Radiology

ORIGINAL RESEARCH • PEDIATRIC IMAGING

Subdural Hemorrhage in Asymptomatic Neonates: Neurodevelopmental Outcomes and MRI Findings at 2 Years

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Background: Subdural hemorrhage (SDH) is thought to have a benign course in asymptomatic neonates. However, effects on neurodevelopmental outcomes have not been established.

Purpose: To evaluate neurodevelopmental outcomes, gray matter volumes, and MRI findings in asymptomatic neonates with SDH compared with control neonates.

Materials and Methods: This retrospective analysis was conducted between 2003 and 2016 and was based on data from the University of North Carolina Early Brain Development Study. Neurodevelopmental outcomes were evaluated at 2 years of age by using the Mullen Scales of Early Learning (MSEL). All infants were imaged with 3.0-T MRI machines and were evaluated for SDH at baseline (neonates) and at ages 1 and 2 years. Volumetric MRI for brain segmentation was performed at ages 1 and 2 years. A secondary analysis was performed in neonates matched 1:1 with control neonates. Differences in categorical variables were measured by using the Fisher exact test, and the *t* test was used for continuous variables.

Results: A total of 311 neonates (mean gestational age ± standard deviation, 39.3 weeks ± 1.5), including 57 with SDH (mean gestational age, 39.5 weeks ± 1.2), were evaluated. The subgroup included 55 neonates with SDH (mean gestational age, 39.6 weeks ± 1.2) and 55 matched control neonates (mean gestational age, 39.7 weeks ± 1.2). Fifty-five of 57 neonates with SDH (97%; 95% CI: 92, 100) were delivered vaginally compared with 157 of 254 control neonates (62%, 95% CI: 56, 68; $P < .001$). Otherwise, there were no differences in perinatal, maternal, or obstetric parameters. There were no differences in composite MSEL scores (115 ± 15 and 109 ± 16 at 2 years, respectively; $P = .05$) or gray matter volumes between the neonatal SDH group and control neonates (730 cm^3 ± 85 and 742 cm^3 ± 76 at 2 years, respectively; $P = .70$). There was no evidence of rebleeding at follow-up MRI.

Conclusion: Neurodevelopmental scores and gray matter volumes at age 2 years did not differ between asymptomatic neonates with subdural hemorrhage and control neonates.

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Neonatal intracranial hemorrhage was initially thought to be rare and associated with substantial morbidity and mortality (1–3). However, recent studies show that intracranial hemorrhage, particularly subdural hemorrhage (SDH), is a common MRI finding in asymptomatic neonates within the 1st month of life, with a prevalence of 26%–46% (4–6). Unlike SDH associated with abusive head trauma, which is frequently located along the interhemispheric fissure or cerebral convexities (5,7–9), SDH in otherwise healthy or asymptomatic neonates is predominantly found in the posterior fossa or overlying the occipital lobes near the tentorium (5,6). Although a large body of literature documents the long-term disability associated with abusive head trauma (10), no controlled studies have addressed the neurodevelopmental effect of incidentally discovered SDH in neonates.

The purpose of this study was to assess whether there are differences in standardized measures of cognitive, language, and motor development between asymptomatic neonates with SDH and those with a normal neonatal brain MRI study by using the Mullen Scales of Early Learning (MSEL). In addition, because the total surface area of gray matter is associated with MSEL scores (11), a secondary goal was to determine any differences in gray matter volumes at 2-year follow-up.

Materials and Methods

This study was compliant with the Health Insurance Portability and Accountability Act and was approved by our institutional review board. This retrospective analysis is based on prospectively acquired data from the University of North Carolina Early Brain Development Study (12,13), which recruited pregnant women for evaluation of prenatal and early childhood brain development. Seventy of 318 neonates included here were described in the report of a prior study on the prevalence of intracranial

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Abbreviations

FDR = false discovery rate, MSEL = Mullen Scales of Early Learning, SDH = subdural hemorrhage

Summary

Neurodevelopmental outcomes and gray matter volumes at age 2 years did not differ between asymptomatic neonates with subdural hemorrhage and those with normal neonatal MRI findings.

