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Subdural Hematoma in Infants Without Accidental or Nonaccidental Injury: Benign External Hydrocephalus, a Risk Factor

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Abstract

Benign external hydrocephalus (BEH) is considered a self-limiting condition in infants. Subdural hematoma (SDH) in infants without a history of trauma indicates nonaccidental injury (NAI). The authors studied whether SDH can complicate BEH without apparent trauma. Out of 45 children younger than 3 years with nontraumatic SDH, 9 (7 boys) with mean age 6 months had BEH as risk factor. Symptoms included increasing head size (8), fussiness, and irritability (3). Three had up-gaze restriction, 1 axial hypotonia, and 6 normal examination. Neuroimaging showed prominent extra-axial spaces; SDH was bilateral (6), subacute (5). Other etiological workup for SDH was negative except NAI in 1. Two required evacuation of SDH and subdural–peritoneal shunt; others managed conservatively. Development was normal in 8 on follow-up. On follow-up imaging of 8, SDH completely resolved in 3, markedly reduced in 3, and remained stable in 2. BEH is a risk factor for SDH in infants, thus not always benign.

Keywords

benign external hydrocephalus, subdural hematoma, nonaccidental injury, children

Introduction

Benign external hydrocephalus (BEH) is an age-related self-limiting condition characterized by enlargement of the extra-axial fluid spaces, particularly the subarachnoid and subdural spaces. It usually occurs in infants and resolves spontaneously by about 2 to 3 years of age.[1-3] The term *benign* is coined for this condition as it has been observed that on follow-up these children attain normal developmental milestones and do not require any neurosurgical treatment.[4-8] The etiology of BEH remains poorly understood. These children are usually macrocephalic, and there may be a familial predisposition as noted by increased parental head size.[4,9] BEH has however been rarely associated with subdural hematoma (SDH) requiring operative intervention.[10,11] So the question arises, is this entity really "benign?" The incidence of SDH in children with BEH is not well studied. Nishimura et al[12] described 3 cases of SDH among 20 patients with BEH. There is also scarcity of data regarding association of BEH in children with SDH. The mechanism behind genesis of SDH in the setting of BEH is thus subject to speculation. Presumably, excess stretch on the thin bridging veins crossing the wide subarachnoid space may be one of the factors responsible.[11,13] It may thus be postulated that the children with BEH may be more prone to develop SDH either spontaneously or following trivial head trauma.[1,9,10,12]

Presence of SDH in an infant always warrants investigation to rule out nonaccidental injury (NAI).[14] In addition, bleeding diatheses, metabolic or genetic conditions leading to weak vessel wall need to be ruled out based on the appropriate clinical setting. In our practice, we observed some infants with BEH develop SDH without any other predisposing factor(s) as mentioned above. So we reviewed all children with BEH who had SDH on neuroimaging and tried to establish the hypothesis that BEH by itself predisposed these children to develop SDH.

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Table 1. Clinical Features of the children with BEH and SDH

Serial No.	Age	Gender	Presenting Symptoms	Increased Parental Head Size	Head Size at Birth	Clinical Findings
1	3 mo	Male	Fussiness and irritability, poor feeding	No	Normal	Decreased up-gaze, axial hypotonia, brisk reflexes
2	8 mo	Male	Increasing head size	Yes	NA	Normal
3	2 y	Male	Developmental delay, fussiness and irritability, seizures	No	NA	Normal
4	4 mo	Male	Increasing head size	No	Normal	Normal
5	4 mo	Female	Increasing head size	Yes	Increased	Normal
6	4 mo	Male	Increasing head size	Yes	Normal	Mild up-gaze restriction
7	3 mo	Male	Increasing head size	Yes	NA	Restricted up-gaze
8	5 mo	Female	Fussiness and irritability	No	NA	Normal
9	5 mo	Male	Increasing head size	No	Normal	Normal

Abbreviations: BEH, benign external hydrocephalus; SDH, subdural hematoma; NA, not available.

Methods

After approval from the institutional review board, all patients, 18 years old or younger who were either admitted or seen in the outpatient clinic at the Children's Hospital Cleveland Clinic, between 2000 and 2010, were identified by searching for the diagnoses of hydrocephalus (International Classification of Diseases, Ninth Revision, codes 331.3 and 331.4) and subdural hematoma, nontraumatic (International Classification of Diseases, Ninth Revision, code 432.1).

Inclusion criteria were as follows: children who had evidence of enlarged extra-axial space detected on computed tomography (CT)/magnetic resonance imaging (MRI) associated with SDH.

