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CHILD ABUSE IMAGING

Birth-related subdural hemorrhage: prevalence and imaging morphology

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Abstract

Background Birth trauma accounts for 1–2% of the mortality in newborns with significant intracranial injuries presenting in the immediate postnatal period. However, a significant number of asymptomatic neonates harbor birth-related intracranial hemorrhage (ICH), with birth-related subdural hemorrhage (SDH) being a common occurrence on infant brain CT and MRI studies performed as a standard of care for a variety of reasons. Although clinically insignificant, birth-related SDH is frequently brought up in courts as an alternative explanation for SDH in suspected abusive head trauma.

Objective The aim of this study was to determine prevalence, imaging morphology and distribution of birth-related SDHs on brain CT and MRI studies obtained as a standard of care in infants up to 1 month old. We further tried to ascertain the relationship of birth-related SDHs with mode of delivery and birth weight.

Materials and methods Infants up to the age of 1 month who had CT or MRI of the brain performed between Jan. 1, 2018, and March 29, 2020, were included in this retrospective observational study. In addition to the imaging data, we reviewed clinical history, birth history including birth weight and mode of delivery, and final diagnoses.

Results Two hundred six infants younger than 30 days (range 0–29 days, mean 11.9 days, median 11 days and standard deviation [SD] 8.4 days) had a CT or MRI study during the study period. Among these, 58 infants were excluded as per the exclusion criteria. Among the included 148 infants, 88 (59.5%) had no imaging evidence of SDH. An additional 56 (37.8%) infants were assessed as having birth-related SDH based on review of clinical data. Within the birth-related SDH cohort (56 infants), only supratentorial SDH was identified in 5 (8.9%), only infratentorial SDH was identified in 14 (25%), while SDHs within both compartments were identified in 37 (66.1%) infants. The most common location for supratentorial birth-related SDH was along the occipital lobes (31/42, 73.8%), with other common locations being along the posterior interhemispheric fissure (30/42, 71.4%) and fronto-parietal convexity (9/42, 21.4%). The distribution of posterior fossa SDH was along the tentorium (38/51, 74.5%), along the cerebellum (38/51, 74.5%) and in both the locations (25/51, 49.0%). The rate of SDH was significantly higher in vaginal delivery group (46/84, 54.7%) as compared to caesarean section group (10/57, 17.5%) ($P<0.05$). We did not find any statistically significant difference between the birth weights of normal and birth-related SDH cohorts ($P>0.05$).

Conclusion Birth-related SDH is a common occurrence, with our study suggesting a prevalence of 37.8%. The most common distribution of birth-related SDH is within both the supra- and infratentorial compartments (66.1%) followed by infratentorial compartment (25%). The rate of birth-related SDH was significantly higher in vaginal delivery group as compared to caesarean section group.

Keywords Birth · Computed tomography · Hemorrhage · Infant · Magnetic resonance imaging · Subdural hematoma

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Introduction

Birth trauma accounts for 1–2% of the mortality in newborns, with significant intracranial injuries presenting in the immediate postnatal period with irritability, poor feeding, emesis, apnea, disordered breathing, bradycardia, seizures or disordered mentation [1, 2]. The reported incidence of symptomatic intracranial hemorrhage (ICH) ranges from 3 to 4 in 10,000 to 10–25 in 10,000 newborns [3–5]. A significant number of asymptomatic neonates harbor birth-related ICH, with birth-related subdural hemorrhage (SDH) being a common occurrence on infant brain CT and MRI studies performed for a variety of reasons. The exact prevalence of these birth-related SDHs is unknown, with various studies reporting a prevalence up to 26–63% of uncomplicated vaginal deliveries [6–9]. In addition, subdural hemorrhage is the most commonly observed intracranial pathology in young infants with abusive head trauma (AHT), occurring in up to 90% of cases [1, 6]. What complicates the matter is an unsubstantiated proposal, frequently proffered in the court, that acute rebleeding in a parturitional SDH can lead to acute collapse, coma or death months after delivery [1]. Hence, a morphological study of birth-related SDHs is important in the forensic evaluation of AHT because birth-related trauma is, not infrequently, used in courts of law as an explanation for SDH in infants with suspected non-accidental injury [6].

