1 Benign Enlargement of the Subarachnoid Spaces and Subdural Collections

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1.1 Introduction

Benign extracerebral collections are commonly found in infants, especially with the availability and growing use of various imaging modalities. Most of these collections, presenting as dilated subarachnoid spaces on imaging, are the most common cause of macrocephaly [1–3] in infancy. Though this disorder has been named as benign enlargement of subarachnoid space (BESS) in recent literature, there is great confusion surrounding the nature of this entity demonstrated by the various names used for its description like benign external hydrocephalus (BEH), extraventricular hydrocephalus, benign subdural effusion, benign extracellular fluid collection, extraventricular obstructive hydrocephalus, subdural hygroma, pseudo-hydrocephalus, benign extra-axial collections, subarachnomegaly, and subdural effusions of infancy [4, 5].

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The variety in nomenclature reflects the differing views on their being considered one entity and difficult neuroimaging differentiation [3]. The anatomical substrate, whether subdural fluid or cerebrospinal fluid (CSF) in the subarachnoid space, has been a subject of disagreement amongst researchers [6, 7]. This review will try to address various issues about BESS viz. clinical manifestation, incidence and progression of macrocephaly, long-term prognosis, need for shunting, association with subdural collections, relation to non-accidental injury(NAI) and finally will discuss whether all cases are truly benign. The terms BESS and BEH will be used interchangeably throughout this review.

1.2 Definition

In 1918, external hydrocephalus was first defined by Dandy as a condition with increased intracranial pressure and dilated subarachnoid spaces in infancy. He had also subclassified hydrocephalus in several ways including division into internal and external hydrocephalus [8, 9].

In current literature, external hydrocephalus is commonly defined as a large or rapidly growing head circumference in infants combined with enlarged subarachnoid spaces and moderate to no ventricular enlargement on neuroimaging [10–12]. Kumar recommended additional criteria of the absence of "clinicoradiological features of

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raised intracranial pressure," e.g., ventriculomegaly without periventricular lucency, and non-tense fontanels [4].

Macrocephaly, an essential component of BESS, is defined as head circumference more than two standard deviations above the population mean or above the 98th centile [13].

1.3 Epidemiology

A Norwegian retrospective population based study found an incidence of BESS to be 0.4 per 1000 live births [14]. In this study (86.4%) of the patients were male, and mean age at referral was 7.3 months.

A tertiary pediatric neurology center in Pennsylvania, USA, in a review of incidental findings, found that 0.6% of the children had external hydrocephalus [15]. Two thirds of these infants were found to be boys in another study [16]. BEH was associated with prematurity in many studies [10, 17].

Several studies have reported a familial predisposition in BEH [6, 18] with a 40% chance of a family member being macrocephalic while two reports have found this coherence as high as 80–90% [10, 19].

An autosomal dominant mode of transmission for the non-syndromic macrocephaly cases has been assumed by many [20–22], although a multifactorial model of inheritance with a polygenic genetic base was proposed by a 1996 study [23]. This group's findings challenged the assumption of autosomal dominant inheritance for BESS since risk of recurrence appears to be much lower than it should have been if the assumption was true. There are many genetic conditions, on the other side of the spectrum, which are associated with dilatation of subarachnoid spaces like Mucopolysaccharidoses types I, II and III, Achondroplasia, Soto's syndrome, Glutaric aciduria type I [24].

1.4 Etiology

Most cases are without a known cause and hence termed "idiopathic." However, numerous conditions such as prematurity and intraventricular hemorrhage [16, 25, 26], meningitis [25, 27], metabolic disorder [28], steroid therapy [29], chemotherapy [30], neurosurgery [31], and trauma [25, 27] may be responsible for its causation. Many premature infants can have intraventricular hemorrhage and subarachnoid hemorrhage which can go undetected due to lack of symptoms, thereby rendering the idiopathic nature of BESS uncertain in those cases [32, 33].

