Subdural fluid collection

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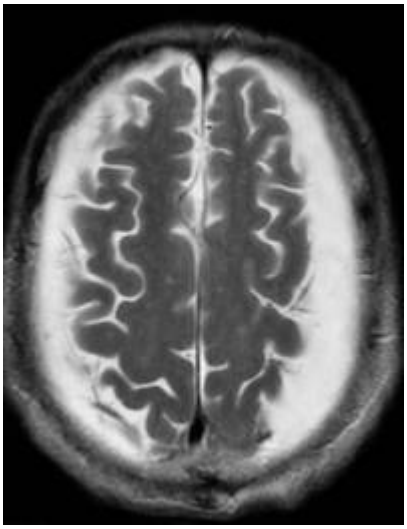

A subdural effusion is a collection of fluid trapped between the surface of the brain and the dura mater.

Broad, umbrella term that includes both:

 Subdural hygroma (CSF-only)

 Subdural effusion (CSF or CSF-like, possibly with inflammatory or xanthochromic content)

 Subdural empyema (infected/purulent fluid)

 Subacute/chronic subdural hematoma (blood breakdown products in liquid form)

Classification

Subdural fluid collections can be classified based on their characteristics, including their density on

imaging studies such as CT (computed tomography) or MRI (magnetic resonance imaging). Here are some common classifications:

Acute Subdural Hematoma (ASDH):

An accumulation of blood between the dura mater (the outermost layer of the meninges) and the arachnoid mater (the middle layer). It typically appears hyperdense on CT scans.

Subacute Subdural Hematoma:

This refers to a subdural hematoma that is in an intermediate stage between acute and chronic. The blood may show varying degrees of density on imaging, and the symptoms may evolve over days to weeks.

Chronic Subdural Hematoma (CSDH):

A collection of old blood and fluid that has accumulated gradually between the dura mater and arachnoid mater. It often appears hypodense on CT scans. Hygroma:

A subdural hygroma is a collection of clear fluid, typically cerebrospinal fluid (CSF), between the dura mater and the arachnoid mater. It is usually associated with trauma or other underlying conditions and may appear hypodense on imaging.

Empyema:

In rare cases, a subdural collection may contain pus, indicating an infection. This is referred to as a subdural empyema.

Etiology

Subdural fluid collections (SFC) are characteristic complications of shunting for idiopathic normal pressure hydrocephalus (iNPH) [1].

Subdural fluid collections following SAH can occur as a result of head trauma, external hydrocephalus, or as a treatment complication of cerebrospinal fluid shunting and craniotomies. It is critical to differentiate simple hygromas from external hydrocephalus since their response to CSF diversion is entirely different [2].

Subdural fluid collections following intraventricular and/or paraventricular procedures in pediatric neurosurgery are common and can be hard to treat [3].

Spontaneous intracranial hypotension.

Postoperative contralateral subdural effusion.

Clinical features

Bulging fontanelles in babies

Increased head circumference

Lethargy

Persistent fever

Seizures

Separated sutures in babies

Vomiting

Weakness or loss of movement on both sides of the body

Exams and Tests

CT scan of the head

Head size (circumference) measurements

MRI scan of the head

Ultrasound of the head

Differential diagnosis

see Subdural empyema.

see Subdural hematoma.

see Subdural hygroma.

see External hydrocephalus

Usually, the natural course of subdural effusion developing after intracranial surgery is self-limiting showing spontaneous resolution with passage of time leading to resolution of mass effect or very rarely may have slow progression requiring neurosurgical intervention [4) 5)].

Subdural effusion in the setting of dural metastases is very rare and may be difficult to be distinguished from chronic subdural hematoma.

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 [6].

Postoperative contralateral subdural effusion

see Postoperative contralateral subdural effusion.

Treatment

Surgery to drain the effusion is often necessary. Rarely, a permanent drainage device (shunt) is needed to drain fluid. Antibiotics may need to be given through a vein.

see subduroperitoneal shunt.

Mirone et al. describe a technique to close cortical defects by the aid of a fibrin adhesive and subsequent Ringer inflation with the aim to avoid cortical mantle collapse and to prevent the development of subdural fluid collections. They report the preliminary results of a prospective study on a consecutive series of 29 children who underwent 37 transcortical or transcallosal surgical procedures since 2008.

In 17 procedures, they performed a transcortical approach on lesions, and in other 19 operations, we operated by a transcallosal. In 5/17 transcortical approaches (29 %) and in 3/20 transcallosal approaches (15 %), they observed a 5-mm-thick subdural fluid collection of the 5 patients with subdural fluid collections in the transcortical group, 3 patients (17 %) underwent surgery for symptomatic or progressive subdural fluid collections. Of the 3 patients in the transcallosal group, a subduro peritoneal shunt was necessary only for 1 patient (5 %). At the very end of the treatment (including chemotherapy and radiotherapy), it was possible to remove the subduro-peritoneal shunt in all these patients because of disappearance of the subdural fluid collections.

In pediatric patients after transcortical or transcallosal procedures, the use of a fibrin adhesive to seal surgical opening and subsequent inflation of the residual cavity with Ringer lactate solution to avoid cortical mantle collapse seems safe and appears to prevent the development of subdural fluid collections [7].

Case series

Li et al. conducted a retrospective examination of the clinical data obtained from 124 patients who were treated for post-traumatic subdural effusion at our neurosurgery department between March 2017 and March 2021. The data collection process involved reviewing the patients' medical records, radiographic images, and follow-up visits. We used strict criteria for patient selection, including a confirmed diagnosis of post-traumatic subdural effusion, availability of follow-up data, and no prior

history of chronic subdural hematoma. Patients who experienced a progression of subdural effusion to chronic subdural hematoma were assigned to the hematoma group (26 cases). In comparison, those who did not show such progression were categorized into the effusion group (98 cases). We endeavored to identify potential risk factors contributing to the progression from subdural effusion to chronic subdural hematoma. The predictive strengths of these risk factors were evaluated using receiver operating characteristic (ROC) curves.

There were no statistically significant disparities between the two groups in terms of gender, hypertension, COPD, and GCS scores ($P > .05$). However, significant differences were noted in the variables of age, tSAH, the location of subdural effusion, and subdural effusion thickness ($P < .05$). Multivariate logistic regression analysis disclosed age (1.213), tSAH (12.542), and subdural effusion thickness (1.786) as independent risk factors for the conversion of TSE to CSDH ($P < .05$). The ROC curve showed the AUC values of age, tSAH, and subdural effusion thickness for predicting CSDH to be 0.739, 0.670, and 0.820, respectively, with a combined AUC value of 0.942, thereby outperforming the individual tests.

In patients suffering from post-traumatic subdural effusion, the thickness of the subdural effusion emerges as a strong predictor for its progression into a chronic subdural hematoma. Clinicians should be particularly cautious when the effusion thickness exceeds 10.7 mm, as the likelihood of transformation increases significantly. These findings have important implications for clinical practice and patient management, highlighting the need for prompt and effective treatment to prevent chronic complications [8)]

References

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