The exclusion criteria were as follows:

1. presence of significant ventriculomegaly as part of true hydrocephalus with increased periventricular lucency,
2. presence of ventricular shunt,
3. reported significant head injury, and
4. microcephaly with cerebral atrophy.

The charts of included patients were reviewed retrospectively and data were collected regarding patient demographics, clinical features, diagnostic workup, management, and outcome.

Results

Clinical Features

We identified 45 cases younger than 3 years in past 10 years who had nontraumatic SDH. After reviewing all those charts and applying inclusion and exclusion criteria, 9 children were identified to have association of BEH and SDH during the 10-year study period (Table 1). There were 7 boys and 2 girls, with a mean age of 6 ± 6.6 months (range 3 months to 2 years); 8 patients were younger than 1 year at presentation and only 1 presented at 2 years of age. The most common symptom was increasing head size (n = 8) followed by increased fussiness and irritability (n = 3). Poor feeding (n = 1), developmental delay (n = 1), and seizures (n = 1) were the other presenting symptoms. Parents did not report any concern regarding development in 6 patients. There was no significant antenatal or perinatal history concerning any infection or hypoxic ischemic injury. There was no history of preceding trauma. Family history of large head in parents was noted in 4 patients. There was no family history of developmental delay, seizures, or bleeding disorders. One patient was found to have multiple hepatic hemangiomas. On clinical examination, none of them had any dysmorphic features, stigmata of neurocutaneous syndromes, or obvious physical evidence of trauma. One patient had mild hepatosplenomegaly. All patients had increased head size (>95th centile at presentation). Neurological examination was completely normal in 6 patients. Three patients had mild restriction of up-gaze, and 1 patient had axial hypotonia with brisk deep tendon reflexes. Head size at birth was available for 5 patients out of which 1 had increased head size whereas the remaining 4 had normal head size.

Diagnostic Workup

All patients underwent MRI scan of the brain, 8 additionally had preceding CT scan of the head. Figures 1A and 1B show the MRI scan findings of the brain in

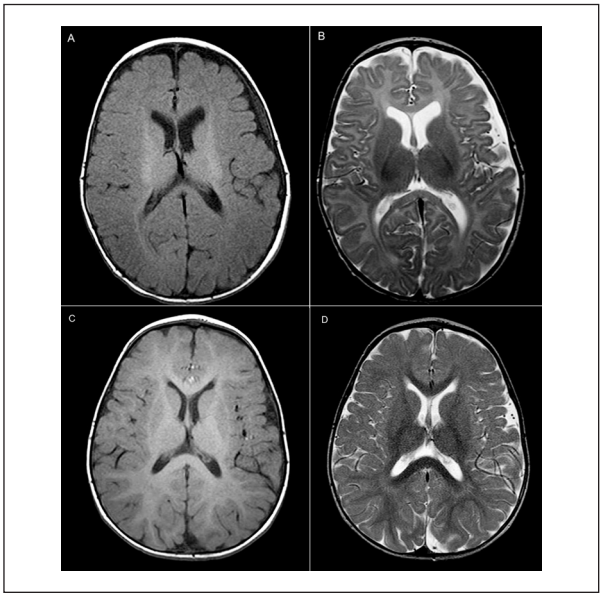

Figure 1. Magnetic resonance imaging scans of the brain (patient 6)
Minimal T1-weighted hyperintensity (A) and prominent T2-weighted hyperintensity (B) suggesting subacute subdural hemorrhage along left frontal convexity; note bilateral enlarged extra-axial space. T1-weighted (C) and T2-weighted (D) images in the same patient after 8 months: resolution of left frontal subdural hematoma and near normalization of enlarged extra-axial space.

Table 2. Neuroimaging Findings in the Children With BEH and SDH

Serial No.	Stage of SDH on MRI	Unilateral/ Bilateral	Mild Mass Effect	Ventricular Dilation
1	C	B	Yes	Yes (all vent)
2	S	U	No	Yes (lat + third)
3	S,A	B	Yes	Yes
4	A → S	B	No	No
5	S → C	U	Yes	Yes (lat + third)
6	S	U	No	No
7	S	B	No	No
8	C	B	Yes	No
9	S	B	No	Yes (temporal horns)

Abbreviations: BEH, benign external hydrocephalus; SDH, subdural hematoma; MRI, magnetic resonance imaging; C, chronic; S, subacute; A, acute; U, unilateral; B, bilateral; Lat, lateral ventricles; third, third ventricle.