1.5 Pathophysiology

1.5.1 Physiological Development of the Subarachnoid Spaces

The major seat of cerebrospinal fluid (CSF) production are the four choroid plexuses in the ventricles. After production, the CSF exits into the basal cisterns, entering the subarachnoid space over the surface of the cortex [34]. The secretory epithelium of the choroid plexus is formed by 6 weeks of gestation [35]. There is uncertainty around the time CSF production begins, but circulation is established from the ventricles to the subarachnoid space by 2 months of age [36]. The separation of the arachnoid membrane from the primitive dura mater leads to the formation of the subarachnoid space. This process then spreads from the ventral portion of the mesorhombencephalon to the spinal cord caudally and to the prosencephalon cranially [37]. CSF is absorbed from the subarachnoid space into the cerebral venous system through herniations of the arachnoid membrane into the dural venous sinuses [38, 39]. Microscopic arachnoid villi are first formed by the microtubular invaginations of the subarachnoid space into the lumen of the

dural venous sinuses in utero [34]. This embryological step probably correlates with the decrease in size of the arachnoid space after 32 weeks of gestation and demonstrable on fetal magnetic resonance imaging studies [40]. Arachnoid villi can be found in the fetus and newborn, and the granulations develop between 6 and 18 months of age [41, 42].

Infants can have varying functional maturity of the arachnoid villi which can result in absorption not keeping pace with CSF production for a period of time. CSF accumulates as a result, preferentially in the subarachnoid space until skull sutures are unfused. The ventricles remain undilated till the bulk of CSF absorption occurs through the subarachnoid channels that cover the cerebral hemispheres [43]. After 2 years of age, the capacity for CSF absorption exceeds the normal rate of production by two to four times [36]. Further growth of the arachnoid villi and granulations leads to the formation of macroscopic Pacchionian bodies which are visible by 3 years of age [34]. This process of evolution of a CSF absorption system can be variable in its timeline thereby leading to the variability in the size of the subarachnoid spaces in normal children [44] and providing a physiologic basis for benign enlargement of the subarachnoid spaces.

1.5.2 Pathogenesis

There are various theories that have been proposed to explain the pathogenesis of BESS.

1. *Delayed maturation of arachnoid villi*: This is the most common theory which suggests that the defective CSF absorption due to immature arachnoid villi leads to CSF accumulation causing dilatation of the subarachnoid spaces and ventricles [43]. As the brain and skull are compliant, it does not lead to an increase in Intracranial pressure [25]. The maturation of villi around 18 months of age ends the CSF accumulation and consequent subarachnoid space dilatation.
2. *Arachnoid tear*: This leads to a one way valve mechanism causing CSF accumulation [45]. This mechanism generally leads to subdural fluid collection but can also cause localized subarachnoid space dilatation.
3. *Loculation of CSF* causing accumulation in the localized subarachnoid space [46].
4. *Subdural fluid impairing CSF absorption* [47].
5. *Communicating hydrocephalus theory*: Some believe external hydrocephalus is a step towards developing internal hydrocephalus [12]. If immature arachnoid villi are the cause, there may be restoration of CSF absorption around 18 months of age leading to resolution of BEH. However, children whose arachnoid villi are absent/grossly underdeveloped end up needing a shunt [48].
6. *Cranio-encephalic disproportion*: When the skull grows faster than the brain, it leads to transient subarachnoid space enlargement [10, 49].
7. *Dural venous sinus patency and positional plagiocephaly*: Cinalli et al. proposed that decreased patency of the venous sinuses and consequent increased venous outflow resistance contributes to the development of BEH in the first 3 years of life. The same authors found that positional plagiocephaly, found to be associated with BEH, contributed to the decreased patency of the homolateral dural sinus [50].

1.6 Clinical Manifestations

1.6.1 Macrocephaly

Hellbusch found 28 (71.8%) out 39 patients with BESS to have macrocephaly which meant that 28.2% of the cases had a normal head circumference [16]. The usual presentation of BESS is an otherwise normal infant presenting with increasing head circumference typically around 6 months of age which stabilizes around 18 months of age [51–53]. There might be marked frontal bossing as a result of the typical frontal subarachnoid space enlargement [54]. Head circumference measured after this period generally stays above but parallel to the 95th–98th centile [17, 18, 43]. On long-term follow-up, 11–87% of children with BESS end up with macrocephaly [6, 55, 56].

