Subdural Hygroma (s. Subdural Effusion)

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**Subdural Hygroma** - excessive CSF collection in subdural space. [Greek *hygros* – wet]

Etiology, Pathophysiology

1. Most common cause - **cranial trauma** with arachnoid tearing and arachnoid-dura separation (→ CSF escape into subdural space) - **traumatic subdural hygroma**.
* develops in ≈ 10% severe head injuries.
* skull fractures are found in 39% cases.
* predisposing factors: *cerebral atrophy* (present in 19% hygromas), vigorous therapeutic dehydration (iatrogenic brain collapse), intracranial hypotension (e.g. in prolonged lumbar drainage), pulmonary hypertension (e.g. in chest trauma, pneumonia).
* CSF is usually xanthochromic.
* may accumulate immediately after trauma or in delayed fashion.
* most likely *locations of arachnoid tears*: sylvian fissure, chiasmatic cistern.
* **"complex hygroma"** - associated with other intracranial lesions (subdural hematoma, epidural hematoma, intracerebral hemorrhage, etc).
1. **Infection**of meninges or skull (most commonly – influenzal meningitis or mastoiditis).
2. Rupture of arachnoid at basal cistern in **communicating** **hydrocephalus**.
3. **Complication of ventricular shunting**; in patients with shunts (esp. if overdrainage occurs), disruption\* of arachnoid can lead to hygroma.

\*spontaneous or elicited by minor head trauma or previous arachnoid injury (e.g. ventricular tap, intracranial pressure sensor).

* best prevention is use of shunt alternative (third ventriculostomy) or overdrainage-limiting device.
* increasing valve opening pressure or using flow-rate-limiting system can be successful treatment.
1. **Complication** of arachnoid cyst marsupialization or resection.
2. Rare complication of spinal anesthesia causing CSF leak.

Further course

A. Spontaneous resolution of subdural collection along with cerebral expansion.

B. Hygroma progression: transudation / further CSF accumulation (flap-valve mechanism) → increasing brain dislocation → rupture of bridging veins\* → bleeding into newly formed subdural space (well documented *transformation to subdural hematoma*) → neomembrane (capsule) formation (chronic subdural hematoma).

\*stretch of draining veins by hygroma can cause multiple venous infarcts

Clinical Features

1. **Asymptomatic**
2. May increase in size (due to flap-valve mechanism, bleeding) → ***mass effect*** with significant **morbidity** similar (in character and evolution) to subdural hematoma:
3. **ICP**↑ (headaches, nausea, decreased level of consciousness)
4. **Focal signs**

Complications

1. Brain **herniation**
2. Transformation into **subdural empyema**

Diagnosis

**Neuroimaging** - crescent-shaped extraaxial collection with CSF density (hard to separate from chronic subdural hematoma!!!; H: MRI); commonly bilateral.

* **differentiation from brain atrophy**:
	+ in ***hygroma*** gyri are significantly displaced away from calvaria, occasional slight mass effect, no widening of cortical sulci (sulci even may be obliterated due to mass effect).
	+ in ***cerebral atrophy***, appearance of bilateral frontal “subdural hygromas” may be seen when patient is supine; similar finding can be seen in ***young children*** (benign enlargement of subarachnoid space - should resolve in first 2 years of life).
	+ **"*cortical vein sign*"** on gadolinium MRI - cortical veins and their branches are seen traversing widened CSF spaces over cerebral convexities - evidence of ***cerebral atrophy*** (rules out diagnosis of subdural hygroma\*).

\*hygroma displaces cortex and cortical veins → cortical veins seen only at margin of displaced cortex, and do not traverse fluid collections over cerebral convexities.

Definitive diagnosis - only by **trephine openings** in skull:

* + classically **chronic subdural hematoma** contains dark "motor oil" fluid which does not clot.
	+ if subdural fluid is *clear*, collection is termed **subdural hygroma**; hygroma fluid (i.e. CSF) contains *prealbumin* (not present in subdural hematoma) and may be under high pressure.