patient 6. The initial CT scan of the head missed SDH in 2 patients, which were picked up by MRI. Prominent extra-axial cerebrospinal fluid (CSF) spaces were noted around the frontal and temporal poles, sylvian fissures and interhemispheric fissures in all of them. SDH was bilateral in 6 patients, and unilateral in 3. The most common stage of SDH noted was subacute (n = 5) followed by chronic (n = 2) and acute to subacute (n = 2) with one of them showing evidence of rebleeding. Mild mass effect was noted in 4 and underlying mild brain atrophy was initially suspected in 2 patients, which was disproved later on follow-up imaging. Prominent ventricles were reported in 5 patients (only lateral ventricles [2], lateral and third ventricles [2], and all ventricles [1]), but there was no evidence of periventricular lucency. None of the patients had overlying skull fractures (Table 2).

As presence of SDH on neuroimaging in a small child without clear history of trauma raises the question of NAI, all our patients underwent workup for NAI. These included skeletal survey, ophthalmologic examination to look for retinal hemorrhages, interview by social worker, and involvement of the child protective services when deemed necessary. Out of the 8 patients who underwent skeletal survey, one had multilevel lateral rib fractures (4th to 8th ribs on the left side) highly suggestive of NAI, and another patient had stable bilateral metaphyseal irregularities of the distal femurs medially without significant interval change or periosteal new bone formation suggesting normal developmental variant rather than NAI. None of them had skull fractures. Dilated eye examination was done in 6 patients, which was negative for retinal hemorrhages in all, including the child who had rib fractures. Social work and child protective services interview definitely confirmed NAI in one patient who was transferred to foster parents to ensure his safety.

The platelet counts, prothrombin time, and activated partial thromboplastin time were normal in all patients. Workup to look for any metabolic or genetic causes was done in 4 patients, which were normal (Table 3).

Management and Follow-Up

Two patients underwent operative procedures in our study. Both underwent evacuation of SDH followed by subdural–peritoneal shunt. All others were managed conservatively with close follow-up (Table 4). The follow-up ranged from 7 months to 8 years (39 ± 31 months). Mild up-gaze restriction noted in 3 patients normalized on follow-up. Development was normal in 8 patients. Residual mild left-sided spasticity was noted in 1, mild developmental delay in 1, and attention deficit/hyperactivity disorder (ADHD) was noted in 1 patient. Both the patients who underwent surgery for SDH had normal development on follow-up. Follow-up imaging was done in 8 patients; 1 patient who had small unilateral SDH, and had normal development, was followed only clinically. Complete resolution of SDH was noted in 3 (Figures 1C and1D,

Table 3. Diagnostic Evaluation of the Children With BEH and SDH

Serial No.	Screening Tests for Bleeding Diatheses	Skeletal Survey	Eye Examination	Metabolic Workup	NAI
1	Normal	Neg	NA	Neg	No
2	Normal	Neg	Neg	NA	No
3	Normal	Left 4th-8th rib fractures	Neg	Neg	Yes
4	Normal	Stable bilateral distal femoral metaphyseal irregularities	Neg	NA	No
5	Normal	Neg	Neg	Neg	No
6	Normal	Neg	Neg	NA	No
7	Normal	NA	NA	NA	No
8	Normal	Neg	Neg	Neg	No
9	Normal	Neg	NA	NA	No

Abbreviations: BEH, benign external hydrocephalus; SDH, subdural hematoma; Neg, negative; NA, not available; NAI, nonaccidental injury.

Table 4. Management and Follow-Up of the Children With BEH and SDH

Serial No.	Operative Procedure	Follow-Up	Head Size (Centile)	Developmental Delay/ Deficits	Follow-Up Imaging
1	None	2 y	90th	Normal	Marked reduction
2	None	4 y	90th	Normal	Marked reduction
3	Yes	8 y	>95th	Normal	R
4	None	7 mo	>95th	Normal, mild left spasticity	U
5	None	3 y	95th	Normal	NA
6	None	11 mo	>95th	Mild delay	R
7	None	7 y	50th-75th	ADHD	U
8	Yes	3 y	90th	Normal	Marked reduction
9	None	7 mo	95th	Normal	R

Abbreviations: BEH, benign external hydrocephalus; SDH, subdural hematoma; ADHD, attention deficit/hyperactivity disorder; R, resolution; U, unchanged; NA, not available.

patient 6), marked reduction in another 3, and remained stable in 2 patients. None of the subjects showed evidence of hydrocephalus, brain atrophy, or mass effect on follow-up scans. Specifically, follow-up MRI scans of the brain were normal in 2 children with initial suspicion of cerebral atrophy.