Most studies report no signs and symptoms of increased intracranial pressure (irritability, lethargy, vomiting, tense and bulging anterior fontanel) though they can be seen occasionally [1, 2, 4]. Rarely studies reported a tense anterior fontanel [18, 57], dilated scalp veins [58], hypotonia [59], ataxia [4], and seizures [6]. However, the sunset sign is not reported in any study [3]. The children generally achieve normal developmental milestones though mild motor delay has been reported attributable to the large head [4, 10].

1.7 Neuroimaging Findings

The classic Neuroimaging picture of BESS is that of enlarged frontal subarachnoid spaces beyond the upper limit with normal to moderately enlarged ventricles [3]. Concurrent findings include wide interhemispheric fissure, enlarged basal cisterns and third ventricle, no flattening of underlying gyri, CSF following gyral pattern and normal sulci posteriorly [4, 60, 61] (Fig. 1.1a, b).

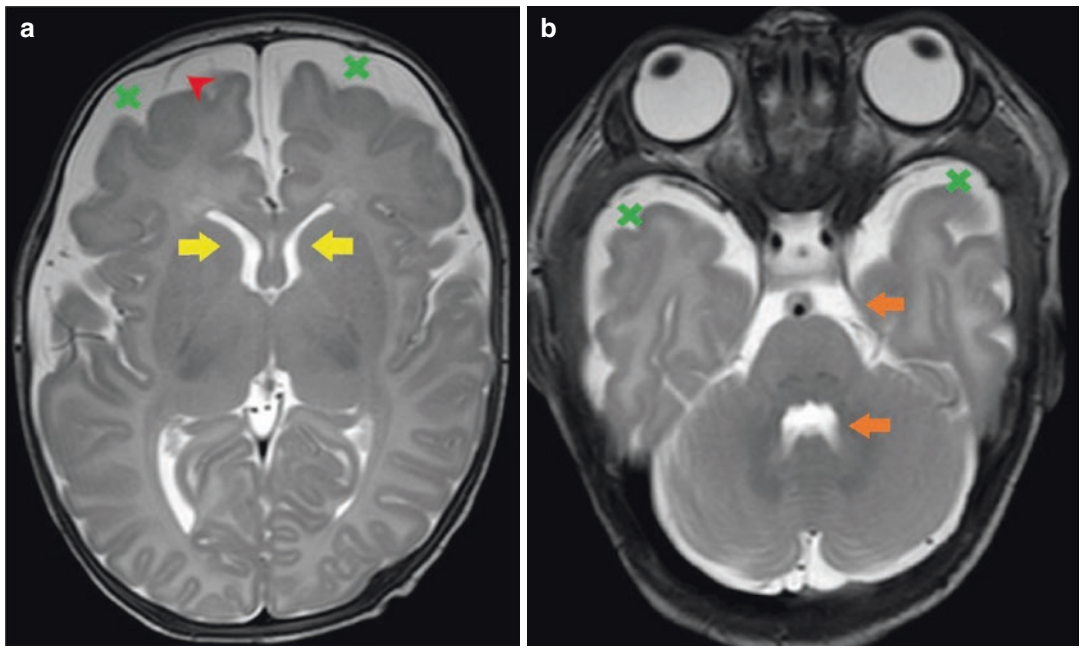

Fig. 1.1 A 1-month-old boy with incidental classical findings of enlarged subarachnoid spaces on axial T2W images. (**a**) The CSF spaces are seen following the gyral contour with no flattening of adjacent gyri. There is an increase in bifrontal subarachnoid spaces (green cross), while the lateral ventricles appear normal (yellow arrow). a distinctive feature of BESS, "cortical vein sign" is noted, comprising of elongated flow voids of bridging veins (red arrow). (**b**) In lower cuts, an enlargement of basal cisterns and fourth ventricle is noted (orange arrow) with conspicuous temporal subarachnoid spaces (green cross)

