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Short Communication

Benign enlargements of subdural space (BESS) with subdural haemorrhages in infancy

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Introduction

Subdural haematomas (SDH) in infants pose a significant differential diagnostic challenge, as they can be observed prenatally, as a result of birth trauma, or spontaneously without any apparent cause – or as a result of coagulation disorders (e.g. rare platelet dysfunction [1]), metabolic disorders (glutaric aciduria type 1), congenital connective tissue disorders, vascular malformations, accidents, non-accidental trauma (e.g. in the form of shaken baby syndrome) or benign enlargement of the subarachnoid space (BESS).

The pathophysiological correlate of such subdural haemorrhages is, in most cases except for cerebral vascular malformations, the rupture of bridging veins. Bridging veins are very delicate, thin blood vessels with a length of approx. 3 to 7 cm and a diameter of 0.5 to 5.3 mm, which penetrate the dura mater and arachnoid membrane and flow into the superior sagittal sinus or other regional sinuses. The vessel walls of the bridging veins consist of three layers (endothelium on the inside, smooth muscle cells in the middle layer, and mainly collagen fibres aligned in the longitudinal direction, as well as fibroblasts and elastin in the outer layer). Medium and small bridging veins are less elastic than larger vessels because they contain fewer elastic fibres and more circularly arranged smooth muscle cells [2].

In the immediately subdural section, bridging veins may have a significantly lower wall thickness compared to the subarachnoid course, making the subdural vessel sections particularly fragile [3,4]. Light and electron microscopic examinations have shown that the subdural space is therefore the weak point in terms of the resilience of the bridging veins [4]. Tensile stresses caused by stretching appear to play a particular role in the rupture of bridging veins [2].

One of the first autopsy case reports of an acute subdural haematoma with rupture of bridging veins in infancy can be found in a case series of 102 patients from New York from 1940 to 1949. The infant was 6 months old. The cause was reported to be a fall on the head [5]. In 1946, Caffey reported on 5 infants with chronic subdural haematomas and bone fractures, which are now classified as non-accidental trauma [6]. The first anatomical description of the bridging veins is attributed to Jean Baptiste Paulin Paul Trolard (1868) [7].

The question of whether benign enlargements of the subdural space (BESS) in infancy are really just benign physiological variants – or whether BESS is associated with an increased risk of subdural haematomas (SDH) – has been debated repeatedly for several years. Clarification of this question is of fundamental importance in the differential diagnosis of subdural haematomas, as it remains unclear whether ruptures of bridging veins as a common terminal route for SDH indicate an increased incidence of external violence or whether BESS should be considered as a differential diagnosis of non-accidental injuries (such as 'shaken baby syndrome').

Method

A search of the PubMed medical database on 10 January 2026 using the keywords '(benign enlargement [Title/Abstract]) AND (subdural haematoma [Title/Abstract])' yielded eight studies, which are discussed below.

Results

1. Alshareef et al. (2022) found a total of 109 children with BEES among all children examined for macrocephaly using cMRI, with a mean age of 8 + 4.6 months, including 64 (59%) boys and 55 (50%) patients with no medical history. Subdural haematomas (SDH) were present in 11 patients, of whom only 1 SDH was described in connection with abusive head trauma. Spontaneous BESS regression was observed in 31 of the BESS patients within 33 months.

The authors therefore consider BESS to be a self-limiting pathology in infants with macrocephaly, which was observed between the 3rd and 13th month and often regressed by the 33rd month. However, BEES poses a potential risk for SDH, with SDH observed in 11/109 infants with macrocephaly and BESS, of whom only 1/11 SDH was attributable to shaken baby syndrome [8].

2. Baig et al. (2022) reported on dizygotic twins born at 34 weeks of gestation who were diagnosed with BEES using cMRI. There was no evidence of external violence. Only the first twin (male) presented with recurrent vomiting coinciding with progressive head circumference above the 97th percentile. Large bilateral collections were found in cMRI imaging, which were classified as SDH due to the detection of haemosiderin and were treated with a valveless subdural-peritoneal shunt. Two years later, both children remained neurologically stable. Head circumference was between the 98th and 99th percentiles. The authors suggest that BESS appears to follow an autosomal multifactorial inheritance pattern and promotes subdural haemorrhages [9].

