TBI) patients undergoing emergency neurosurgery have an acute subdural hematoma (ASDH) evacuated.

Etiology

Acute subdural hematoma (ASDs) are caused by blood from hemorrhagic contusions and traumatic subarachnoid hemorrhage that extends to the subdural space due to tears of the arachnoid membrane. In other cases, ASDs are caused by rupture of bridging veins, which run between the surface of the brain and the skull and are especially numerous along the superior sagittal sinus. Excessive movement of the brain causes rupture of these vessels, which are attached to the skull. Individuals with brain atrophy, in whom the bridging veins are stretched and there is more room for the brain to move, are especially prone to developing subdural hematoma. Such ASDs may occur with mild or trivial head trauma. The same thing may happen in patients with hydrocephalus, if the ventricles collapse rapidly after shunting. Less commonly, subdural hematomas result from rupture of arteries that accompany bridging veins.

Significant trauma is not the only cause of subdural hematoma.

Chronic subdural hematoma can occur in the elderly after apparently insignificant head trauma.

Diagnosis
Acute laminar traumatic subdural hematoma

**Treatment**

see Acute subdural hematoma treatment.

**Outcome**

This type of head injury also is strongly associated with delayed brain damage, later demonstrated on CT scan. Such presentations portend devastating outcomes, and overall mortality rates are usually quoted at around 60%.

Patients with traumatic acute subdural hematoma were studied to determine the factors influencing outcome. Between January 1986 and August 1995, 113 patients underwent craniotomy for traumatic acute subdural hematoma. The relationship between initial clinical signs and the outcome 3 months after admission was studied retrospectively. Functional recovery was achieved in 38% of patients and the mortality was 60%. 91% of patients with a high Glasgow Coma Scale (GCS) score (9-15) and 23% of patients with a low GCS score (3-8) achieved functional recovery. All of 14 patients with a GCS score of 3 died. The mortality of patients with GCS scores of 4 and 5 was 95% to 75%, respectively. Patients over 61 years old had a mortality of 73% compared to 64% mortality for those aged 21-40 years. 97% of patients with bilateral unreactive pupil and 81% of patients with unilateral unreactive pupil died. The mortality rates of associated intracranial lesions were 91% in intracerebral hematoma, 87% in subarachnoid hemorrhage, 75% in contusion. Time from injury to surgical evacuation and type of surgical intervention did not affect mortality. Age and associated intracranial lesions were related to outcome. Severity of injury and pupillary response were the most important factors for predicting outcome ².

**Case series**
2017

Lenzi et al. present a retrospective analysis of 316 consecutive cases of post-traumatic aSDH operated on between 2003 and 2011 at our institution.

Mortality was 67% (n = 212); a useful recovery was achieved in 16.4% cases (n = 52). Age >65 years, a preoperative Glasgow coma scale (GCS) ≤ 8, specific pre-existing medical comorbidities (hypertension, heart diseases) were found to be strong indicators of unfavorable outcomes and death during hospitalization.

Our results, compared with those of the inherent literature, led the authors to question both the “aggressiveness” of neurosurgical care indications in certain subpopulations of patients being known to fare worse or even die regardless of the treatment administered and the relevance of the results concerning mortality and functional recovery reported by third authors 3).

2015

All cases of acute traumatic SDH (869) presenting over a 4-year period were reviewed. For all conservatively treated SDH, the proportion of delayed surgical intervention and the Glasgow Outcome Scale score were taken as outcome measures. Multiple factors were compared between patients who required delayed surgery and patients without surgery.

Of the 869 patients with acute traumatic SDH, 646 (74.3%) were initially treated conservatively. A good outcome was achieved in 76.7% of the patients. Only 6.5% eventually required delayed surgery, and the median delay for surgery was 9.5 days. Factors associated with deterioration were as follows:

1) thicker SDH (p < 0.001)
2) greater midline shift (p < 0.001)
3) location at the convexity (p = 0.001)
4) alcohol abuse (p = 0.0260)
5) history of falls (p = 0.018). There was no significant difference in regard to age, sex, Glasgow Coma Scale score, Injury Severity Score, abnormal coagulation, use of blood thinners, and presence of cerebral atrophy or white matter disease.