Key Results

- In a comparison of asymptomatic neonates aged 2 years with a history of subdural hemorrhage versus those with normal neonatal MRI findings, there were no differences in Mullen Scales of Early Learning scores (115 ± 15 vs 109 ± 16; $P = .053$) or total gray matter volumes (730 cm^3 ± 85 vs 742 cm^3 ± 76; $P = .70$), respectively.
- At 2-year follow-up MRI, there was no evidence of rebleeding in asymptomatic neonates.

hemorrhage in asymptomatic neonates and associated perinatal risk factors (5). That study found a high prevalence of SDH in asymptomatic neonates. The current study focuses on a different end point by evaluating standardized neurodevelopmental outcomes and differences in intracranial volumes at 2 years of age in a larger number of neonates. The neonates' parents provided written informed consent.

Neonates

Pregnant mothers were recruited from obstetrics and gynecology clinics at the University of North Carolina Hospitals in a consecutive series. All participants were born between 2003 and 2014 and included *(a)* children born to mothers with no psychiatric history, *(b)* offspring of mothers with a psychiatric history, and *(c)* children previously diagnosed with fetal isolated mild ventriculomegaly at prenatal US. Pregnancies in the last two subgroups were classified as high risk (Table 1). Mothers with substance use or major illness during pregnancy were excluded from the study. Exclusions for this analysis included lack of neonatal brain MRI, incomplete or nondiagnostic MRI examinations, twin pregnancies, neonates with pathologic abnormalities at neonatal brain MRI other than SDH, additional pathologic abnormalities or major medical conditions identified before the 1- or 2-year timepoints, lack of MSEL scores, or outliers based on patient demographic characteristics.

Developmental Testing

Cognitive development was assessed at age 2 years by trained staff through use of MSEL, which is designed to assess five developmental domains: visual perception, expressive language, receptive language, fine motor skills, and gross motor skills. The MSEL has been standardized in a large sample of children in the United States and has well-established validity and reliability (14–16). Age-adjusted T-scores (mean ± standard deviation, 50 ± 10) for the first four parameters are combined to create the Early Learning Composite, which reflects general cognitive function. The composite score is also standardized for age, with a mean score of 100 ± 15. T scores for subscales and the MSEL composite scores were calculated based on gestational age at testing. More detail is available in Appendix E1 (online).

MRI Protocol

MRI examinations were performed between 2003 and 2016 on a Trio 3.0-T machine or an Allegra 3.0-T machine (both from Siemens Healthineers, Erlangen, Germany) (17). Neonatal images were acquired without sedation by using the feed-and-swaddle technique to minimize motion. The imaging protocol has been described previously (11) and consisted of T1-weighted, proton density, and T2-weighted sequences. Volumetric MRI was used for brain segmentation to assess for differences in cerebral volumes at follow-up MRI at 1 and 2 years of age. Because the type of MRI machine may affect quantification of gray matter volumes, we included it as a covariate for data analysis (18). Detailed MRI parameters can be found in Appendix E2 (online).

Image Analysis

All infants underwent neonatal MRI between 1 and 5 weeks after birth; this testing was used for dichotomization into SDH and control (ie, normal MRI findings) groups. MRI findings at baseline (neonate) and at ages 1 and 2 years were independently reviewed by a board-certified neuroradiologist (J.K.S.) with 24 years of experience who was blinded to clinical and imaging report data, except for the MRI timepoint. There was a single readout session per infant. SDH was diagnosed at neonatal MRI and defined as signal abnormalities compatible with subacute blood products (T1 hyperintense and T2 hypointense relative to cerebrospinal fluid) between brain and calvaria or along dural reflections that did not enter the sulci.

Statistical Analysis

Statistical analyses were performed by using SAS software, version 9.4 (SAS Institute, Cary, NC). For demographic characteristics, frequency distributions were calculated for categorical variables and means (± standard deviations) were calculated for continuous variables. Group differences in demographic characteristics were measured by using the two-sided Fisher exact test for categorical variables and *t* tests for continuous variables. Means (± standard deviations) were calculated for global brain tissue volumes and cognitive measures. Group differences for these variables were evaluated by using Pearson partial correlations adjusted for covariates relevant to the measures analyzed and for different demographic characteristics between groups.

Because a group difference in age at neonatal MRI may have biased detection of SDH, a secondary analysis was performed in a group of neonates with SDH matched 1:1 with control neonates. We also performed two separate sensitivity analyses to evaluate robustness of the primary analysis. All statistical hypothesis tests were two tailed, and results with $P < .05$ were considered to indicate a significant difference. Further detail on statistics is found in Appendix E3 (online).