The purpose of this study was to determine imaging morphology and distribution of birth-related SDHs on CT and MRI studies of the brain in infants up to 1 month of age, obtained as a standard of care. We further tried to ascertain the relationship of SDHs with mode of delivery and birth weight.

Materials and methods

The institutional review board approved the study protocol. All infants up to the age of 1 month who had CT or MRI of the brain performed between Jan. 1, 2018, and March 29, 2020, were included in this retrospective observational study. In addition to imaging data, we reviewed clinical history, birth history such as birth weight and mode of delivery, and final diagnoses. We obtained the final diagnoses, as determined by the clinical team, from the clinical notes/summary available in electronic medical record. We excluded infants with suspected AHT and known causes of intracranial hemorrhage such as germinal matrix/intraventricular hemorrhage, parenchymal hemorrhage, hemorrhagic infarcts and dural venous sinus thrombosis. Traumatic SDH cases were defined by a documented history of witnessed accidental trauma or a final diagnosis of accidental trauma on discharge. AHT cases were determined by their final disposition, as determined by the institutional child protection team.

Imaging protocol

Imaging studies were obtained on a Signa 3-tesla (T) positron emission tomography (PET) MR scanner (GE Healthcare, Chicago, IL) or a Somatom Definition FLASH CT scanner (Siemens Healthcare, Erlangen, Germany). For MRI, we used the following sequences: 3-plane localizer, sagittal 3-D BRAVO (brain volume imaging) with axial and coronal reconstructions (repetition time/echo time [TR/TE] 8.5/3.4 ms, matrix 256×256, number of excitations [NEX] 1, slice thickness 0.8 mm, skip 0.4 mm); sagittal 3-D T2-weighted with axial and coronal reconstructions (TR/TE 2,500/85.9 ms, matrix 320×320, NEX 1, slice thickness 0.8 mm, skip 0.4 mm); axial susceptibility-weighted imaging (SWI; TR/TE 38.4/23.7 ms, matrix 224×320, NEX 1, slice thickness 2 mm, skip 1 mm); axial FLAIR (fluid attenuated inversion recovery; TR/TE 10,010/142.19 ms, matrix 224×320, NEX 1, slice thickness 3 mm, skip 3 mm); and axial diffusion-weighted imaging (DWI; TR/TE 8,000/68.6 ms, matrix 128×128, slice thickness 4 mm, skip 4 mm). The CT images were acquired on a Siemens Somatom Definition FLASH 256-slice dual-energy scanner. The reconstructions performed include axial soft-tissue windows with slice thickness of 0.6 mm and increments of 0.6 mm; axial, coronal and sagittal soft-tissue windows with slice thickness of 5.0 mm and increments of 5.0 mm; axial bone windows with slice thickness of 2.0 mm and increments of 2.0 mm; and 3-D volume-rendered reconstructions of the skull.

The MRI and CT studies and the clinical data were independently reviewed by two fellowship-trained neuroradiologists (V.V.K. and R.M.N., with 12 and 4 years of experience, respectively), and a pediatric radiology fellow (V.K. with greater than 5 years of general radiology experience). The positive cases of SDH were categorized based on detailed assessment of clinical and radiologic data as (1) no SDH, (2) traumatic SDH and (3) birth-related SDH. The birth-related SDH cohort was defined by the exclusion criteria: absence of accidental trauma, abusive head trauma and other identifiable causes of intracranial hemorrhage (germinal matrix/intraventricular hemorrhage, hypoxic–ischemic encephalopathy, cerebral infraction, dural venous sinus thrombosis, choroid plexus hemorrhage, subarachnoid hemorrhage, coagulopathy, etc.).

Overall prevalence of birth-related SDH was calculated within this retrospective cohort. To compare prevalence of SDH among the delivery groups, we used a chi-square test. We used a Student's *t*-test to compare the average birth weights of infants with birth-related SDH and those without. A P-value <0.05 was significant. Additionally, within the birth-related SDH cohort, we analyzed morphology of SDH including location and maximal width in either axial or coronal plane, calculated using electronic calipers in supra- and infratentorial compartments. In infants with SDH in multiple locations, we recorded the width of the largest SDH in supra- and infratentorial compartments.

