Chronic subdural hematoma

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Chronic subdural hematoma (CSDH) is an encapsulated collection of old blood, mostly or totally liquefied and located between the dura mater and the arachnoid mater.

They are arbitrarily defined as those hematomas presenting 21 days or more after injury. These numbers are not absolute, and a more accurate classification of a subdural hematoma usually is based on imaging characteristics.

History

The first description of a chronic subdural hematoma was made in 1658 by J.J. Wepfer, followed in 1761 by Morgagni. A possible case was described by Honoré de Balzac in 1840 including its traumatic origin and surgical treatment.

Virchow, in 1857, denied a traumatic origin, and gave the name of “pachymeningitis hemorrhagica interna” to this pathology which he explained by inflammatory processes.

The traumatic etiology of chronic subdural hematoma was recognized in the XXth century, especially by Trotter in 1914. Pathophysiology was considered later on in the XXth century.

It was first described by Rudolf Ludwig Karl Virchow, in 1857, as “an internal hemorrhagic pachymeningitis” 1).

Later, in 1914, Trotter launched the theory of traumatic brain injury and the consecutive lesion of the “bridging veins”, as being the cause of what he called “hemorrhagic subdural cyst” 2).

Epidemiology

Chronic subdural hematoma epidemiology.

Etiology

see Chronic subdural hematoma etiology.

Classification

Unilateral chronic subdural hematoma.
Bilateral chronic subdural hematoma.

Isodense chronic subdural hematoma.

Bilateral isodense chronic subdural hematoma.

Ossified chronic subdural hematoma.

Septated chronic subdural hematoma.

Pathophysiology

Chronic subdural hematoma pathophysiology.

Neuropathology

Chronic subdural hematoma (CSDH) is characterized by an “old” encapsulated collection of blood and blood breakdown products between the brain and its outermost covering (the dura).

It is delimited by an outer and inner membrane. In between are blood, plasma, cerebrospinal fluid, membranes, and a mixture of inflammatory angiogenic fibrinolytic and coagulation factors. These factors maintain a self-perpetuating cycle of bleeding, lysis, and growing of neo-membranes and neo-capillaries.

Clinical Features

see Chronic subdural hematoma clinical features.

Scales

Glasgow Coma Scale.

Markwalder grading score.

Modified Rankin Scale.

Diagnosis

Its clinical symptomatology often debuting with memory and attention disorders, so that the patient is usually referred to psychiatrists or neurologists, only a paraclinical investigation (CT scan or MRI) being able to establish the diagnosis. Even the appearance of the lateral signs is subjected to many diagnosis confusions because patients deny the existence of a trauma in over 50% of the cases.
**Computed Tomography**

see Computed Tomography for chronic subdural hematoma.

**MRI**

see Chronic subdural hematoma magnetic resonance imaging.

**Biomarkers**

The association between the biomarkers of inflammation and angiogenesis, and the clinical and radiological characteristics of CSDH patients, need further investigation. The high number of biomarkers compared to the number of observations, the correlation between biomarkers, missing data and skewed distributions may limit the usefulness of classical statistical methods.

Pripp et al. explored lasso regression to assess the association between 30 biomarkers of inflammation and angiogenesis at the site of lesions, and selected clinical and radiological characteristics in a cohort of 93 patients. Lasso regression performs both variable selection and regularization to improve the predictive accuracy and interpretability of the statistical model. The results from the lasso regression showed analysis exhibited lack of robust statistical association between the biomarkers in hematoma fluid with age, gender, brain infarct, neurological deficiencies and volume of hematoma. However, there were associations between several of the biomarkers with postoperative recurrence requiring reoperation. The statistical analysis with lasso regression supported previous findings that the immunological characteristics of CSDH are local. The relationship between biomarkers, the radiological appearance of lesions and recurrence requiring reoperation have been inclusive using classical statistical methods on these data, but lasso regression revealed an association with inflammatory and angiogenic biomarkers in hematoma fluid. They suggest that lasso regression should be a recommended statistical method in research on biological processes in CSDH patients.

**Differential diagnosis**

Chronic subdural hematoma (CSDH) is a disease of the meninges and is to be distinguished from hygroma and subdural empyema.

Subdural effusion in the setting of dural metastasis is very rare and may be difficult to be distinguished from chronic subdural hematoma. Such lesions could be missed and could be the cause of recurrence in CSDH. A contrast-enhanced brain CT scan is recommended to diagnose dural metastases.

