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Surgical treatment of acute subdural hygroma in children

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Abstract. During the years 1967–1984, 91 children were operated on because of acute compressive traumatic intracranial hematoma; 16 (17%) had traumatic acute subdural hygromas. These were unilateral in 12 cases and bilateral in 4. The causes of injury were traffic accidents in 11 children, a fall in 1, and acute deceleration injuries in 5. Nine children suffered multiple injuries to the thorax, inferior extremities, and pelvis. Clinical manifestations and evolution of clinical symptoms included changes in conscious level, palsy, high fever, nystagmus, maximum dilation of either pupil and spontaneous, irregular breathing. The diagnosis was made on the basis of the clinical picture and supplementary clinical investigations: CT, EEG, echoencephalograhy, isotope cisternography, and arteriography. Treatment was by simple trephination of the cranium and evacuation of hygromatous liquid. All children survived the surgical treatment; 1 child died after the operation and 2 developed hydrocephalus.

Key words: Head injury – Acute subdural hygroma – Surgical treatment.

By acute subdural hygroma we mean the accumulation of a clear or slightly blood-stained fluid in the subdural spaces. During its evolution it exerts greater or lesser compression on the brain, its vessels, and the brain stem.

The main cause of the development of traumatic, acute subdural hygroma is a severe head injury, where the moments of acceleration and deceleration are great, resulting in movement of the intracranial structures by radial forces emanating from the impact site. Various authors have reported differences in the etiopathogenesis of hygromas in children and adults. In children, the subdural hygroma (effusion) may result from congenital, traumatic, infective, metabolic, or toxic sources; in adults, however, they are exclusively traumatic in origin [4–6, 10, 12].

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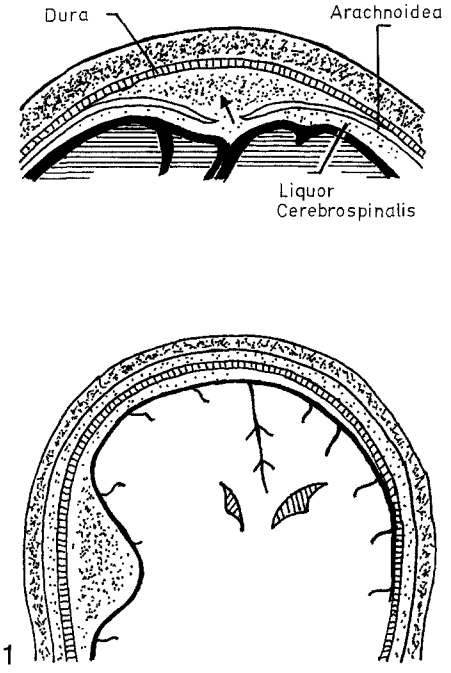
In fact, for many years hygroma formation could not be explained and its expansion in the subdural space resulted in its behaving like a compressive subdural hematoma. The name "subdural hygroma" itself went through many phases in its history. In 1857, Virchow first described intracranial subdural compression as "Pachimeningitis hemorrhagica interna," which was mentioned in 1897 by Beninghause and by Quinke [6, 12]. In 1932, Dandy first used the term "subdural hygroma," though a few years earlier Naffziger (1924, cited in [1]) had described increased amounts of cerebrospinal fluid in the subdural space. Gitlin and Hill, on the other hand, described the genesis of this subdural hygroma fluid as coming from the capillaries, a result of increased albumin in the subdural spaces. Subsequently, other etiopathogenetic mechanisms were proposed by Peet, Dandy, Dickinson, Petit-Dutaillis, Savov, and Nagoulic (cited in [1]. In the English literature today, subdural collections are also referred to as "subdural hematoma" or, as Jamieson prefers to call them "subdural fluid collections." Other authors refer to this entity as "subdural effusion" or "subdural fluid accumulations," and in contemporary German literature it is known as "traumatisches subdurales Hygrom" [6, 11, 12].

In 1975, we presented a paper concerning the surgical treatment of traumatic subdural hygroma at the 5th European Congress in Oxford. Our report was based on 28 cases, of which 4 were children, with a mortality of 32%. These cases were usually badly injured with deterioration of consciousness into coma. They suffered multiple cerebral injuries complicating fracture of the cranium. The patients who died after surgery were autopsied: almost every case suffered serious contusive damage of the brain stem or of the paraventricular region [8, 9].