Table 1: Demographic and Baseline Neonate Characteristics

Characteristic	Neonatal MRI Showing SDH (n = 57)	Normal Neonatal MRI Findings (n = 254)	P Value*
Gestational age at birth (wk)	39.5 ± 1.2	39.2 ± 1.5	.13
Birth weight (kg)	3.5 ± 0.4	3.4 ± 0.5	.29
Birth head circumference† (cm) (n_1 = 54, n_o = 230)	34 ± 2	34 ± 2	.97
Birth length† (cm) (n_1 = 54, n_0 = 234)	51 ± 2	51 ± 3	.39
Apgar score at 5 minutes	9 ± 0.3	9 ± 0.7	.34
Maternal age at birth (y)	30 ± 5	31 ± 6	.55
Maternal education at birth (y)	16 ± 2	15 ± 3	.32
Total household income at birth (thousands of U.S. dollars)† (n_1 = 54, n_0 = 229)	79 ± 56	70 ± 49	.25
Age at neonatal MRI (d)	17 ± 6	24 ± 10	<.001
MRI machine at neonate examination			.37
Allegra	48 (84)	225 (89)	…
Trio	9 (16)	29 (11)	…
Sex			.66
Female	30 (53)	123 (48)	…
Male	27 (47)	131 (52)	…
Maternal race or ethnicity			.88
Asian	1 (2)	4 (2)	…
Black	12 (21)	49 (19)	…
White	44 (77)	201 (79)	…
Hispanic	3 (5)	20 (8)	…
Non-Hispanic	54 (95)	234 (92)	…
Stay in NICU			.14
No	57 (100)	241 (95)	…
Yes	0 (0)	13 (5)	…
Method of delivery			<.001
Vaginal	55 (96)	157 (62)	…
Cesarean	2 (4)	97 (38)	…
Risk level of pregnancy			.06
High risk	8 (14)	67 (26)	…
Not high risk	49 (86)	187 (74)	…
Age at follow-up MRI (d)			
1 year† (n_1 = 44, n_0 = 140)	377 ± 20	383 ± 21	.09
2 years† (n_1 = 27, n_0 = 127)	740 ± 16	749 ± 27	.11
Age at MSEL composite score (d)			
1 year† (n_1 = 56, n_0 = 234)	374 ± 19	380 ± 19	.05
2 years (n_1 = 57, n_0 = 254)	744 ± 18	744 ± 24	.92

Note.—Unless otherwise indicated, data are presented as mean ± standard deviation for continuous variables and as number (percentage) for categorical variables. MSEL = Mullen Scales of Early Learning, NICU = neonatal intensive care unit, n_1 = number of neonates in group 1, n_0 = number of neonates in group 2, SDH = subdural hemorrhage.

* Fisher exact test for categorical variables and t test for continuous variables.

† Data are missing for some neonates.

Results

Neonates

Of 584 children enrolled in the University of North Carolina Early Brain Development Study, 311 (mean gestational age ± standard deviation, 39.3 weeks ± 1.5) were included for primary analysis after exclusions (Fig 1). A total of 57 patients had SDH (mean gestational age, 39.5 weeks ± 1.2). Fifty-five of 57 neonates with SDH (97%; 95% CI: 92, 100) were delivered vaginally compared with 157 of 254 control neonates (62%; 95% CI: 56, 68; $P < .001$). Except for method of delivery, perinatal, maternal, or obstetric parameters did not differ between neonates with SDH and those with normal neonatal MRI results. For the entire cohort, neonatal MRI was performed at a mean age of 17 days ± 6 in the SDH group and 24 days ± 10 in the control group ($P < .001$, Table 1). Because of this difference and the possibility that a small SDH may have resolved and may not have been detected on images obtained at later ages, we also analyzed a subgroup of 110 neonates composed of 55 neonates with SDH (mean