Results

Two hundred six infants younger than 30 days had a CT or MRI study during the study period of Jan. 1, 2018, to March 29, 2020. The various indications for imaging were acidosis (*n*=2), apnea (*n*=7), suspected birth trauma following vacuum-assisted delivery (*n*=1), suspected congenital malformations (*n*=16), hypoxic–ischemic encephalopathy/cerebral infarction/dural venous sinus thrombosis (*n*=62), hydrocephalus (*n*=2), hypoglycemia (*n*=5), hypotonia (*n*=5), infection (*n*=15), lethargy (*n*=3), prematurity (*n*=28), seizures (*n*=25), stridor (*n*=1), suspected non-accidental trauma (*n*=3), trauma (*n*=30) and persistent vomiting (*n*=1). Among these, 58 infants were excluded as per the exclusion criteria (germinal matrix/intraventricular hemorrhage, *n*=22; parenchymal hemorrhage [hypoxic–ischemic encephalopathy, cerebral infarction, dural venous sinus thrombosis]), *n*=25; choroid plexus hemorrhage, *n*=1; subarachnoid hemorrhage *n*=2; either repeat or limited artifact-degraded studies, *n*=8). After exclusions, 148 infants were included in the study.

The cohort ranged 0–29 days old, with a mean of 11.9 days, median of 11.0 days and standard deviation (SD) of 8.4 days. The mode of delivery and birth weight data were not available for 3 and 5 infants, respectively. Seventy-four (50%) boys and 74 (50%) girls were included in this study. Among the 145 infants for which mode of delivery data were available, 85 infants (58.6%) were born by vaginal delivery, either spontaneous or assisted, and 60 infants (41.4%) were born via cesarean section. Of the total 148 infants, 41 (27.7%) underwent CT and 107 (72.3%) underwent MRI of the brain.

Among the 148 included infants, 88 (59.5%) had no imaging evidence of SDH. The final assessment category — birth-related SDH — was assigned to 56 (37.8%) infants based on detailed review of clinical findings, laboratory investigations such as absence of coagulopathy, and final diagnoses given by the clinical team. The other 4/148 (2.7%) infants had traumatic SDH.

When only MRI studies (*n*=107) were included in the cohort, the frequency of final assessment categories were: no imaging evidence of SDH, 57 (53.3%); birth-related SDH, 48 (44.9%); traumatic SDH, 2 (1.9%).

Within the total birth-related SDH cohort of 56 infants, 46 (82.1%) were delivered vaginally and 10 (17.9%) were delivered via caesarean section. Also within the total birth-related SDH cohort of 56 infants (48 with MRI, 8 with CT), only supratentorial SDH was identified in 5 (8.9%) infants, only infratentorial SDH was identified in 14 (25.0%), and SDHs within both compartments were identified in 37 (66.1%) infants (Figs. 1 and 2) (Table 1).

The thickness of supratentorial SDH ranged 0.5–4.0 mm, with a mean of 1.9 mm, median of 2.0 mm and SD of 0.75 mm (Table 1). The most common location for supratentorial birth-related SDH was along the occipital lobes (31/42, 73.8%), with other common locations being along the posterior interhemispheric fissure (30/42; 71.4%) and fronto-parietal convexity (9/42; 21.4%). Within the supratentorial SDH cohort, 26/42 (61.9%) infants had SDH in more than one location (Table 2).

The thickness of infratentorial SDH ranged 0.5–4.5 mm, with a mean of 1.8 mm, median of 2.0 mm and SD of 0.75 mm. The distribution of posterior fossa SDH was along the tentorium in 38/51 (74.5%), along the cerebellum in 38/51 (74.5%) and in both the locations in 25/51 (49%) infants (Table 3).