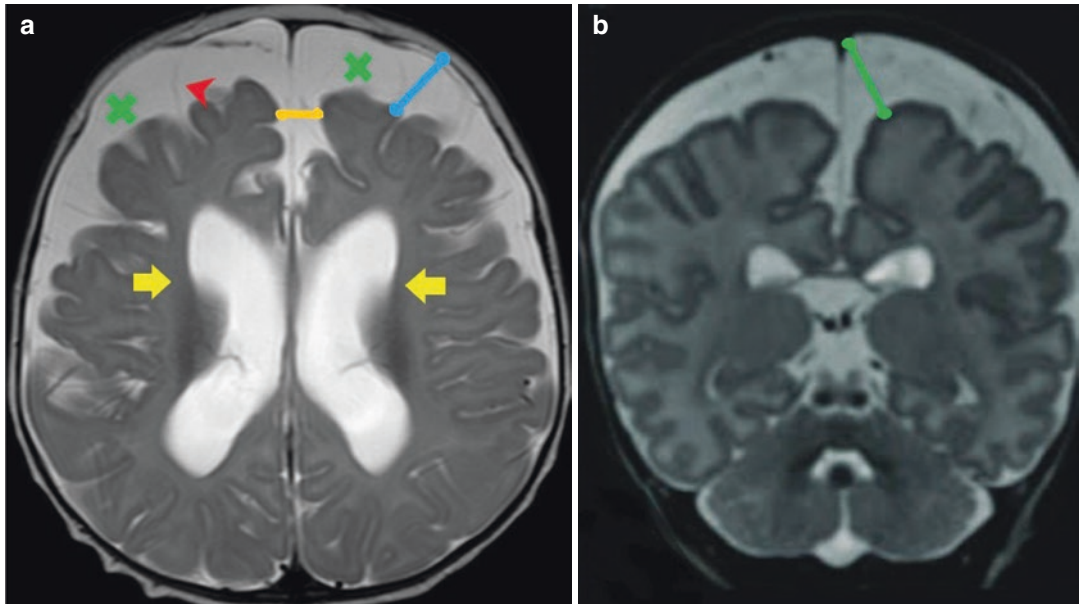

Fig. 1.2 A 5-month-old male with incidental findings of BESS. (**a**) A small variation in imaging showing some prominence of lateral ventricles (yellow arrow) and enlarged subarachnoid spaces in bilateral fronto-temporal regions (green cross), as seen on axial T2W image. The flow voids of bridging cortical veins are prominent, ratifying the "cortical vein sign" (red arrow). there is a classical increase in the interhemispheric distance (yellow line) and cortico cranial width (blue line). (**b**) A coronal T2W view showing an increased sino-cortical width (SCW) (green line)

Some degree of ventricular enlargement is reported in most studies but there are no studies with exact measurements [62, 63]. There is a possible correlation between ventricular size and width of interhemispheric fissure [64]. Maytal et al. [65] found that the sequence of enlargement in BESS was the interhemispheric fissure followed by the frontoparietal convexity subarachnoid space with basal cistern and ventricular enlargement being a late radiological finding (Figs. 1.2a, b and 1.3).

1.7.1 Normal Range [66–69]

The measurements used to quantify BESS are craniocortical width (CCW), interhemispheric fissure width (IFW) and sino-cortical width (SCW). Sinocortical width is defined as the distance from the lateral wall of the superior sagittal sinus to the cerebral cortex (Fig. 1.2a, b). There is no consensus on the cut-off values for any radiological measurement [5, 70]. The range of upper limits for the CCW is 4–10 mm (infants <1 year of age) and

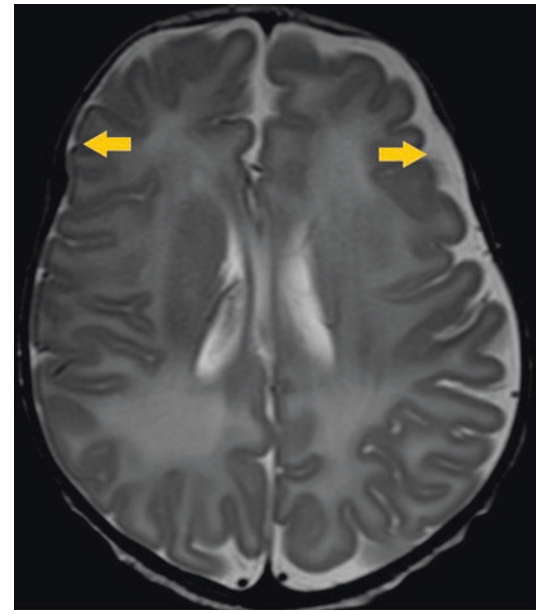

Fig. 1.3 Another child with small variation in imaging with T2W axial view showing BESS presenting as an unilateral asymmetrical enlargement of the left subarachnoid space (left frontotemporo parietal) compared to the right side (orange arrows)