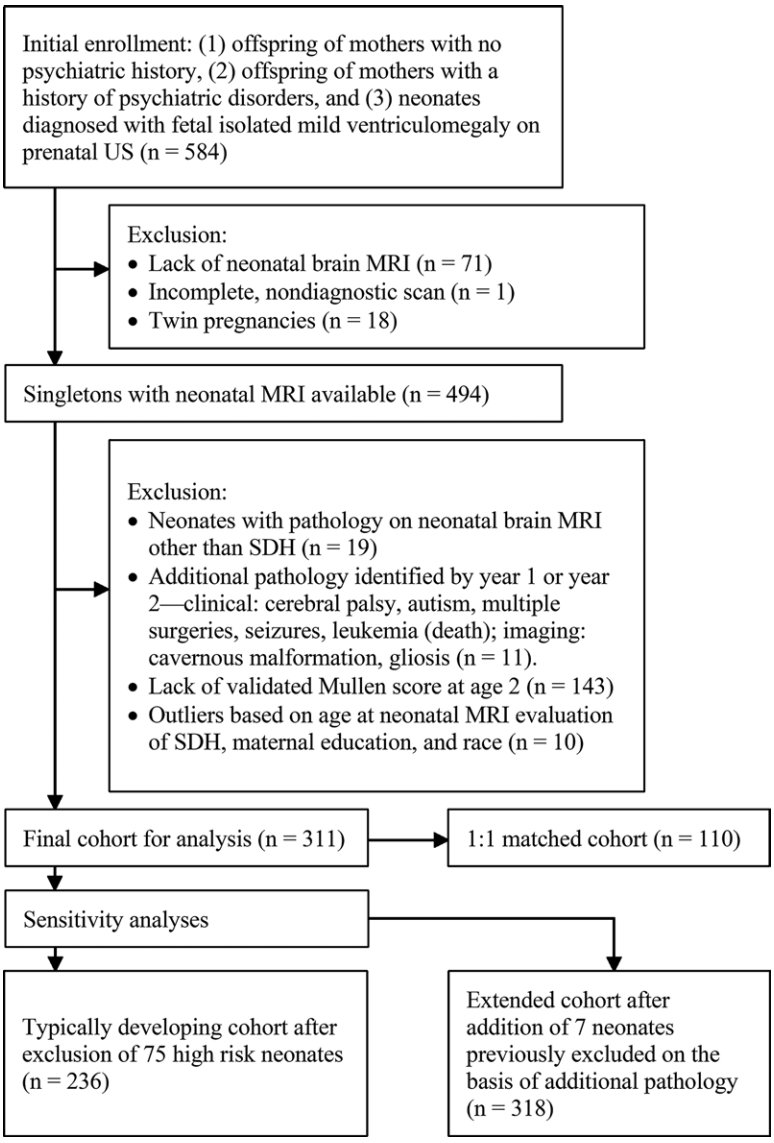


Figure 1: Flowchart shows neonate inclusion and exclusion criteria. SDH = subdural hematoma.

gestational age, 39.6 weeks ± 1.2) and 55 matched control neonates (mean gestational age, 39.7 weeks ± 1.2) to account for age at neonatal MRI assessment and other variables, as detailed in the Materials and Methods. Analysis of this matched cohort yielded no differences in perinatal, maternal, or obstetric parameters (Table E1 [online]).

Developmental Testing

Because of the difference in delivery method between neonates with and those without neonatal SDH, we tested for a potential effect of this variable on MSEL composite scores at age 2 years and found no association (MSEL composite score, 111 ± 15 and 108 ± 16, respectively; P = .13).

Composite MSEL scores did not differ between neonates with SDH and control neonates (mean, 115 ± 15 and 109 ± 16 at 2 years, respectively; P = .05) after adjustment for the following covariates: age at developmental testing, neonatal age at MRI assessment of SDH, type of MRI machine used for evaluation of neonatal hemorrhage, gestational age at birth, birth weight, sex, and years of maternal education at enrollment in the study (Table 2). Similarly, there were no differences in composite MSEL scores in the matched cohort between neonates with SDH and control neonates (mean, 116 ± 13 and 113 ± 15 at 2 years, respectively; P = .54) (Table E2 [online]). Further analysis of the five developmental domains showed a difference in receptive language in the entire cohort, which did not persist after false discovery rate (FDR) correction for multiple comparisons (mean, 61 ± 9 in the SDH group and 57 ± 10 in the group with normal neonatal MRI findings [P = .02]; FDR P = .10) or on analysis of the matched cohort (mean, 61 ± 8 and 58 ± 12, respectively; P = .16).

Imaging

Table 3 and Table E3 (online) summarize MRI parameters at 1 and 2 years of age in the entire cohort and in the matched cohort, respectively. In the group with neonatal SDH, there was one instance of Chiari 1 malformation detected at the 2-year timepoint. In the group with normal neonatal MRI findings, there were four and five infants with Chiari 1 malformations, respectively, detected at 1 and 2 years of age and one instance of mild ventriculomegaly at 1 year of age. Neither group showed evidence of intercurrent hemorrhage (Figs 2, 3).