The incidence of birth-related SDH versus the mode of delivery is shown in Table 4. The rate of SDH was significantly higher in vaginal delivery group (46/84, 54.7%) as compared to caesarean section group (10/57, 17.5%) ($P<0.05$).

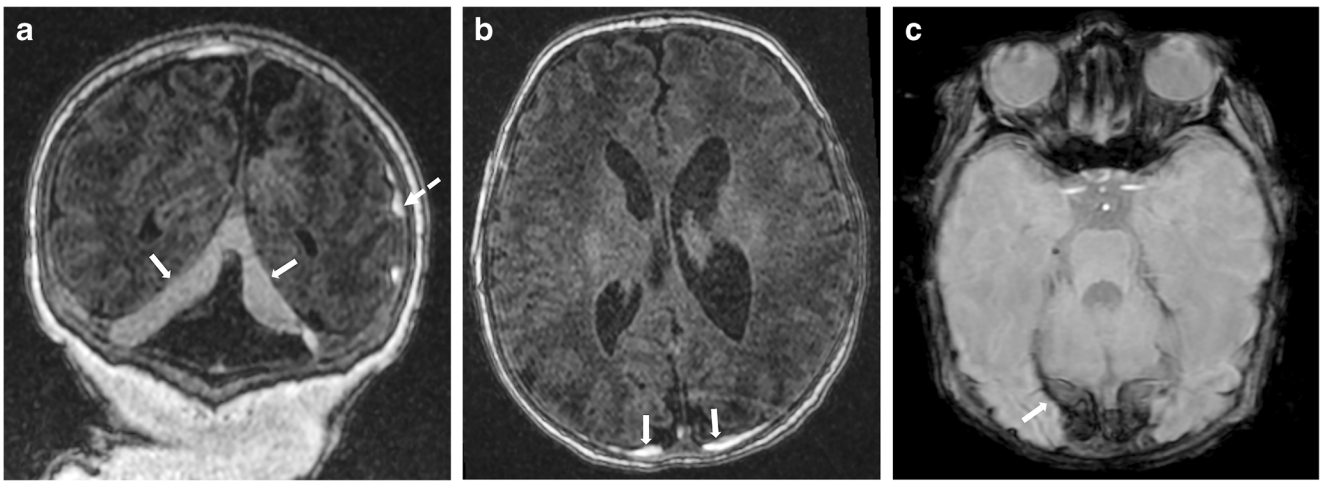

Fig. 1 Birth-related subdural hemorrhage (SDH) in a 6-day-old girl born by vaginal delivery. **a** Coronal T1-weighted MR image demonstrates hyperintense subdural hemorrhage along the tentorium (*solid arrows*) and, to a lesser extent, along the left parieto-occipital convexity (*dashed arrow*). **b** On axial T1-weighted MR image, a small amount of T1-hyperintense SDH is identified within the posterior cranium, along the parieto-occipital lobes (*arrows*). **c** Axial susceptibility-weighted image further corroborates the findings, as suggested by hypointense signal along the tentorium and posterior cranium (*arrow*)