Materials and methods

During the period 1967–1984, we observed and operated upon 328 cases of acute, compressive, intracranial, traumatic hematomas, of which 91 were children under 15 years old (Table 1).

In addition, 36 children with clinical manifestations and course of subdural hygroma were observed. On the basis of the evolution of the pathological process in the subdural space, they



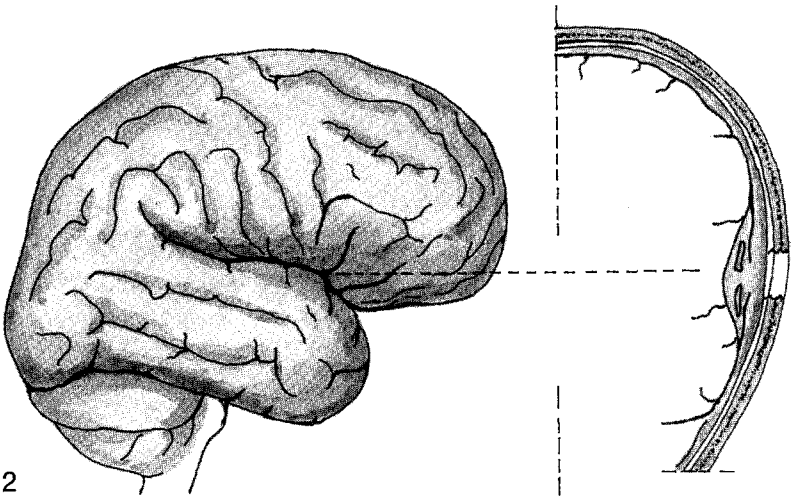


Fig. 1. Mechanism of formation of subdural acute subdural hygroma. Closing of the draining mechanism and production of brain compression, with most common localization in frontotemporal region

Fig. 2. The most common localization of acute subdural traumatic hygroma

Table 1. Localization of compressive intracranial hematomas

Location and nature	No. of children
Extradural hematoma	15
Acute subdural hematoma	32
Acute intracerebral hematoma	17
Acute subdural hygroma	16
Posterior fossa hematoma	11
(extradural, acute subdrual and intracerebellar)	11
Total	91

Table 2. Age distribution of cases

Age range	No. of children
0 to 1 month	1
2 to 5 months	2
6 to 12 months	4
2 to 3 years	2
4 to 10 years	4
11 to 15 years	3
Total	16

are divided into two groups. The first group consists of 16 children with an acute subdural hygroma, and the second group of 20 children had acute clinical signs and symptoms of the compressive syndrome, but with chronic evolution of the subdural hygroma. (The second group is similar to the chronic subdural hematoma. The diagnosis and surgical treatment are considered to resemble differentiated and undifferentiated subdural effusions of traumatic or nontraumatic origin.) All 20 children were treated with subdural-peritoneal shunts. There were no operative or postoperative deaths. After a follow-up of a few years, 3 children were reoperated upon because of blockage of the drainage system; one died of meningitis. Because of particular features in the first group of 16 children treated for acute subdural hygroma, the etiology, symptomatology, evolution, and results of surgical treatment make it mandatory for us to analyze acute subdural hygroma and consider it a significant factor in acute compressive intracranial bleeding. Of 91 cases with acute intracranial hematomas, 16 were found to have traumatic acute subdural hygroma (Table 2).

There were 11 males and 5 females. All cases suffered disturbances of consciousness: sopor, 8; coma, 7; deep coma, 1. Paresis or paralysis, convulsions, high fever, and nystagmus were found in all 16 children. Eleven children suffered pupil asymmetry.

CT was performed on 12 children, with characteristics of high density in 3 children, isodensity in 2, and low density in 7. Arteriography was performed on 8 children. Only 2 children had bilateral subdural effusions. The arteriograms showed no midline shift. Gamma cisternography was performed on 2 children and showed loculation in the subdural space with ventricular reflux and penetration of the isotope into the subdural space.