There were no differences in gray matter volumes at either time point (entire cohort at 1 year: 634 cm^3 ± 65 in the SDH group and 637 cm^3 ± 63 in the control group, P = .91; entire cohort at 2 years: 730 cm^3 ± 85 in the SDH group and 742 cm^3 ± 76 in the control group, P = .69; matched cohort at 1 year: 630 cm^3 ± 63 in the SDH group and 634 cm^3 ± 66 in the control group, P = .78; entire cohort at 2 years: 725 cm^3 ± 87 in the SDH group and 742 cm^3 ± 59 in the control group, P = .12).

Sensitivity Analyses

Seventy-five neonates at high risk were removed for the first sensitivity analysis, and seven neonates previously excluded based on additional pathologic abnormalities were added to the second analysis. After covariate adjustment in the first analysis, composite MSEL and receptive language domain scores were higher in children with neonatal SDH than in control neonates at age 2 years, from which only receptive language remained different after adjustment for multiple comparisons (composite MSEL: 118 ± 11 in the SDH group and 112 ± 15 in the control group [P = .03], FDR P = .09; receptive language: 62 ± 7 in the SDH group and 58 ± 10 in the control group [P = .007], FDR P = .04). In the second analysis, there was a difference in receptive language that did not persist after

Table 2: Outcomes of Mullen Scales of Early Learning at 2 Years

2-Year MSEL Score	Neonatal MRI Showing SDH (n = 57)	Normal Neonatal MRI Findings (n = 254)	Adjusted Correlation with Group*		
			R Value	P Value	FDR P Value
Composite	115 ± 15	109 ± 16	0.11	.05	.16
Gross motor†	55 ± 7	54 ± 8	0.00	.95	.95
Visual reception	55 ± 10	53 ± 11	0.06	.29	.35
Fine motor	56 ± 8	54 ± 7	0.09	.11	.22
Receptive language	61 ± 9	57 ± 10	0.14	.02	.10
Expressive language	58 ± 10	55 ± 10	0.07	.22	.32

Note.—Scores for Mullen Scales of Early Learning are presented as standardized values ± standard deviation. Standard scores are 100 ± 15 for the composite score and 50 ± 10 for the individual subscores. FDR = false discovery rate, MSEL = Mullen Scales of Early Learning, SDH = subdural hemorrhage.

* Group 1 = neonatal SDH, group 0 = normal neonatal MRI findings. Adjusted for age at 2-year MSEL score assessment, neonatal age at MRI assessment of SDH, MRI machine type at neonatal assessment of SDH, gestational age at birth, birth weight, sex, and years of maternal education.

† Group 1, n = 57; group 2, n = 251.

Table 3: MRI Parameters at 1 and 2 Years (n = 311)

Variable	Neonatal MRI with SDH	Normal Neonatal MRI Findings	Adjusted Correlation with Group*†	
			r Value	P Value
MRI status at 1 year (n_1 = 41, n_0 = 134)				
Abnormal	0	5	…	…
Normal	41	129	…	…
MRI status at 2 years (n_1 = 26, n_0 = 123)				
Abnormal	1	6	…	…
Normal	25	117	…	…
ICV at 1 year (cm^3)†				
Total	929 ± 107	934 ± 98	0.02	.85
Gray matter	634 ± 65	637 ± 63	0.01	.91
White matter	225 ± 35	229 ± 32	0.00	.99
Cerebrospinal fluid	70 ± 12	68 ± 10	0.09	.29
ICV at 2 years (cm^3)‡				
Total	1066 ± 128	1083 ± 116	−0.03	.73
Gray matter	730 ± 85	742 ± 76	−0.03	.69
White matter	264 ± 39	267 ± 34	−0.02	.83
Cerebrospinal fluid	72 ± 10	73 ± 10	−0.02	.79