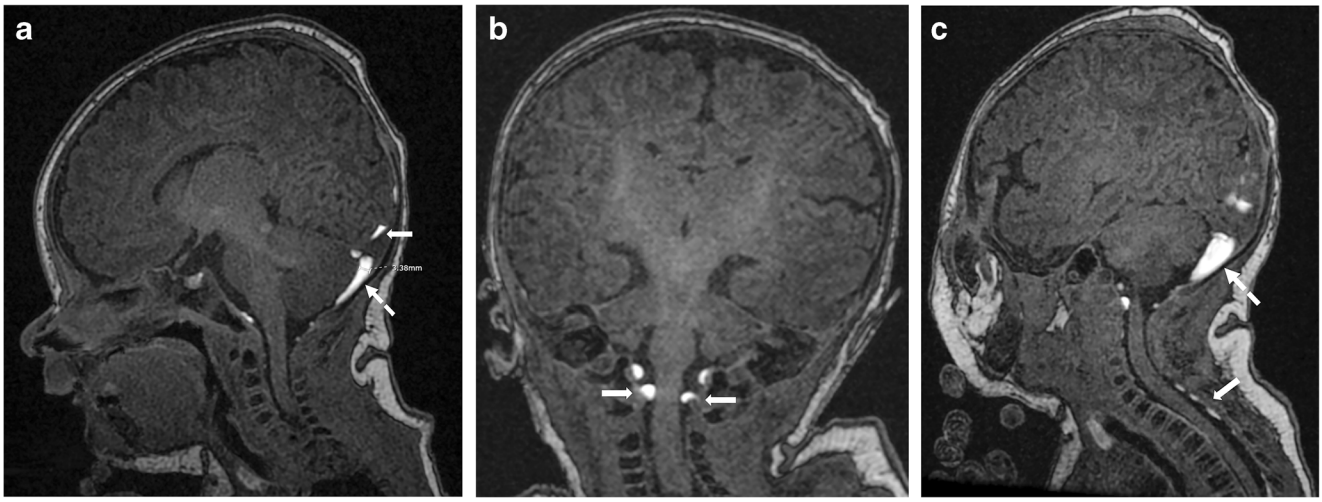

Fig. 2 Birth-related subdural hemorrhage (SDH) extending into the spinal canal in a 12-day-old boy with MRI performed for seizure-like episodes. The boy was full-term, born via cesarean section following a prolonged labor. Maternal history was significant for gestational diabetes. **a** Sagittal T1-weighted MR image demonstrates hyperintense SDH within the posterior cranium along the occipital lobes (*solid arrow*) and within the posterior fossa (*dashed arrow*), with placement of electronic calipers for measuring SDH width. **b** Coronal T1-weighted MR image shows small foci of extra-axial/subdural blood products at the cranio-vertebral junction (*arrows*). **c** Parasagittal T1-weighted MR image depicts hyperintense subdural blood products not only within the posterior cranium (*dashed arrow*), but also within the cervico-thoracic spinal canal (*solid arrow*). Multiple electroencephalographic studies were normal. At 12-month follow-up, the boy had normal neurodevelopment, was off phenobarbital, and had no seizure activity. The subdural hemorrhages were deemed to be birth-related in origin. This case emphasizes the fact that in rare instances, birth-related SDH can extend into the spinal canal

The birth weight in birth-related SDH cohort ranged 1,820–4,488 g, with a mean of 3,277 g and SD of 579.7 g. The birth weight in the cohort without SDH ranged 1,460–5,046 g, with a mean of 3,090 g and SD of 672.4 g. We did not find any statistically significant difference between the birth weights of normal and birth-related SDH cohorts ($P>0.05$). Figure 3 compares the frequency of birth-related SDH cases to the age of infants at the time of scan. The frequency of birth-related SDH peaked in the first week, followed by a steep decline, supporting that the prevalence of birth-related SDH is related to the age of the infant at the time of the imaging.

Discussion

Birth trauma accounts for 1–2% of the mortality in newborns with significant intracranial injuries presenting in the immediate postnatal period, with irritability, poor feeding, emesis, apnea, disordered breathing, bradycardia, seizures or disordered mentation [1, 2]. Birth-related SDH is an extremely common occurrence, with a prevalence of 26–63% in uncomplicated vaginal deliveries [6–10]. In our study, the prevalence of birth-related SDH was 37.8%. Rooks et al. [6], in their prospective study of 101 asymptomatic neonates, reported an incidence of 46%.

The various imaging modalities used for evaluating neonatal ICH include ultrasonography, CT and MRI, with a higher-field-strength (3-T) MRI with multiplanar imaging being considerably superior for identifying and characterizing neonatal ICH. Susceptibility-weighted imaging, because of its exquisite sensitivity for identifying blood products, further augments detection [11].

