Intracranial acute traumatic subdural hematoma

- Association between cranial surgery and mortality among patients with firearm-related traumatic brain injury resulting in subdural hematoma
- Survival of a patient with an acute traumatic subdural hematoma and high-grade liver injury with associated IVC injury without surgical intervention
- Multimodal Imaging Reveals Labbe Vein Thrombosis Mimicking Subdural Hematoma: A Diagnostic Pitfall in Emergency Neuroimaging
- Rare Presentation of Moyamoya Disease with an Acute Subdural Hemorrhage from a Rare Location of Aneurysm-Related Moyamoya Disease
- Adjunctive middle meningeal artery embolization for non-acute subdural hematoma: A GRADE-assessed meta-analysis and trial sequential analysis on randomized trials
- Progression of intracranial hemorrhages in neurotrauma patients: A clinical and radiological comparison of isolated versus multiple areas of hemorrhage and associated transfusion treatment strategies
- Neurosurgical management of the acute phase of adult and pediatric traumatic brain injury: 2025 guidelines of the French Society of Neurosurgery
- Evaluating the efficacy and safety of low-molecular weight heparin as a chemoprophylactic agent in stable traumatic brain injury

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

Classification

Intracranial large acute traumatic subdural hematoma.

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 [1].

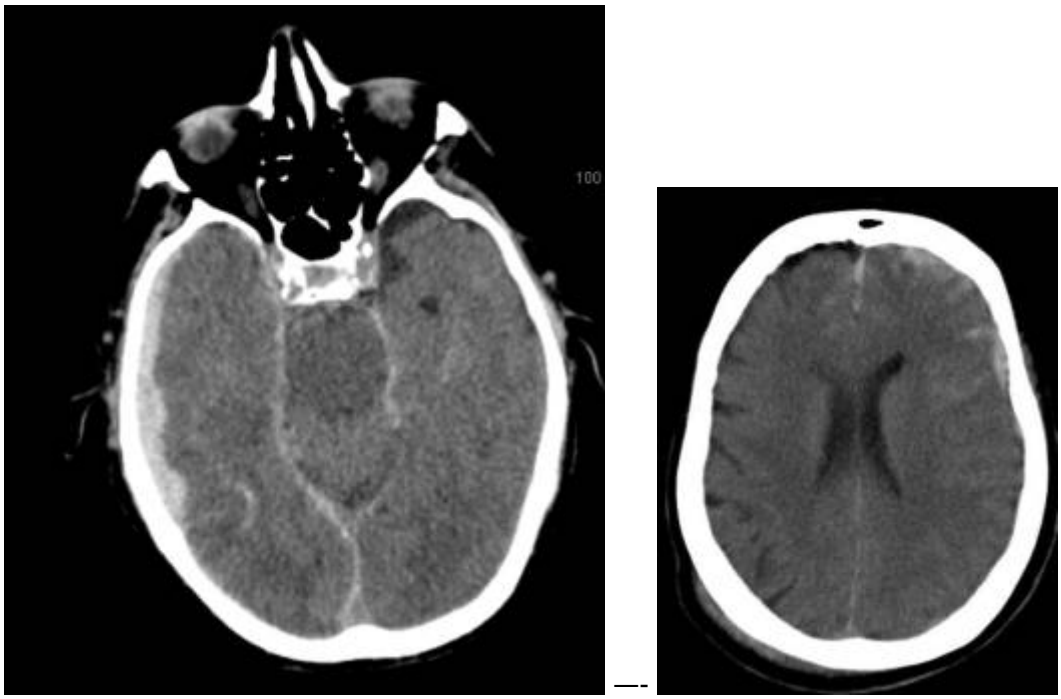
Etiology

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

Biomarkers

Brain natriuretic peptide (BNP) is a reliable biomarker in the acute phase of traumatic brain injury. However, the relationship between BNP and traumatic acute subdural hematoma (aSDH) has not yet been addressed. A study aimed to analyze BNP levels on admission in surgically treated patients and assess their relationship with early postoperative seizures (EPS) and functional outcomes.

Patients with unilateral traumatic aSDH who were surgically treated between July 2017 and May 2020 were included in the study. BNP was preoperatively measured. Patients' neurologic condition, radiographic variables on initial cranial computed tomography, sodium serum levels on admission, and occurrence of EPS were prospectively assessed. Functional outcome was assessed using the modified Rankin Scale (mRS) at discharge and follow-up (at 2-3 months). A poor outcome was defined by a mRS score > 3.

EPS occurred in 20 (19.6%) of 102 surgically treated patients in the final cohort on the median day 3. A significant association between EPS and a poor Glasgow Coma Scale score at the 7th postoperative day was found, which in turn independently predicted a poor functional outcome at discharge and follow-up. Nonetheless, EPS were not associated with poor functional outcomes. The multivariate analysis revealed BNP > 95.4 pg/ml (aOR = 5.7, p = 0.003), sodium < 137.5 mmol/l (aOR = 4.6, p = 0.009), and left-sided aSDH (aOR = 4.4, p = 0.020) as independent predictors of EPS.

In the early postoperative phase of traumatic aSDH, EPS was associated with worse neurologic conditions, which in turn independently predicted poor outcomes at discharge and follow-up. Although several EPS risk factors have already been elucidated, this study presents BNP as a novel reliable predictor of EPS. Further larger studies are needed to determine whether a more precise estimate of EPS risk using BNP levels can be reached [2)].

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 [3].

Case series

see Acute subdural hematoma case series

Case reports

Intracranial acute traumatic subdural hematoma case reports.

1)
Bullock MR, Chesnut R, Ghajar J. et al.Surgical management of acute subdural hematomas. Neurosurgery 200658S16–24.24
2)
Chihi M, Darkwah Oppong M, Quesada CM, Dinger TF, Gembruch O, Pierscianek D, Ahmadipour Y, Uerschels AK, Wrede KH, Sure U, Jabbarli R. Role of Brain Natriuretic Peptide in the Prediction of Early Postoperative Seizures Following Surgery for Traumatic Acute Subdural Hematoma: A Prospective Study. Neurol Ther. 2021 Aug 3. doi: 10.1007/s40120-021-00269-w. Epub ahead of print. PMID: 34342872.
3)
Koç RK, Akdemir H, Oktem IS, Meral M, Menkü A. Acute subdural hematoma: outcome and outcome prediction. Neurosurg Rev. 1997;20(4):239-44. PubMed PMID: 9457718.