Subdural Hygroma (s. Subdural Effusion)

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.
- subfractures 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.).

2. Infection of meninges or skull (most commonly – infected meningitis or mastoiditis).

3. Rupture of arachnoid at basal cistern in communicating hydrocephalus.

4. 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.

5. Complication of arachnoid cyst marsupialization or resection.

6. 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) → meningeal (capsule) formation (chronic subdural hematoma).

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

CLINICAL FEATURES

A. Asymptomatic

B. May increase in size (due to flap-valve mechanism, bleeding) → mass effect with significant morbidity, similar (in character and evolution) to subdural hematoma:

1. ICP* (headaches, nausea, decreased level of consciousness)

2. Focal signs

COMPLICATIONS

1. Brain herniation
2. Transformation into subdural empyema

DIAGNOSIS

Neuroimaging - crescent-shaped extracral 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 (5th day) - signs of subdural bleeding into hygroma space.
C. Reduction of hygroma, with probable nomenbrane (11th day).
D. Resolution of subdural collection (75th day).
A. CT - Bilateral frontal subdural hygroma (12th day).
B. T1-MRI (no contrast) - Laminar subdural hematoma, without compression on underlying brain (99th day).
C. T1-MRI (with contrast) - Peripheral enhancement (191st day).
D. CT - Disappearance of subdural collection (300th day).

1. CT - Bilateral frontoapical subdural hygroma.
2A. MRI - Bilateral frontoapical subdural hygroma, more intracranial fluid than CT.
2B. MRI - Compressive brainstem deformation.
3. MRI - Brainstem morphology has returned to normal.

Gadolinium T1-MRI - Diffuse high enhancement of pachymeninges (small black arrows) together with bifrontal hygromas compressing frontal lobes (black arrows).
Small, low-density, extra axial collection over right frontal lobe; slight mass effect; adjacent sulci are compressed.

Design enlargement of subarachnoid space in child (CT) – no mass effect; normally resolves within first 2 years of life.

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).

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:
  a) craniotomy to locate site of CSF leak (may be very difficult)
  b) subdural-peritoneal shunt.

BIBLIOGRAPHY for ch. “Head Trauma” → follow this LINK >>