3.3–5 mm (neonates). Upper limit ranges for IHW and SCW are 6–8.5 mm and 2–10 mm respectively. Tucker et al. suggested a grading system for BESS based on the depth of subarachnoid space as Grade 0 (<5 mm), Grade 1 (5–9 mm), Grade 2 (5–9 mm) and found association of incidental subdural collections with higher grades [71].

1.7.2 Imaging Modalities

1. Cranial ultrasound (US)—Often the first procedure as it is easy to perform on an open fontanel. However limited posterior fossa visualization makes it difficult to rule out important causes of obstructive hydrocephalus in a macrocephalic child [72].
2. Computerized tomography (CT)—Good visualization of neuroanatomy but risk of ionizing radiation leading to 0.07% increased lifetime risk of cancer mortality per scan [73, 74].
3. Magnetic resonance imaging (MRI): Maximizes visualization and minimizes risk of radiation. Imaging modality of choice.
4. CSF flow studies: Either by injection of an isotope or a contrast medium intrathecally. Studies have reported slow to no flow especially over cerebral convexities [18, 75].

1.7.3 Long-Term Neuroimaging Outcomes

Most studies show that the frontal subarachnoid space enlargement disappears within 2–3 years of age [4, 55]. Longest follow-up study of 19 years by Muenchberger showed that all patients eventually had a normal MRI [63].

1.8 Differential Diagnosis

There are some conditions that have to be differentiated from BESS on clinical and radiological grounds.

1. Cerebral atrophy: It does not present with increasing head circumference in contrast to BESS. Radiologically, the presence of global widening of cerebral sulci points towards atrophy as BESS typically presents with enlargement of frontal subarachnoid spaces and interhemispheric fissure [76] (Fig. 1.4a, b).
2. Subdural fluid collections: These can be differentiated from BESS by the "cortical vein sign" on MRI or US [77, 78]. A positive sign suggests that the fluid collection is caused by an enlarged subarachnoid space and not a subdural collection which would compress the subarachnoid space and the veins traversing it. On intrathecal injection of dye, the immediate influx of a contrast medium from CSF into a fluid collection suggests external hydrocephalus, whereas no influx indicates a subdural effusion [79]. Ment et al. observed that the enlargement of the basal cisterns often were seen in external hydrocephalus but not in subdural hygromas [54].
3. Convexity and Galassi I arachnoid cyst: These can sometimes masquerade as a loculated extra-axial collection like BESS or subdural effusion as it follows CSF on all sequences [80] (Fig. 1.5). This may be more widespread over convexity in rare instances of ruptured arachnoid cyst [81].