Discussion

Various terms have been proposed to describe the condition of excess extra-axial fluid collection around the brain, which include "benign subdural collections of infancy,"[15] "external hydrocephalus,"[4-6] "benign subdural effusion in infants,"[16] "benign communicating hydrocephalus in children,"[17] and "benign enlargement of the subarachnoid spaces in the infant."[6,18] All the above terms refer to the dilatation of the subarachnoid and subdural spaces in infants without prominent dilatation of the ventricles. Currently, the most common term used is "benign external hydrocephalus." Many hypotheses have been proposed for the pathogenesis of this condition, but none are proven. Although the subarachnoid spaces in infants have normal variations in size, the spaces over the frontal convexities observed by CT scan or MRI in normal infants are usually less than 0.5 mm in width.[19] The most popular pathogenetic theory is delayed maturation of the arachnoid villi preventing CSF absorption. Consequently, a mismatch between CSF production and absorption causes enlargement of the subarachnoid space, which is termed as external hydrocephalus.[20] Age-dependent maturation of arachnoid villi increases the CSF absorption with normalization of extra-axial space by 2 to 3 years of age. The patients with hydrocephalus develop dilated ventricles, whereas infants with BEH do not exhibit progressive dilatation of the ventricles.

Subarachnoid fluid collection in infants may mimic cerebral atrophy on CT scan or MRI. This explains the initial suspicion of cerebral atrophy in 2 of our cases;

however, that suspicion was proven wrong by follow-up scan. In most cases, however, this apparent atrophy can be differentiated from that seen in true cerebral atrophy.[21] Patients with cerebral atrophy exhibit prominent sulci without disproportionate bifrontal widening of the subarachnoid space as noted in BEH.[5] Follow-up studies showed that this extra-axial fluid collection eventually resolves over time without any intervention. Almost all patients attain normal developmental milestones with time, and head growth tends to stabilize along a curve parallel to the 95th percentile by 2 years of age.[7] Observation from our study also supports that conclusion. Even after complication with SDH, brain imaging on follow-up did not show any interval development of hydrocephalus. There was complete resolution of SDH in 3 patients and marked reduction in another 3 patients.

In our study, BEH was complicated by SDH[1,9,10,12] without any overt head injury, CSF shunting, bleeding disorder, or any metabolic disorder associated with weak vessel wall. This fact proves that BEH may not always be benign. Presence of SDH in an infant with no apparent history of trauma always raises the question of NAI. The diagnosis of NAI is not always straightforward and requires a thorough investigation. Several patterns of clinical and radiographic findings allow a definitive diagnosis. These include a history of trivial or no trauma, acute subdural hemorrhage, clear physical or radiologic evidence of head impact with SDH.[14] Although not necessary for the diagnosis, the findings of retinal hemorrhages or multiple fractures in different stages of healing make the diagnosis more certain.[22] Almost all patients (8 out of 9) in our series underwent skeletal survey of which only one had definite evidence of NAI determined by multiple level lateral rib fractures. Retinal hemorrhages were not seen in those examined (n = 6) even in the child with rib fractures. A social worker was involved in all cases. Based on those evaluations a diagnosis of NAI was established in only one child. Child protective services were appropriately involved and that child was shifted to the foster parents to ensure safety.

A variety of coagulopathies are associated with intracranial hemorrhage in infants, including hemophilia and hypoprothrombinemia caused by vitamin K deficiency.[23] These disorders are suggested by the clinical history, physical findings, and laboratory tests. Screening tests should include assessment of the platelet count, prothrombin time, activated partial thromboplastin time, and bleeding time; abnormal values merit further evaluation. Glutaric aciduria type I is a metabolic disorder caused by a defect of glutaryl coenzyme A dehydrogenase. The onset may be acute or insidious; the clinical features include developmental delay, hypotonia, dyskinesia, cortical atrophy, and subdural collections. Urinary screening for this disorder should be considered in infants with appropriate clinical findings.[24] Menkes disease is an X-linked inherited disorder of intestinal copper absorption resulting in copper deficiency. Cardinal features include hair abnormalities, facial dysmorphism, severe neurological impairment, hypothermia, arterial anomalies, bone abnormalities, and a fatal outcome. Menkes disease, like glutaric aciduria type 1, should be included in the differential diagnosis of unexplained SDH and neurological deficits in infants.[25] In our series, screening tests for bleeding disorders (platelets, prothrombin time, activated partial thromboplastin time) were negative in all. Metabolic testing to rule out glutaric aciduria Menkes disease was done in 4 patients when deemed clinically necessary but was negative in them.

