Traumatic intracranial subdural hygroma

From the Greek hygros, meaning wet. AKA traumatic subdural effusion, AKA hydroma. Excess fluid in the subdural space (may be clear, blood tinged, or xanthochromic and under variable pressure).

Traumatic subdural hygroma (TSH) is an accumulation of cerebrospinal fluid (CSF) in the subdural space after head injury.

Epidemiology

It appears to be relatively common, but its onset time and natural history are not well defined.

Etiology

It is almost always associated with head trauma, especially alcohol-related falls or assaults [1].

Skull fractures were found in 39% of cases. Distinct from a chronic subdural hematoma, which is usually associated with an underlying cerebral contusion, and usually contains darker clots or brownish fluid (“motor oil” fluid), and may show membrane formation adjacent to the inner surface of the dura (hygromas lack membranes).

Secondary to tear in the arachnoid mater

post surgical, e.g. haematoma evacuation, ventricular drainage

see Traumatic subdural hygroma after endoscopy.

see Postoperative contralateral subdural effusion.

It has been described as a complication of cerebellopontine angle tumor requiring surgical evacuation [2].

see Subdural hygroma after cerebellopontine angle tumor surgery.

Classification

Traumatic intracranial subdural hygroma classification.

Pathogenesis

Considered a benign epiphenomenon of trauma, the pathogenesis of TSH is still unclear and many questions remain unanswered.

Tsuang et al., have found that many patients with mass effect have concomitant hydrocephalus. Patient experiencing this occurrence were studied, and the pathogenesis of this phenomenon was discussed in the context of recent advances in the understanding of CSF circulation [3].

A trivial trauma can cause a separation of the dura-arachnoid interface, which is the basic requirement for the development.

If the brain shrinks due to brain atrophy, excessive dehydration or decreased intracranial pressure, fluid collection may develop by a passive effusion. Most resolve when the brain is well expanded. However, a few become chronic subdural haematomas, when the necessary conditions persist over several weeks. Since the majority of patients do not show a mass effect, surgery is rarely required. Outcome is closely related to the primary head injury

The complexity depends on various factors including the dynamics of absorption and expansion, duration of observation, and indication and rate of surgery, besides variety of the primary head injury in types and severity [4].

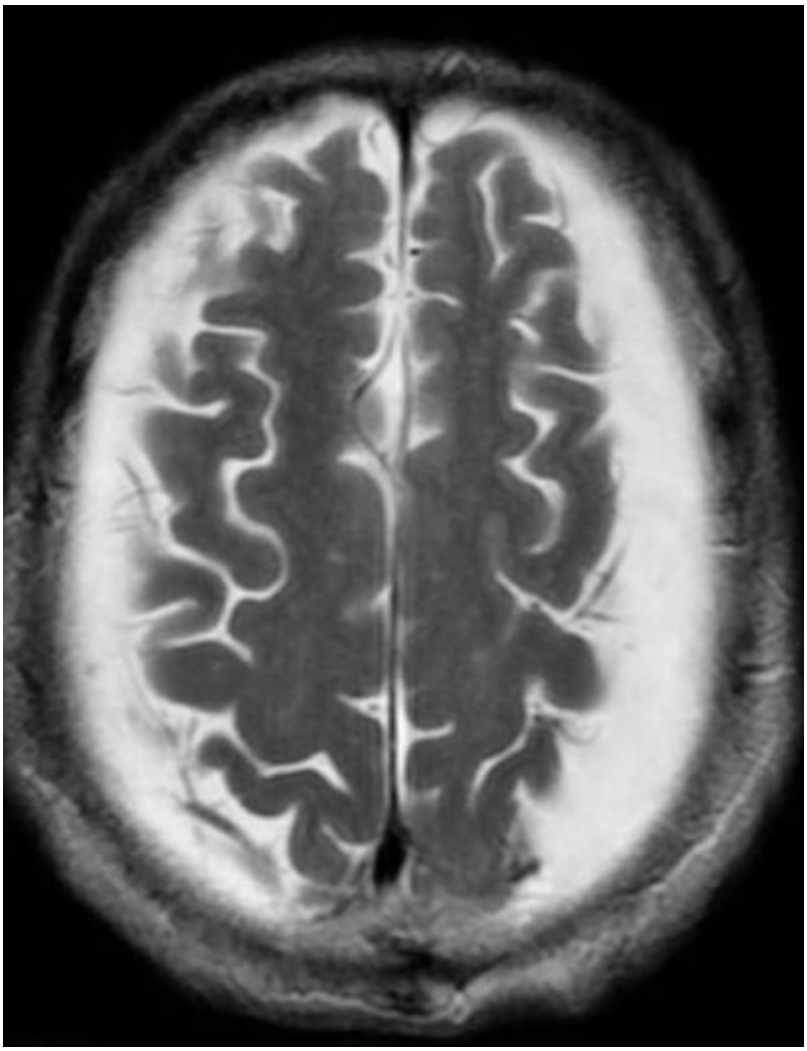
Small subdural effusion detected in the first 24 hours posttrauma evolved into TSH suggesting that this is an early lesion [5].