3. Nasiri et al. (2021) assume that BESS is the most common cause of macrocephaly in infancy [18-22]. SDH is the most serious complication of BESS, which can occur spontaneously or after minimal trauma [22,23]. Of 32 infants with BESS (28 boys, 87.5%; 5/32 were premature, 15.6% respectively) recorded at a single centre between 2012 and 2016, SDH was recorded in two children (2/32 = 6.3% of BESS cases at this clinic). Only one of these two children with SDH showed signs of external violence. 23/32 (71.9%) of these BESS cases were associated with macrocephaly in the family. BEES was recorded at a mean age of 6.8 + 3.2 months. At 18 months of age, 83.3% (28/32) of these children still had macrocephaly. At 24 months of age, 22% (7/32) of these children had developmental delays according to the Denver Developmental Screening Test (DDST-II) [10].

4. Hansen et al. (2018) found 149 children under the age of 2 with SDH in a retrospective monocentric analysis over a period of 5 years. These were mild (N= 43) or severe SDH (N=16). BESS was present in 22.8% (34/149) of these children. Fifty percent (17/34) of children with BESS had mild SDH and 50% (17/34) had severe SDH. In 50% (17/34) of BESS cases, there were indications of violence [11].

5. Tucker et al. (2016) evaluated studies of 538 published radiologically examined cases of macrocephaly in children up to 2 years of age. Incidental subdural collections were described in 3.9% (21/538) of cases with macrocephaly. The risk of SDH was 3.68 times higher in children with BESS (OR 3.68, confidence interval 1.12-12.1, p = 0.0115), although BESS cannot be assessed as an indication of external violence from the outset [12].

6. Wittschieber et al. (2015) stated, based on older clinical studies and a study on the biomechanical properties of bridging veins from 1979 to 2008, that the risk of subdural hygromas associated with BESS was not increased. These assessments have been superseded by the more recent clinical studies mentioned above and, in particular, by current biomechanical studies [2]. Papasian and Frim (2000) showed in model experiments that stretching of the bridging veins as a result of BESS can exceed breaking points, which then lead to rupture of the bridging veins and thus to SDH [13]. Ghosh and Ghosh (2011) found bilateral SDH in a similar manner in 6/45 children under the age of 3 with 'benign external hydrocephalus' (BEH); only one child with SDH had non-accidental trauma. The authors therefore concluded that BEH is not always benign and represents a risk factor for SDH in infants [14].

7. McNeely et al. (2006) found a total of 7 patients (including 5 boys) with BESS who were diagnosed with SDH at the age of 3.6 to 17.8 months in the patient population of the Montreal Children's Hospital from 1998 to 2004, after excluding patients with shaken baby syndrome or coagulation disorders. In 5/7 cases, there was no evidence of external violence. In 2 cases, accidents preceded the injury (1 motorcycle accident with additional retinal and pre-retinal haemorrhages, 1 fall with skull fracture). Macrocephaly was present in 3/7 cases, i.e. BESS is not necessarily associated with the main symptom of macrocephaly [15].

8. Azais and Echenne (1992) reported on 41 infants with BESS who developed megacephaly in 72% of cases. 5/41 of these infants had SDH, which was associated with permanent neurological disorders in 2/5 cases, leading the authors to conclude: 'These complications call into question the benignity of this syndrome, whose long-term outcome, particularly in terms of cognitive function, is as yet unknown.' [16].

Conclusion

The available data provide a concise overview of the questions posed at the outset, encouraging a much broader systematic review, which should also examine issues relating to the biomechanics of bridging veins.

In our view, the following conclusions can be drawn from the studies presented here:

1. BESS appears to occur relatively frequently in infancy. The developmental prognosis is predominantly good and there is a tendency towards spontaneous regression.

2. BESS is a risk factor for the development of subdural haematomas in infancy, so that in the presence of subdural

haematomas, BESS should always be considered as a differential diagnosis to suspected shaken baby syndrome.

3. The detection of haemosiderin in the colliquations (SDH and/or subdural hygromas) cannot be considered pathognomonic for the presence of shaken baby syndrome, as SDH in BESS is also broken down via haemosiderin without external force.

4. BESS is not necessarily associated with megacephaly, but it is the most common cause of megacephaly in infants.

5. BESS appears to run in families in some cases. Premature babies and boys seem to be affected more frequently.

6. Current biomechanical studies indicate that deformation of the cross-section of bridging veins has a significant influence on the tearing of these sensitive and very thin vessels [2].

Summary

Ruptures of bridging veins are considered the pivotal factor in the development of subdural haematomas (SDH). However, in the presence of SDH, not only should external violence be considered, but BESS should also be taken into account as a differential diagnosis.

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Conflicts of Interest

The authors declare that there are no conflicts of interest.

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