Rosai–Dorfman disease may be mistaken for a CSDH on imaging. This disease is an uncommon, benign systemic histioproliferative disease characterized by massive lymphadenopathy, particularly in the head and neck region, and is often associated with extranodal involvement. CSDH can also develop in multifocal fibrosclerosis (MFS) which is a rare disorder of unknown etiology, characterized by chronic inflammation with dense fibrosis and lymphoplasmacytic infiltration into the connective tissue of various organs. The mechanism of the formation of CSDH is presumed to involve reactive
granular membrane together with subdural collection. On the other hand, the extramedullary erythropoiesis within CSDH can be confused with metastatic malignant tumors, such as lymphoma, carcinoma, and malignant melanoma ⁶).

A 44-year old woman with gastric adenocarcinoma was presented with headache and a hypodense subdural collection in right fronto-parietal in brain CT. Burr-hole irrigation was performed with the impression of chronic subdural hematoma, but nonhemorrhagic xantochromic fluid was evacuated without malignant cell. Brain CT on the 11th day depicted fluid re-accumulation and noticeable midline shift, necessitating craniotomy and removing the affected dura.

Because the affected dura can be supposed as the main source of subdural effusion, resection of the involved dura is obligatory for the appropriate palliative management of such patients ⁷).

**Treatment**

see Chronic subdural hematoma treatment.

**Routine postoperative CT**

Routine post-operative CT brain for burr hole drainage of CSDH may be unnecessary in view of the good predictive value of pre-operative volume, and also because it is not predictive of the clinical outcome ⁸).

Scheduled postoperative cranial imaging with indwelling drains was not shown to be beneficial and misses information of intracranial damage inflicted by removal of drains. Brokinkel et al recommend CT-scanning after drainage removal ⁹).

**Complications**

see Chronic subdural hematoma surgery complications.

**Recurrence**

see Chronic subdural hematoma recurrence.

**Postoperative pneumocephalus**

see Tension pneumocephalus after chronic subdural hematoma evacuation.

**Outcome**

see Chronic subdural hematoma outcome.
Systematic Reviews

A study aimed to quantify the heterogeneity of data elements in the pre-operative, operative, and post-operative phases of care, and build the basis for the development of a set of common data elements (CDEs) for CSDH. This systematic review adhered to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement and was registered with the PROSPERO register of systematic reviews (CRD42014007266). All full-text English studies with more than 10 patients (prospective) or more than 100 patients (retrospective) published after 1990 examining clinical outcomes in CSDH were eligible for inclusion. One hundred two eligible studies were found. Only 40 studies (39.2%) reported the main presenting symptom/feature and 24 (23.5%) reported additional symptoms/features. Admitting neurological/functional status was classified by the Glasgow Coma Scale (25 studies; 24.5%), the Markwalder Score (26 studies; 25.5%) and the modified Rankin Scale (three studies; 2.9%). Fifty-four studies (52.9%) made some mention of patient comorbidities and 58 studies (56.9%) reported the proportion or excluded patients on anticoagulant medication. Eighteen studies (17.6%) reported baseline coagulation status. Sixty-four studies (62.7%) stratified or assessed severity based on radiological findings, although the methods used varied widely. There was variable reporting of surgical technique and post-operative care; 32 studies (31.4%) made no mention of whether the operations were performed under general or local anesthetic. This study, a part of the Core Outcomes and Common Data Elements in CSDH (CODE-CSDH) project, confirms and quantifies the heterogeneity of data elements collected and reported in CSDH studies to date. It establishes the basis for the consensus-based development of a set of common data elements, facilitating robust cross-study comparisons and resulting improvements in patient outcomes.

Case series

see Chronic subdural hematoma case series.

Case reports

see Chronic subdural hematoma case reports.

Experimental models

Attempts to create CSDH have been made in mice, rats, cats, dogs and monkeys. Methods include injection or surgical implantation of clotted blood or various other blood products and mixtures into the potential subdural space or the subcutaneous space. No intracranial model produced a progressively expanding CSDH. Transient hematoma expansion with liquification could be produced by subcutaneous injections in some models. Spontaneous subdural blood collections were found after creation of hydrocephalus in mice by systemic injection of the neurotoxin, 6-aminonicotinamide. The histology of the hematoma membranes in several models resembles the appearance in humans. None of the models has been replicated since its first description.

D'Abbondanza et al. did not find a report of a reproducible, well-described animal model of human CSDH.
References