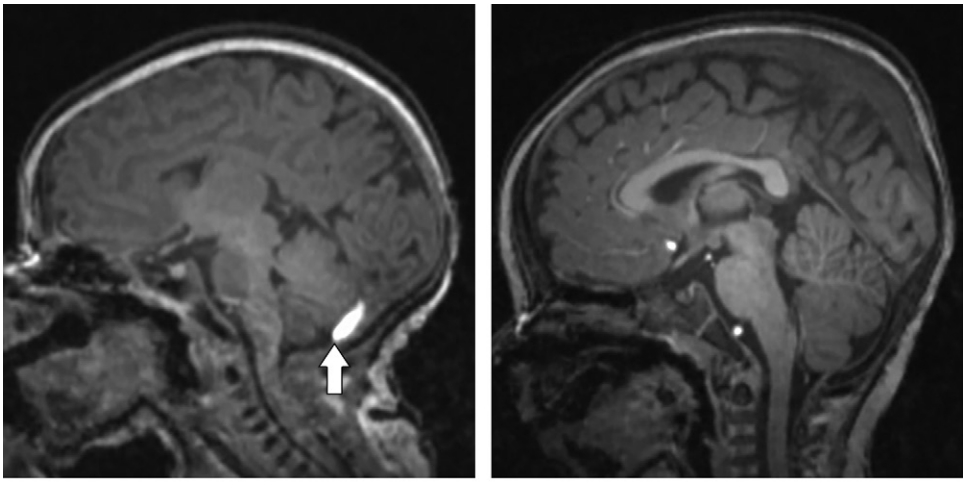
Note.—Unless otherwise indicated, data are numbers for categorical variables and mean ± standard deviation for continuous variables. ICV = intracranial volume, SDH = subdural hemorrhage.

* Group 1 = neonatal SDH, group 0 = normal neonatal MRI. Adjusted for age at 2-year Mullen Scales of Early Learning score assessment, neonatal age at MRI assessment of SDH, MRI machine type at neonatal assessment of SDH, gestational age at birth, birth weight, sex, and years of maternal education.

† Number of neonates by MRI machine: group 1, n = 35 (Allegra scanner = 25, Trio scanner = 10); group 0, n = 111 (Allegra scanner = 96, Trio scanner = 15).

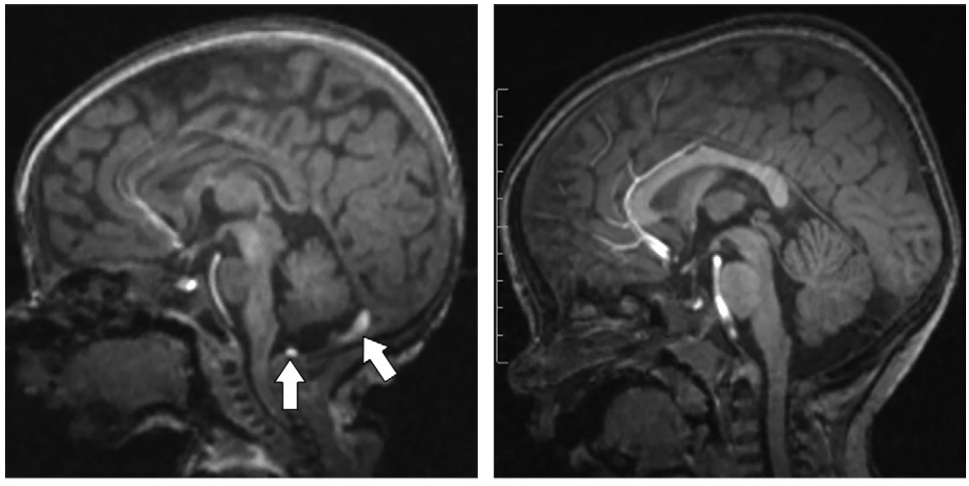
‡ Number of neonates by MRI machine: group 1, n = 25 (Allegra scanner = 19, Trio scanner = 6); group 0, n = 117 (Allegra scanner = 101, Trio scanner = 16).

FDR correction (60.5 ± 9 in the SDH group and 56 ± 10 in the control group [P = .02], FDR P = .14). There were no differences in gray matter volumes at 1 or 2 years of age for either analysis (first analysis at 1 year: 625 cm^3 ± 57 in the SDH group and 641 cm^3 ± 58 in the control group, P = .15; first analysis at 2 years: 720 cm^3 ± 83 in the SDH group and 742 cm^3 ± 70 in the control group, P = .29; second analysis at 1 year: 635 cm^3 ± 64 in the SDH group and 639 cm^3 ± 63 in the control group, P = .73; second analysis at 2 years: 730 cm^3 ± 84 in the SDH group and 744 cm^3 ± 76 in the control group, P = .57). Results from the two sensitivity analyses are summarized in Tables E4–E7 (online).



a. b.

Figure 2: **(a)** Sagittal T1-weighted magnetization-prepared rapid gradient-echo MRI scan in a 15-day-old neonate shows hyperintense subdural hematoma in the posterior fossa (arrow). **(b)** Follow-up sagittal T1-weighted magnetization-prepared rapid gradient-echo MRI scan at 12 months is normal, showing resolution of the previous hematoma.



a. b.