Pathology

They generally occur along the supratentorial cerebral convexities; occurrence in the posterior fossa is generally rare [6].

Diagnosis

CT/MRI



Crescentic CSF density/signal accumulation of CSF in subdural space that does not extend into the sulci. Vessels may cross through the lesion in contrast enhanced studies.

It is not uncommon for chronic subdural hematomas (SDHs) on CT reports for scans of the head to be misinterpreted as subdural hygromas, and viceversa. Magnetic resonance imaging (MRI) should be done to differentiate a chronic SDH from a subdural hygroma, when clinically warranted. Elderly patients with marked cerebral atrophy, and secondary widened subarachnoid CSF spaces, can also cause confusion on CT. To distinguish chronic subdural hygromas from simple brain atrophy and CSF space expansion, a gadolinium-enhanced MRI can be performed. Visualization of cortical veins traversing the collection favors a widened subarachnoid space as seen in brain atrophy, whereas subdural hygromas will displace the cortex and cortical veins.

Differential diagnosis

Imaging differential considerations include

chronic subdural haematoma: MRI may required to differentiate as they can have an identical appearance on CT

atrophy/involutional change with enlargement of the subarachnoid space.

Outcome

About half of the asymptomatic ultimately evolve into chronic subdural hematomas (CSDHs), most of which will be inevitably treated by surgical evacuation. With the emergence of subdural hydroma (SDH), rupture of bridge-veins, bleeding of the hydroma wall, hyperfunction of fibrinolysis and increasing protein content in the hydroma are some of the traditionally cited explanations of the pathogenesis evolving into CSHD.

Compared with peripheral venous blood, inflammatory cytokines were elevated in TSH and CSDH demonstrated by a number of investigators. Neoformation of capillaries, vascular hyper-permeability, serum protein exudation and other characteristics of aseptic inflammatory reaction were observed. Meanwhile, steroid was applied to treat CSDH in several groups, which was generally used as an effective anti-inflammatory agent. Based on systemic thinking, we hypothesize that TSH and CSDH are different stages, with different appearances, of the same inflammatory reaction. The evolution from TSH into CSDH and propagation of CSDH seem to be the results of local aseptic inflammation [7] [8].

It is one possible origin of chronic subdural hematoma (CSDH). The clinical characteristics of TSH evolving into CSDH include polarization of patient age and chronic small effusion. The injuries usually occur during deceleration and are accompanied by mild cerebral damage [9].

Systematic review and meta-analysis

A relationship between traumatic subdural hygroma (SDG) and chronic subdural hematoma (CSDH) has been proposed. However, the role of traumatic SDG in the development of CSDH has not been well characterized. This systematic review aimed to estimate the rate of evolution of traumatic SDG to CSDH and to identify risk factors associated with traumatic SDG evolution to CSDH. We searched MEDLINE, EMBASE, and Cochrane Library databases from inception to May 26, 2021, using the combination of the terms “subdural hygroma” and “chronic subdural hematoma.” Using a random-effects model, we calculated a pooled estimate of the rate of evolution of traumatic SDG to CSDH. In addition, we conducted a systematic review of studies of risk factors for traumatic SDG evolution to CSDH. Nineteen studies with 1,335 patients met the inclusion criteria for meta-analysis. The pooled estimate of the evolution rate was 25.0 % (95 % CI, 19.3 %-30.7 %; I2 = 85.6 %), with significant heterogeneity among studies (P < 0.01). Age ≥ 60 years was associated independently with traumatic SDG evolution to CSDH, after adjustment for study design using multivariate meta-regression. Risk factors associated with the evolution of traumatic SDG to CSDH were radiological characteristics such as thicker SDG and higher SDG CT value. The rate of traumatic SDGs' evolution to CSDH is approximately 25 %. Patients aged 60 or older with traumatic SDGs are at increased risk of CSDH development. Thicker SDG and higher SDG CT values, are commonly reported risk factors for traumatic SDG evolution to CSDH. However, higher-quality studies are needed [10].

Case reports

A previously healthy 14-month-old male who presented to the Emergency Department with vomiting after a fall and was found to have esotropia without other focal neurological deficits and a CT scan

consistent with a subdural cerebrospinal fluid collection with midline shift. The patient was treated conservatively and his symptoms resolved.

Arachnoid cyst rupture is a rare complication which can lead to increased intracranial pressure with devastating consequences. Clinical manifestation can be similar to that of other intracranial pathologies. Prompt diagnosis is required to avoid life-threatening symptoms.

Intracranial arachnoid cyst rupture should be considered when evaluating patients with non-specific neurological symptoms following trauma [11].

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