Dural anatomy and pathophysiology of birth-related subdural hemorrhage

Familiarity with dural anatomy and differentiating features of infant/fetal dura as compared to adults is vital for understanding the pathophysiology of birth-related SDH and the morphological differences between SDH in infants versus that seen in older children and adults. The outermost endosteal or periosteal layer, the middle or meningeal layer, and the inner dural border cell layer constitute the three components of dura mater [12]. The dural border cell layer is characterized by flattened cells with sinuous processes, extracellular spaces containing an amorphous material, and presence of intercellular junctions. There is no evidence to suggest the presence of subdural space intervening between the arachnoid barrier cell and dural

Table 1 Distribution and widths of birth-related subdural hemorrhage (SDH)

Birth-related SDH	Count	Percentage	Thickness of SDH
Supratentorial	5/56	8.9%	0.5–4.0 mm
Infratentorial	14/56	25.0%	0.5–4.5 mm
Both	37/56	66.1%	

Table 2 Location of birth-related subdural hemorrhage (SDH) in supratentorial compartment

Supratentorial SDH	Count	Percentage
Posterior IHF	30/42	71.4%
Occipital lobes	31/42	73.8%
Fronto-parietal convexity	9/42	21.4%
Greater than one location	26/42	61.9%

IHF interhemispheric fissure

border cell layers, with subdural hemorrhage most frequently occurring within the dural border cell layer [13].

One of the remarkable differentiating features of infant/fetal dura, as compared to that of an adult, is the presence of extensive, dense venous plexus between the periosteal and meningeal layers, forming sinuses in the tentorium, posterior falx, and the dura of the floor of the posterior cranial fossa [14, 15]. These fetal channels gradually shrink, dwindle or disappear, and they are absent in adolescents and adults [14]. The predominant distribution of birth-related SDH along the posterior aspects of brain, posterior falx, tentorium and posterior fossa, conforming to the distribution of dense venous plexi, suggests that neonatal SDH arises from the tributaries of the dural sinuses within the dural folds [16].

It has been proposed that tears of the falx and tentorium, or bridging cortical veins secondary to stretching, difficult delivery or abnormal labor, underlie development of birth-related SDH [2, 6, 17]. As per one hypothesis, increased circumferential pressure and squeezing of the head in the birthing canal result in overlap at the sutures, mechanical compression and shearing of the bridging veins during delivery, resulting in SDH [18]. However, imaging and autopsy studies frequently lack evidence of bridging vein injury in birth-related SDH, in contrast to bridging vein injury (lollipop sign) associated with AHT [19].

Risk factors

The risk factors associated with increased incidence of birth-related SDH include vaginal delivery, both spontaneous and assisted; prolonged first and second stages of labor; presence of cephalohematoma; and higher birth weight [6]. Our study demonstrated a statistically significant relationship between the mode of delivery and incidence of birth-related SDH ($P<0.05$), with rates of SDH significantly higher in our vaginal delivery group. However, we did not find any statistically significant difference between the birth weights of normal and birth-related SDH cohorts. Additionally, patient age at the time of imaging is an important factor dictating the incidence of birth-related SDH [6] because most SDHs resolve by 1 month of age [6, 20] (Fig. 3). One of the earliest works suggesting an association between the mode of delivery and ICH among term infants was a retrospective analysis published by Towner et al. [17] in 1999. Their observations were that the rates of ICH were higher among infants delivered with vacuum extraction, forceps and cesarean section during labor than among infants with spontaneous vaginal delivery. The authors concluded that complicated/abnormal labor, rather than the mode of delivery, is the risk factor for ICH [3, 17]. Holden et al. [21] in 1999 conducted a pilot study using a 1.5-T MR scanner, with normal asymptomatic neonates including eight born via uncomplicated spontaneous vaginal delivery and three by cesarean section, and found that SDH was present in four (50%) of the neonates delivered vaginally and none of those with cesarean section, further suggesting that spontaneous vaginal deliveries can be traumatic to the normal term newborn. Whitby et al. [20] in 2004 prospectively scanned normal term asymptomatic infants with a 0.2-T magnet within 48 h of birth. Among the 111 asymptomatic neonates in their cohort, 84 were born via vaginal delivery (including spontaneous and assisted) and 27 by cesarean section. Nine infants (8%) had SDH, all delivered by vaginal delivery; three had uncomplicated spontaneous vaginal delivery, five were delivered by forceps after an attempted ventouse delivery, and one had a traumatic ventouse delivery [20]. The SDH in all the nine infants had resolved on follow-up imaging at 4 weeks, and the infants were neurologically normal [20]. The reported low incidence of SDH by Whitby et al. [20] is probably related to a low-field-strength magnet. Looney et al. [9], in their prospective study of 88 asymptomatic neonates, reported that all ICH occurred in vaginally delivered infants with a frequency of 26%, the overwhelming majority of these being SDH. One of the most notable studies addressing the prevalence and risk factors of birth-related SDH was published by Rooks et al. [6] in 2008. Their cohort included 101 asymptomatic neonates, 79 born via vaginal delivery and 22 by cesarean section, with MRI performed within 72 h of birth [6]. The overall frequency of SDH was 46% and was significantly more frequent with vaginal delivery than cesarean section ($P<0.01$). There was significant association between SDH and prolonged second stage of labor ($P<0.01$) [6]. The incidence of cephalohematoma was greater in