Figure 3: **(a)** Sagittal T1-weighted magnetization-prepared rapid gradient-echo MRI scan in a 14-day-old neonate shows small amounts of hyperintense subdural hematoma in the posterior fossa (arrows). **(b)** Follow-up sagittal T1-weighted magnetization-prepared rapid gradient-echo MRI scan at 2 years is normal, showing no evidence of intercurrent hemorrhage.

Discussion

Recent studies have shown a high frequency of subdural hemorrhage (SDH) in asymptomatic neonates, which follows a characteristic distribution along the occipital lobes, tentorium, or posterior fossa (4–6,19). Although the long-term sequelae of SDH resulting from abusive head trauma have been reported previously (10), no controlled studies using standardized testing have assessed the neurocognitive impact of SDH found with MRI in asymptomatic neonates. Our study showed that cognitive, motor, or language development on standardized testing at age 2 years did not differ between neonates with asymptomatic SDH and neonates in the control group (composite Mullen Scales of Early Learning scores of 115 ± 15 vs 109 ± 16, respectively; $P = .05$). We also found no difference in gray matter volumes between the group with neonatal SDH and control neonates (730 mm^3 ± 85 and 743 mm^3 ± 76, respectively; $P = .70$) and no evidence of rebleeding at 2-year follow-up MRI.

One study following 43 asymptomatic neonates with SDH until age 2 years found no gross motor delays but noted speech delays in six children and possible autism spectrum disorder in one child (4). However, that study had no control group, and only 18 infants were available for follow-up imaging. Other studies observing a high prevalence of SDH in asymptomatic neonates postulated that it likely resolves without complications and may not adversely affect early childhood development (4,20). Our study helps elucidate such premises with objective neurodevelopmental assessment and volumetric MRI data. To our knowledge, this study represents the largest cohort of asymptomatic neonates with SDH and is the first one to compare neurodevelopmental outcomes and gray matter volumes with a matched control group. We previously reported that the total surface area of gray matter in neonates and 1- and 2-year-olds is correlated with MSEL scores at age 2 years, which is consistent with our current findings (11).

With more use of imaging studies in all age groups, incidental SDH will likely be recognized more frequently in asymptomatic or otherwise healthy neonates. Our results should reassure family and clinicians of the benign nature of this form of hemorrhage, which is largely associated with vaginal delivery. In cases of abusive head trauma, our results weaken the argument that birth trauma may predispose individuals to future intracranial hemorrhage; we found no evidence of rebleeding during the 2-year timeframe of our study. Previous studies have shown no evidence of atraumatic rebleeding in infants with incidentally discovered SDH (4,6). Our results validate such findings in a larger cohort with longer MRI follow-up. The only abnormality detected at follow-up MRI of infants with neonatal SDH was one instance of Chiari 1 malformation, which is unlikely to be related to the presence of hemorrhage on the initial examination. Our finding of no differences in the developmental or

neuroimaging follow-up of asymptomatic neonates with SDH is consistent with prior studies (4,6,20).

Our analysis had some limitations. First, these infants did not undergo imaging immediately after birth but within the first 1–5 weeks of life. This timing may overlook a subset of neonatal SDH resolved before the initial imaging examination and may have classified some neonates with resolved hemorrhage within the control group. All visible sequelae of SDH resolving before initial imaging would lend credence to a benign hemorrhage, however. Second, the SDH group had a higher retention rate because parents informed of the SDH during the initial study period were more likely to bring their infants back for follow-up. Nonetheless, this should not have a tangible effect on our analysis. In addition, a higher retention rate in the SDH group might have increased the likelihood of discovering abnormalities. Third, the MSEL is not as sensitive as the standardized tests used in older populations, and these neonates were followed only until age 2 years. Subtle cognitive differences may exist between the SDH and control groups that the MSEL could not demonstrate or had not yet become manifest. We previously found that the 2-year MSEL composite score is moderately predictive of IQ at age 6 years (21). Moreover, infants with neonatal SDH in our study scored higher than control neonates and higher than the national average, suggesting that neonates with SDH would likely not have scored lower than control neonates, regardless of the metric used.

In conclusion, our study builds on the previously established high prevalence of subdural hemorrhage (SDH) in asymptomatic neonates and shows no differences in neurodevelopmental scores and gray matter volumes during a 2-year follow-up. These findings raise the question of whether this pattern of SDH in the neonatal population should be viewed as a pathologic abnormality or considered part of the spectrum of normal postnatal findings. We recommend future longer-term longitudinal studies with more sensitive instruments to address cognitive function in the asymptomatic neonatal population with SDH as they age.