The majority of patients with SDH are treated conservatively. Of those, only 6.5% later required surgery, for raised intracranial pressure or SDH progression. Patients at risk can be identified and followed more carefully 4).

2014

A retrospective review was performed of 522 consecutive patients admitted to a single center from 2006-2012 who underwent emergent craniectomy for acute subdural hematoma. After excluding patients with unknown time of injury, penetrating trauma, concurrent cerebrovascular injury, epidural hematoma, or intraparenchymal hemorrhage greater than 30 mL, there remained 45 patients identified for analysis. Using a multiple regression model, they examined the effect of surgical timing, in addition to other variables on in-hospital mortality (primary outcome), as well as the need for tracheostomy or gastrostomy (secondary outcome). They found that increasing injury severity score (odds ratio [OR] 1.146; 95% confidence interval [CI] 1.035-1.270; p = 0.009) and age (OR1.066; 95%CI 1.006-1.129; p = 0.031) were associated with in-hospital mortality in multivariate analysis. In this
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Based on a retrospective study, the records of all patients admitted between 2001 and 2007 to a large emergency hospital with acute SDH resulting from traumatic brain injury (TBI) were analyzed. An initial Glasgow coma score (GCS), clinical state, and Glasgow outcome score (GOS) were recorded for all patients. All computer assisted tomography and MRI scans obtained from patients were saved on an electronic storage device and were reviewed by a neurosurgeon and a neuroradiologist. The coagulation parameters were analyzed for all patients. Coagulopathy was defined as international normalized ratio more than 1.2 or partial thromboplastin time more than 37s. One hundred and five women and 214 men aged between 1 and 100 years (mean 59 years) were included in the study. Patients with coagulopathy had a significantly worse outcome. Almost twice as many patients died in the coagulopathy group (mean GOS 3.10 ± 1.46) than in the group without coagulopathy (mean GOS 2.16 ± 1.45), (P < 0.001). In-hospital mortality is twice as frequent in patients with coagulopathy with traumatic SDH compared with noncoagulopathic patients, even if the initial severity of the TBI does not differ.

**2012**

1,427 patients had a mean age of 58 years, and most of them were male (63%). Glasgow Coma Scale (GCS) score on presentation was greater than 12 in 58%; the average Injury Severity Score (ISS) was 27.5. Mean length of stay was 9.6 days (range, 1-110), with 40% spending 2 or more days in the intensive care unit. Twenty-eight percent experienced medical complications. At discharge, 94% had GCS score of 13 or greater. Independence with expression, feeding, and locomotion at discharge was noted for 92%, 81%, and 43%, respectively. Inpatient mortality was 16% and did not differ significantly between the evacuated group (15%) and the nonevacuated group (17%).

This large cohort of patients with acute traumatic subdural hematoma demonstrated a lower mortality rate than those of previous reports, including among patients requiring surgical evacuation.

**1997**

Patients with traumatic acute subdural hematoma were studied to determine the factors influencing outcome. Between January 1986 and August 1995, 113 patients underwent craniotomy for traumatic acute subdural hematoma. The relationship between initial clinical signs and the outcome 3 months after admission was studied retrospectively. Functional recovery was achieved in 38% of patients and the mortality was 60%. 91% of patients with a high Glasgow Coma Scale (GCS) score (9-15) and 23% of patients with a low GCS score (3-8) achieved functional recovery. All of 14 patients with a GCS score of 3 died. The mortality of patients with GCS scores of 4 and 5 was 95% to 75%, respectively. Patients over 61 years old had a mortality of 73% compared to 64% mortality for those aged 21-40 years. 97% of patients with bilateral unreactive pupil and 81% of patients with unilateral unreactive pupil died. The mortality rates of associated intracranial lesions were 91% in intracerebral hematoma, 87% in subarachnoid hemorrhage, 75% in contusion. Time from injury to surgical evacuation and type of surgical intervention did not affect mortality. Age and associated intracranial lesions were related.
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**Case reports**

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