Table 3 Location of birth-related subdural hemorrhage (SDH) in infratentorial compartment

Infratentorial SDH	Count	Percentage
Cerebellum	38/51	74.5%
Tentorium	38/51	74.5%
Greater than one location	25/51	49.0%

Table 4 The incidence of birth-related subdural hemorrhage (SDH) versus the mode of delivery

	Vaginal deliveries	Caesarean sections
Total births	84	57
Infants with birth-related SDH, *n* (%)	46 (54.7)	10 (17.5)
P-value[a]	<0.05	

[a] $P<0.05$ is significant

neonates with SDH than in those without. Additionally, the mean birth weight of neonates with SDH on MRI was higher than for those with normal findings [6].

Imaging morphology

The common locations for small birth-related SDHs include along the tentorium, parieto-occipital convexity, inter-hemispheric fissure and retro-cerebellar posterior fossa [1]. In our cohort, the most common distribution of birth-related subdural hemorrhages was within both supra- and infratentorial compartments (66.1%), followed by infratentorial compartment (25.0%). The maximum width of the supratentorial SDH ranged 0.5–4.0 mm, and for infratentorial SDH the range was 0.5–4.5 mm. The most common location for supratentorial birth-related SDH was along the occipital lobes (73.8%), followed by along the posterior interhemispheric fissure (71.4%) and fronto-parietal convexity (21.4%). Within the supratentorial SDH cohort, 67.9% had SDH in more than one location. The predominant distribution of posterior fossa SDH was along the tentorium (74.5%) and along the cerebellum (74.5%).

In the cohort reported by Rooks et al. [6], all 46 infants with ICH had supratentorial SDH, with common locations being along the posterior interhemispheric fissure (65%), along the occipital lobes (63%) and over the tentorium (48%). Most neonates had SDH in more than one location. Posterior fossa SDHs were identified in 43% of cases. There were no epidural, subarachnoid or intra-parenchymal hemorrhages [6]. The overwhelming majority of SDHs were ≤3 mm in thickness (95.9%), with a range of 1.0–4.3 mm and mean of 2.1 mm. All SDHs were homogeneous in signal intensity on all MRI sequences [6]. Similar findings of distribution of birth-related SDHs within the posterior cranium were reported by Holden et al. [21], Tavani et al. [8], Whitby et al. [20] and Looney et al. [9]. The distribution of birth-related SDHs along the posterior aspects of brain, posterior falx, tentorium and posterior fossa conforms to the vascular dural folds, supporting the fact that neonatal SDH arises from the tributaries of the dural sinuses within the dural folds [16]. In contrast, imaging findings of SDH that are strongly suggestive of abusive head trauma include mixed attenuation/mixed signal intensity hemorrhage, unilateral hemispheric location, association with diffuse cerebral edema, cranio-vertebral junction injury, severe retinal hemorrhages and presence of venous injury, commonly at the junction of the bridging vein and superior sagittal sinus complex [1, 12, 19]. Interhemispheric SDHs have been reported with both accidental and birth-related trauma, and do not confer specificity to the type or mechanism of injury [6, 22].