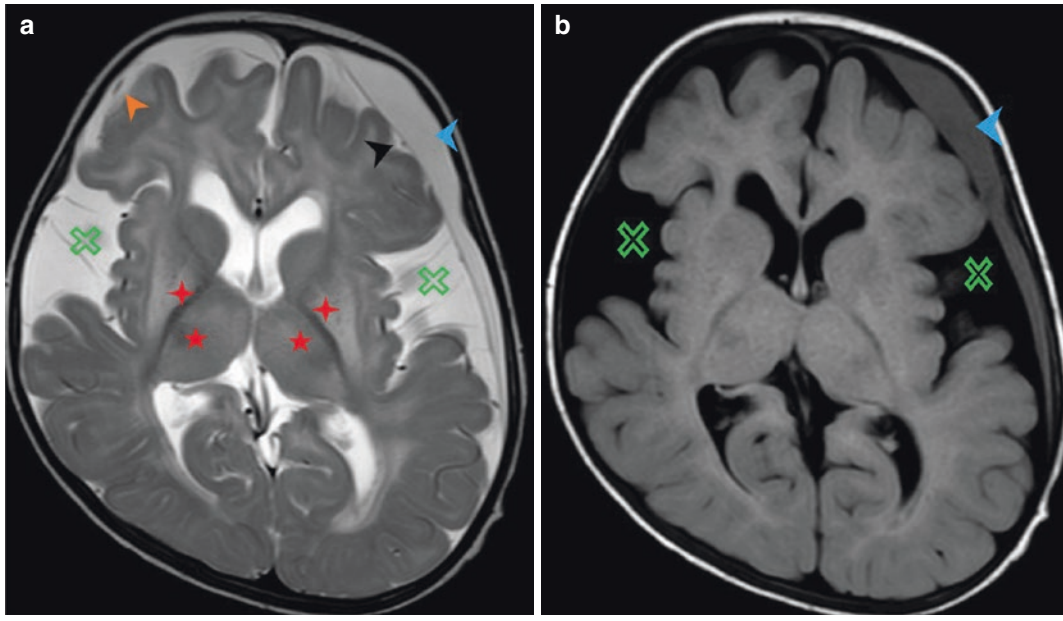

Fig. 1.4 Eight-month-old child with global developmental delay and macrocephaly. Bilateral incomplete opercularization of slyvian fissures and prominent CSF spaces in bitemporal regions, with a left subdural collection due to volume loss and signal changes in bilateral basal ganglia and thalami consistent with glutaric aciduria type 1 noted on T2W axial (**a**) and T1W axial imaging (**b**). (**a**) Cerebral atrophy with resultant temporal subarachnoid space enlargement with widening of Sylvian fissures (green cross) along with bilateral symmetrical hyperdensities in Globus Pallidus (red cross) and subthalamic nucleus (red star). There is the presence of a left subdural collection (blue arrow) showing displacement and compression of the traversing cortical veins (black arrowhead), in stark contrast to the right widened temporal and Sylvian subarachnoid spaces with prominent cortical vein flow voids (orange arrowhead). (**b**) A T1W axial image showing a thin left subdural collection due to volume loss (blue arrow) and enlargement of temporal subarachnoid spaces with widening of Sylvian fissures

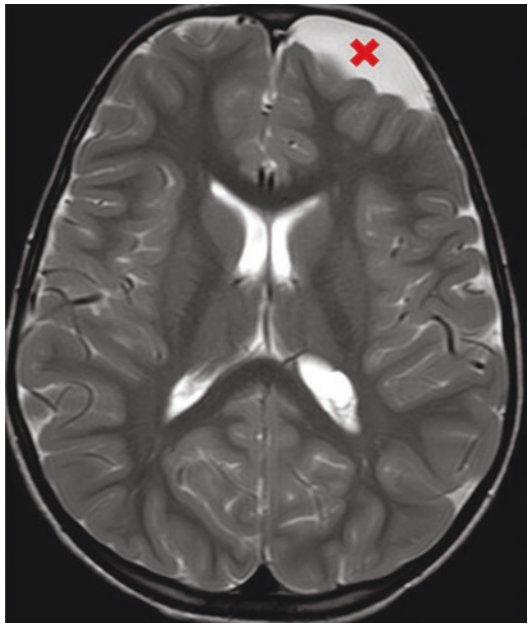

Fig. 1.5 A 3-year-old male showing a left frontal convexity arachnoid cyst on T2W axial image, which can mimic a localized subarachnoid space enlargement following the CSF spaces (red arrow)

1.9 Natural History

A developmental delay is commonly seen at some stage in infancy. Short-term outcomes have been reported by studies which generally found transient developmental delay, primarily gross motor delay and to a lesser extent delay in language development which decreased and corrected by 1–4 years [4, 58, 82]. A study by Muenchberger et al. [63] followed 15 children with BESS, nine of them had detailed neuropsychological assessment up to school with a mean final follow-up of 19 years. Though the final neurological assessment was normal and neuropsychological assessment found normal intellectual ability, several patients showed reduced performance on two tests associated with attention, and two patients with speech delay at 2 years of age performed at below-average levels in most psychological tests at long-term follow-up. Specific learning problems in reading and mathematics or a diagnosis of a psychiatric disease were found in 10 out of 15 patients and eight children had to repeat grades or go to special classes.