Surgical treatment

All 16 children in this series underwent surgical treatment, namely trephination in the region of the diagnosed compression. We used two surgical procedures: (1) dural suspension, a cruciate dural opening, evacuation of the subdural contents, and 1.5×1.5 cm opening of the arachnoid; (2) the second procedure had the addition of the application of small pieces of Surgicel (absorbable knitted fabric) or sponge beneath the arachnoid and along the course of the blood vessels. The subdural spaces were irrigated with saline, but drainage was not used. The blood-stained subdural fluid was either frankly or slightly hemorrhagic, this being an important indicator of prognosis. In only one case was no subdural membrane found. In all 16 children the hygroma was located in the temporal regions: 4 bilateral; 12 unilateral.

Results

All 16 children survived the surgical procedure, but 1 died after operation. This case was admitted in deep coma with

concomitant concussion of the brain stem, and petechial bleeding in the paraventricular regions. Two children developed hydrocephalus. Epileptic fits complicated 3 cases. The control CT scan showed that the brain re-expanded into the emptied subdural spaces.

Discussion

The pathogenesis of traumatic acute subdural hygromas is still neither explained satisfactorily nor universally accepted. Many authors have cited Naffziger's hypothesis of arachnoidal rupture in the Sylvian fissure and the optochiasmatic regions, thus enabling the CSF to flow into the subdural space where it remains trapped. Opponents of this hypothesis are Wycis, Pia and Wolf who have suggested the hypothesis of transudate and exudate [5, 6, 11, 12].

Our theory is that most probably the acceleration and deceleration of the brain are followed by the arachnoid, while the dura remains in contact with the bone. When the arachnoid ruptures, CSF fills the increased subdural space and the brain subsequently cannot re-expand into the same space. The CSF liquid is stained with blood, which coagulates because of the rupture of small blood vessels [8, 9].

Acute subdural effusion presses and displaces the brain to the oposite side. Clinically, this manifests itself in changes of consciousness, dilation and constriction of the pupils, dissociated eye movements, decerebration, and neurological changes in the opposite extremities. These symptoms are manifested from the 1st or 2nd day after the injury, so that one observes that the patient goes from bad to worse. This evolution of worsening is not the same picture as in acute intracranial mass-effect bleeding, where maximal dilation of one or both pupils, combined with neurological changes of the extremities, leads to rapid deterioration to coma, worsening of the vital functions, and lethal outcome if not treated surgically. The tolerance time of brain compression in subdural hygromas is longer, and there are greater chances for the use of antiedematous and anticompressive therapy, either in surgical or non-surgical ways.

Subdural hygroma diagnosis in the era before CT scanners was difficult because angiography did not always show positive findings. For example, in basal or Sylvian fissure localizations and with subdural bilateral accumulations, CT scan can reveal the hygroma in a simple and rapid way, through hypodensity, isodensity, or hyperdensity, also revealing compression and displacement signs of brain structures on the side of injury. In some cases, however, the diagnosis of acute traumatic subdural hygroma is not easy, so that surgical treatment at times is not undertaken until the 2nd or 3rd day after the injury. This decreases the quality of outcome.

Postoperative clinical evolution toward improvement or worsening of our cases led us to devise special modifications in the surgical treatment. Besides the small burr hole trephination and usual opening of dura and arachnoid, we now place a small piece of Surgicel or sponge beneath the arachnoid, thus obliterating any communication between subdural and subarachnoidal spaces.

Comparing the operative results of the series with Surgicel and sponge with the series not using them, we have observed a decrease in mortality from 54% to 32% in the whole series of 52 injured cases, who had been observed and operated upon in the course of 16 years using the two methods [7, 8, 9]. All 16 children with acute subdural hygroma were treated surgically with the placement of Surgicel and sponge beneath the arachnoid. The piece of Surgicel or sponge used prevents the adhesion of the arachnoid to the pia mater and, therefore, the communication between the subdural and subarachnoidal space stays open. The Surgicel and sponge are not toxic and do not cause either neurologial disorders or convulsions. Our results suggest that simple trephination is better than subduroperitoneal drainage for acute traumatic subdural hygroma, while in chronic subdural hygroma the opposite is true.

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