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References

1. Hayashi T, Hashimoto T, Fukuda S, Ohshima Y, Moritaka K. Neonatal subdural hematoma secondary to birth injury. Clinical analysis of 48 survivors. Childs Nerv Syst 1987;3(1):23–29.
2. Williamson WD, Percy AK, Fishman MA, et al. Cerebellar hemorrhage in the term neonate: developmental and neurologic outcome. Pediatr Neurol 1985;1(6):356–360.
3. Bergman I, Bauer RE, Barmada MA, et al. Intracerebral hemorrhage in the full-term neonatal infant. Pediatrics 1985;75(3):488–496.
4. Rooks VJ, Eaton JP, Ruess L, Petermann GW, Keck-Wherley J, Pedersen RC. Prevalence and evolution of intracranial hemorrhage in asymptomatic term infants. AJNR Am J Neuroradiol 2008;29(6):1082–1089.
5. Looney CB, Smith JK, Merck LH, et al. Intracranial hemorrhage in asymptomatic neonates: prevalence on MR images and relationship to obstetric and neonatal risk factors. Radiology 2007;242(2):535–541.
6. Whitby EH, Griffiths PD, Rutter S, et al. Frequency and natural history of subdural haemorrhages in babies and relation to obstetric factors. Lancet 2004;363(9412):846–851.
7. Barnes PD, Krasnokutsky M. Imaging of the central nervous system in suspected or alleged nonaccidental injury, including the mimics. Top Magn Reson Imaging 2007;18(1):53–74.
8. Kemp AM, Jaspan T, Griffiths J, et al. Neuroimaging: what neuroradiological features distinguish abusive from non-abusive head trauma? A systematic review. Arch Dis Child 2011;96(12):1103–1112.
9. Lonergan GJ, Baker AM, Morey MK, Boos SC. From the archives of the AFIP. Child abuse: radiologic-pathologic correlation. RadioGraphics 2003;23(4):811–845.
10. Nuño M, Ugiliweneza B, Zepeda V, et al. Long-term impact of abusive head trauma in young children. Child Abuse Negl 2018;85:39–46.
11. Girault JB, Cornea E, Goldman BD, et al. Cortical structure and cognition in infants and toddlers. Cereb Cortex 2020;30(2):786–800.
12. Gilmore JH, Lin W, Prastawa MW, et al. Regional gray matter growth, sexual dimorphism, and cerebral asymmetry in the neonatal brain. J Neurosci 2007;27(6):1255–1260.
13. Knickmeyer RC, Gouttard S, Kang C, et al. A structural MRI study of human brain development from birth to 2 years. J Neurosci 2008;28(47):12176–12182.
14. Swineford LB, Guthrie W, Thurm A. Convergent and divergent validity of the Mullen Scales of Early Learning in young children with and without autism spectrum disorder. Psychol Assess 2015;27(4):1364–1378.
15. Bishop SL, Guthrie W, Coffing M, Lord C. Convergent validity of the Mullen Scales of Early Learning and the differential ability scales in children with autism spectrum disorders. Am J Intellect Dev Disabil 2011;116(5):331–343.
16. Burns TG, King TZ, Spencer KS. Mullen scales of early learning: the utility in assessing children diagnosed with autism spectrum disorders, cerebral palsy, and epilepsy. Appl Neuropsychol Child 2013;2(1):33–42.
17. Gilmore JH, Zhai G, Wilber K, Smith JK, Lin W, Gerig G. 3 Tesla magnetic resonance imaging of the brain in newborns. Psychiatry Res 2004;132(1):81–85.
18. Knickmeyer RC, Xia K, Lu Z, et al. Impact of demographic and obstetric factors on infant brain volumes: a population neuroscience study. Cereb Cortex 2017;27(12):5616–5625.
19. Sirgiovanni I, Avignone S, Groppo M, et al. Intracranial haemorrhage: an incidental finding at magnetic resonance imaging in a cohort of late preterm and term infants. Pediatr Radiol 2014;44(3):289–296.
20. Holden KR, Titus MO, Van Tassel P. Cranial magnetic resonance imaging examination of normal term neonates: a pilot study. J Child Neurol 1999;14(11):708–710.
21. Girault JB, Langworthy BW, Goldman BD, et al. The predictive value of developmental assessments at 1 and 2 for intelligence quotients at 6. Intelligence 2018;68:58–65.