In another study, Laubscher et al. [58] did a long-term follow-up on 22 megalencephalic children with "dilated pericerebral subarachnoid spaces." Twelve of them were developmentally delayed). Eleven of these 12 children who had reached school age at the time that the study ended had a normal school outcome. When compared with 22 children without BESS, looking at psychomotor development and school outcome, there was no significant difference between the two groups.

1.10 Treatment

There is no Class I evidence in literature comparing treatment (medical/surgical) versus no treatment, i.e., observation. As BESS is a condition which is by definition benign, it implicitly means that it does not require any treatment and resolves with time. Therefore, observation is the only form of treatment that is required for most cases. The reported modes of treatment, when required, are surgical and medical and the indications, though varied, are generally signs and symptoms of raised intracranial pressure like bulging fontanel, irritability, vomiting accompanied by a growing head circumference. The various forms of surgical treatment reported are direct shunting or burr hole drainage/prolonged external drainage followed by shunting if necessary.

Robertson and Gomez treated two out of six patients with shunts (one lumboperitoneal and one ventriculoperitoneal) because of excessive head growth, ventricular dilatation, and other signs of increased intracranial pressure [12]. One of them was followed for 7 years and developed normally. Ten out of the 14 patients reported by Tsubokawa et al. had macrocephaly and bulging fontanels [83]. All ten underwent surgery with temporary shunt insertion. At 4–6 months after surgery, neuroimaging normalization was seen, although the ventricle enlargement seemed to retract slower. Seven of the ten children operated had a developmental quotient (DQ) of more than 100 at follow-up, indicating normal development, while two of the four non-operated patients had a DQ of less than 39.

Temporary (48 h) bilateral burr hole drainage of the frontal subarachnoid spaces in a 6-month-old girl with external hydrocephalus and developmental delay was reported by Eidlitz-Markus et al. [84]. The head circumference and psychomotor development normalized within a few months and was sustained till the last follow-up at 2 years of age. However, only modest reduction in the size of the CSF spaces was noted. Similarly, Stroobandt et al. [85] suggested treatment with external drainage of pericerebral collections for a week followed by a shunt if the effusion did not resolve.

1.11 Benign Dilatation of Subdural Spaces

Benign enlargement of subdural spaces (BESDS) has been described by many authors using various terminologies like benign subdural effusion [86], benign subdural collection [47], subdural hygroma [87], etc. This entity has been used without clear differentiation to describe clinico-radiological features identical to BESS in many reviews thus adding to the confusion regarding its existence as a separate entity [47, 86].

Many of these studies were done primarily using CT and clinical findings with or without subdural taps to diagnose subdural collections. However, with the advances in imaging, the radiological differentiation between BESS and BESDS is more distinct. There are certain differentiating radiological criteria favoring BESS over BESDS which include (1) bi-hemispheric extracerebral fluid collections: anterior > posterior, (2) widening of the anterior interhemispheric fissure, (3) enlarged subarachnoid spaces, (4) no evidence of cortical atrophy, (5) enlarged or prominent basal cisterns, (6) mild to moderate ventriculomegaly without periventricular lucency, and (7) absence of restriction of blood flow in the cortex adjacent to fluid collection on diffusion-weighted MRI (DW MRI) [87].

There are various factors contributing to its causation like non-accidental injury (NAI), minor/major traumatic injury, meningitis, encephalitis, tumor, following a VP shunt, and without any specific cause (idiopathic) [88].

Interestingly, many studies have reported that BESS can be complicated by subdural hemorrhage (SDH) either spontaneously or following accidental injury [89–92]. The proposed theory is the stretching of bridging veins in the dilated subarachnoid space [92]. There is a mathematical model of the cranial vault suggested by Papasian and Frim [89] which explains the relationship between bridging vein stretching and width of the extra-axial spaces thereby supporting the above theory. The scenario of BESS predisposing to SDH also needs to be very clearly differentiated from SDH secondary to NAI due to obvious medicolegal implications. Clinically, infants with NAI have a very morbid neurological course and risks of mortality whereas those with SDH in pre-existing BESS have a benign course [92–95]. Radiologically, absence of associated intraparenchymal contusions and presence of features of BESS help differentiating from NAI. Caution should therefore be exercised while dealing with an infant with SDH in a scenario of BESS and presumptive diagnosis of NAI should be avoided when other evidence of NAI like long bone fractures, retinal hemorrhages, etc., are absent.