All the above diagnoses need to be considered before assuming BEH–SDH association. In our cohort of 9 patients of BEH after excluding 1 patient with NAI, we could not find a clear reason other than BEH for the development of their SDH. The most likely mechanism behind development of SDH in the children in our cohort with BEH is minor forgotten trauma sustained during usual activities such as minor fall, car ride, rocking the baby in the crib back and forth while consoling during cry, and while in swing. Widened extra-cerebral space may cause significant brain displacement or rotation during a minor trivial head injury, which in turn leads to the development of SDH or effusion due to arachnoid membrane rupture or tearing of the already stretched bridging veins.[26] Papasian and Frim[27] have created an eloquent mathematical model of the intracranial space mimicking BEH. Using this model, they predicted an increased frequency of venous stretch injury in the situation of widened extra-axial space. Such venous injury may be produced even with forces generated by minor acceleration or deceleration of head or minor forgotten trauma sustained by a toddler.[27] This relationship could underlie a predisposition toward extra-axial bleeding after minor head trauma in infants with BEH. Thus, BEH cannot be considered truly benign as it predisposes the infants to SDH not explained by any significant head injury or other secondary cause(s). SDH associated with BEH may need surgical intervention if it expands or becomes symptomatic. Therefore, all patients with BEH and SDH should be followed carefully until the fluid collection disappears or stabilizes. In our series, 2 patients had to undergo evacuation of SDH with burr holes and irrigation followed by insertion of subdural–peritoneal shunt. This has been the usual practice as noted in the few previous cases described in the literature.[28-30]

All the families in our series had to go through extensive investigation and interrogation to rule out NAI, producing a lot of emotional trauma among the care givers.

In all but one case, extensive interrogation and detail investigation proved negative. We believe that the enlarged extra-axial space made these infants vulnerable to develop SDH with minor forgotten trauma, which was unnoticed by the caregivers. Though presence of SDH prompts a thorough workup to exclude NAI, association of SDH in the setting of BEH should be borne in mind and must be entertained as a possibility when workup for other causes draws a blank. Parents of children with BEH without SDH should be made aware of this possibility so that they can take extra precautions to avoid minor trauma, either direct or indirect, which may predispose these children to develop SDH, somewhat akin to those in the elderly population. This knowledge of association of BEH and SDH may also help in alleviation of unnecessary emotional trauma to the parents or care givers during interrogation to rule out NAI.

The timing of SDH was chronic, subacute, or acute on subacute based on the imaging characteristics of the blood in CT and/or MRI. Subarachnoid and subdural fluid collections cannot always be differentiated by CT scan, especially bilateral chronic low-density subdural hematomas. MR images easily demonstrate the nature and site of the extra-axial fluid.[31] Since methemoglobin formation in subacute or chronic hemorrhage displays high intensity, MRI is better than CT in picking up SDH. MRI thus plays an important role in management in such cases.[32] This has been observed in our series, too. Initial CT scans missed SDH in 2 patients, which were picked up later by MRI.

Our study is a retrospective review of patients with inherent limitations of the retrospective study. Not all of our patients had the same duration of follow-up. Thus we are not sure if there was a later recurrence of SDH or development of other signs of trauma in patients with short follow-up. NAI has been documented in infants presenting with only SDH without any physical evidence of trauma. So caution must be entertained when excluding a diagnosis of NAI.[33]

Conclusion

Benign external hydrocephalus is characterized by widening of the extra-axial fluid spaces, most likely because of physiological immaturity of the development of arachnoid villi preventing CSF absorption. Most children have spontaneous resolution of BEH without surgical intervention and they attain normal developmental milestones. However, the condition is not always benign and can predispose to the development of SDH from trivial trauma, which frequently goes unnoticed by the caregivers. Presence of SDH without clear history of trauma always raises the red flag for NAI. This condition can lead to devastating consequences, if missed. So, appropriate workup is required to exclude this. However, if the workup is negative, BEH–SDH relationship should be borne in mind. Parents or families of children with BEH should be made aware of this complication with regard to the following: (*a*) prevention—they should be advised to take extra precautions to prevent minor head trauma and (*b*) management—exhaustive workup can be avoided. The knowledge of the above fact may help the parents avoid going through emotional trauma associated with the workup for NAI. Infants with BEH and SDH should be monitored closely as they may require surgical intervention if SDH becomes symptomatic.

Declaration of Conflicting Interests

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