The imaging studies in our cohort were performed for a variety of indications, and the diagnosis of birth-related SDH was based on the clinical and radiologic data. Although our study does not offer a true prevalence of asymptomatic birth-related SDH, it mimics the clinical practice.

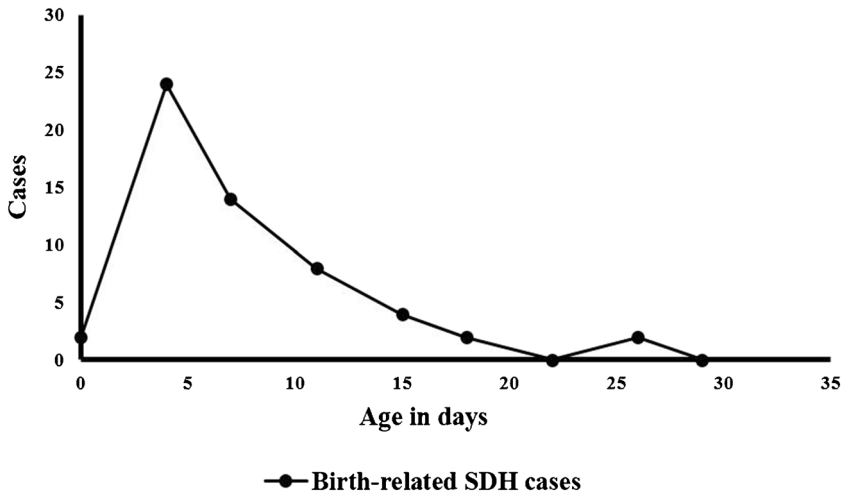

Fig. 3 Graph shows frequency of birth-related subdural hemorrhage (SDH) cases versus the age of infants at the time of imaging. The frequency of birth-related SDH peaks in the first week, followed by a steep decline, supporting that the prevalence of birth-related SDH is related to the age of the infant at the time of the imaging

Clinical significance

Neonates with significant birth trauma usually present within hours to days after birth with a variety of symptoms. Asymptomatic injuries, particularly small birth-related SDHs, are a common occurrence and resolve in the overwhelming majority of infants within 4 weeks [6, 23]. Long-term follow-up studies reported by Holden et al. [21] (average duration of follow-up: 3.9 years) and Rooks et al. [6] (duration of follow-up: 2 years) demonstrated no significant differences in clinical outcome compared to the normal population. Although none of the studies addressed the incidence of subclinical rebleeding in birth-related SDH, normal clinical development corroborates the fact that major clinically significant rebleeding remains an extremely rare phenomenon. Additionally, multiple clinical imaging findings can aid in differentiating rebleeding in chronic-SDH-related innocent causes versus re-injury/inflicted trauma. Clinical findings of minimal symptoms/signs, localized small SDHs, homogeneous SDH and absence of subarachnoid hemorrhage and cerebral edema favor innocent causes [12]. In contrast, findings indicative of re-injury/inflicted trauma include neurologic deterioration, large or multiple new SDHs, heterogeneous SDH and presence of subarachnoid hemorrhage and cerebral edema [12].

There are number of limitations to this study. It is retrospective in nature, and this limits what was available to review. No longitudinal follow-up data were available for our cohort, which limited us from evaluating clinical significance of birth-related SDHs or evolution of disease processes. We tried to minimize limitations by carefully reviewing all the available clinical records and by forming a consensus opinion where required. Also, our cohort is not representative of all births but includes a subset of neonates who underwent imaging. In addition, we think our study underestimates the prevalence of birth-related SDHs because we excluded infants with known intracranial hemorrhage such as germinal matrix hemorrhage, which might harbor a contemporaneous birth-related SDH.

Conclusion

Birth-related SDH is a common occurrence, with our study suggesting a prevalence of 37.8%. The most common distribution of birth-related SDHs is within both supra- and infratentorial compartments, followed by the infratentorial compartment. The rate of SDH was significantly higher in the vaginal delivery group as compared to the caesarean section group. However, we did not find any statistically significant difference between the birth weights of normal and birth-related SDH cohorts.

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Declarations

Conflicts of interest None

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