A. CT - left frontal subdural hygroma (9th day).

B. Enhanced density and heterogeneous appearance (53rd day) – signs of subdural bleeding into hygroma space

C. Reduction of hygroma, with probable neomembrane (117th day).

D. Resolution of subdural collection (730th day).





[Source of pictures: Marco Antonio Zanini et al. “Traumatic Subdural Hygroma”; Arq Neuropsiquiatr 2007;65(1):68-72 >>](http://www.scielo.br/pdf/anp/v65n1/a15v65n1.pdf)

A. CT - bilateral frontal subdural hygroma (12th day).

B. T1-MRI (no contrast) - laminar subdural hematoma, without compression on underlying brain (191st day).

C. T1-MRI (with contrast) - peripheral enhancement (191st day).

D. CT - disappearance of subdural collection (300th day).





[Source of pictures: Marco Antonio Zanini et al. “Traumatic Subdural Hygroma”; Arq Neuropsiquiatr 2007;65(1):68-72 >>](http://www.scielo.br/pdf/anp/v65n1/a15v65n1.pdf)

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| **1.** CT - bilateral frontoapical subdural hygroma.**2A.** MRI - bilateral frontoapical subdural hygroma, more cerebrospinal fluid than CT. D:\Viktoro\Neuroscience\TrH. Head trauma\00. Pictures\Subdural hygroma, postoperative (1).jpg**2B.** MRI - compressive brainstem deformation.**3.** MRI - brainstem morphology has returned to normal.D:\Viktoro\Neuroscience\TrH. Head trauma\00. Pictures\Subdural hygroma, postoperative (2).jpgSource of pictures: Wang Ji-sheng, Ji Nan “Compressive brainstem deformation resulting from subdural hygroma after neurosurgery: a case report” |

Gadolinium T1-MRI - diffuse, high enhancement of pachymeninges (*small black arrows*) together with bifrontal hygromas compressing frontal lobes (*black arrows*):



[Source of pictures: J. S. P. van den Berg et al. “Subdural Hygroma: A Rare Complication of Spinal Anesthesia” >>](http://www.anesthesia-analgesia.org/cgi/reprint/94/6/1625.pdf)

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| Small, low-density, extra axial collection over right frontal lobe; slight mass effect; adjacent sulci are compressed:D:\Viktoro\Neuroscience\TrH. Head trauma\00. Pictures\Subdural Hygroma (CT).jpg | Benign enlargement of subarachnoid space in child (CT) – no mass effect; normally resolves within first 2 years of life:D:\Viktoro\Neuroscience\TrH. Head trauma\00. Pictures\Benign enlargement of subarachnoid space (CT).jpg[Source of picture: Andrew L Wagner, MD, Subdural Hematoma: Multimedia >>](http://emedicine.medscape.com/article/344482-media) |

Postoperative MRI - child with large cystic craniopharyngioma and hydrocephalus; sudden tumor removal and hydrocephalus decompression resulted in subdural hygroma formation (*small arrows*); hygroma stretched draining veins, causing multiple venous infarcts (*open arrow*):



[Source of picture: Julian R. Youmans “Youmans Neurological Surgery”, 4](http://www.amazon.com/gp/product/0721682936%22%20%5Ct%20%22_blank)[th](http://www.amazon.com/gp/product/0721682936%22%20%5Ct%20%22_blank) [ed. (1997); figure 126-7; Publisher: W.B. Saunders Company; ISBN-10: 0721668453; ISBN-13: 978-0721668451 >>](http://www.amazon.com/gp/product/0721682936%22%20%5Ct%20%22_blank)

Treatment

**Asymptomatic** → **observation** (usually resolve spontaneously within several months).

N.B. observation leaves risk of transformation into subdural hematoma (which already requires **craniotomy**).

**Symptomatic** (esp. deteriorating clinical status accompanied by hygroma volume↑ with brain compression → herniation) → **surgery**: **external** **burr-hole drainage**;

* maintain subdural drain for 24-48 hrs post-op; if satisfactory resorption does not occur → shunting of subdural space.
* recurrence following simple burr-hole drainage is common; for recurrent cases:
	1. ***craniotomy*** to locate site of CSF leak (may be very difficult)
	2. subdural-peritoneal ***shunt***.

Bibliography for ch. “Head Trauma” → follow this [link >>](http://www.neurosurgeryresident.net/TrH.%20Head%20trauma%5CTrH.%20Bibliography.pdf)

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