In their study of 20 patients with subdural effusions following minor head injury, Kumar et al. [87] reported that 55% had macrocephaly, 25% had tense AF, 83% presented with seizures, 30% with overt neurological findings like papill-oedema, cranial nerve palsies, etc., and 70% with subtle neurological findings with irritability being the most common symptom. The infants with subtle features could mimic the features of BESS.

The various treatment options are observation, subdural needle aspirations, burr hole evacuation, subduro-peritoneal shunt (unilateral/bilateral), and craniotomy for drainage and excision of neomembranes [87]. It has been suggested that collections with thickness less than 7 mm on CT scan may have a better chance of resolving spontaneously, and hence non-operative approach may be sufficient [96]. Needle aspirations and burr hole drainage may need multiple procedures and are prone to infection. Subduroperitoneal shunts have a very high reported success rate in eliminating subdural collections between 80%

and 100% [97–99]. Unilateral shunt is usually effective in controlling bilateral collections and an unvalved shunt is used in most reports [98, 100, 101]. Subdural shunts, though, have a reported obstruction rate of up to 14% and an infection rate of 5% [98, 99, 102]. Craniotomy is needed only in complex cases.

1.12 Is Benign Enlargement of Subarachnoid Spaces Really Benign?

The usual assumption about BESS is that of an infant presenting with macrocephaly and typical neuroimaging features who has some developmental delay transiently but finally catches up with its peers. This self-limiting nature of the disorder leads to it being perceived as "benign." Many studies reporting a long-term normal outcome are based on clinical and neurological assessments.

However, studies using standardized neuropsychological tests have reported deficits in children with BEH on long-term follow-up. Alvarez et al. using the Denver Developmental Screening Test in 36 children found 14 children with delayed gross motor development, five with delayed language development, and one with global developmental delay at last follow-up at 30 months of age [10]. The same test used by Alper et al. revealed two out of 13 children with fine motor delay. The Peabody picture-vocabulary test used by the same authors showed expressive language delay in two out of seven children older than 2.5 years [6].

Zahl et al., in a retrospective population based study, reported that children and adolescents who were diagnosed with BEH during infancy generally do well. However, for some patients, there appear to be various developmental, social, and cognitive problems, and they seem to struggle more in school than their healthy peers [103]. In addition, various problems like mental retardation [58], epilepsy [104], social behavioral problems [103], autism spectrum disorders [105], and learning disabilities [58, 63] have been reported to be associated in children diagnosed with BEH on long-term follow-up.

In the light of the above evidence, it might be worth questioning the "benign" nature of this condition. However, this does not change the fact that probably most children presenting with this condition will show near resolution of imaging findings and halted progression of macrocephaly without any treatment and will achieve normal development in gross scores of assessment when done by neurologists and neurosurgeons. A more precise and specific outcome assessment by developmental pediatricians and neuropsychologists will be paramount to help establish subtle deficits and the actual impact of this disorder on the quality of life. The impact of timely intervention on long-term outcome is a subject which needs to be analyzed critically with prospective long-term studies. Prophylactic intervention in selected patients in this presumed self-limiting condition will also be a topic of interest and curiosity for future research.

1.13 Conclusion

Benign Enlargement of Subarachnoid spaces and Subdural collections are the most common forms of extracerebral collections found in infancy. Literature is abound with a variety of nomenclature describing these entities. Most cases of BESS present with typical neuroimaging findings and macrocephaly which is expected to settle down within the second year of life. The natural history of BESS favours grossly normal development though long term follow up and detailed neuropsychological tests have unveiled subtle or specific problems in various studies. Most cases do not require any treatment except few which may present with signs of raised intracranial pressure (ICP) warranting some form of surgical intervention. Subdural collections need to be differentiated from BESS. It is also essential to define the etiology of subdural collections as NAI is a significant cause and has profound medical and legal implications. Like BESS, subdural collections also warrant treatment when they present with raised ICP symptoms though many will be asymptomatic.

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