Correlation Between Retinal Abnormalities and Intracranial Abnormalities in the Shaken Baby Syndrome

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• PURPOSE: To report correlation between retinal and intracranial abnormalities and to evaluate pathogenesis of retinal hemorrhages in the shaken baby syndrome (SBS).
• DESIGN: Observational case series.
• METHODS: Seventy-five children with apparent nonaccidental head trauma consistent with SBS had complete physical examination, complete ophthalmologic examination, neuroimaging by CT or MRI, or both, and skeletal radiographic survey. In this retrospective review, ophthalmoscopic and intracranial abnormalities were correlated.
• RESULTS: The age of patients ranged from 2 to 48 months (mean – SD, 10.6 ± 10.4 months). Neuroimaging was abnormal in all 75 cases. Findings included subdural hematoma (70 children, 93%), cerebral edema (33 children, 44%), subarachnoid hemorrhage (12 children, 16%), vascular infarction (nine children, 12%), intraparenchymal blood (six children, 8%), parenchymal contusion (six children, 8%), and epidural hemorrhage (one child, 1%). Sixty-four (64/75, 85%) children had retinal abnormalities, mostly (53/64, 82%) confluent multiple hemorrhages that were subretinal, intraretinal, and preretinal in 47/64 (74%) and bilateral in 52/64 (81%). No association was found between anatomic site (left, right, or bilateral) of intracranial and retinal findings (McNemar test kappa = −0.026–0.106) or between any of the intracranial findings mentioned above and the following retinal findings: normal or abnormal retinal examination, multiple (>10) or few retinal hemorrhages (≤10), symmetric or asymmetric retinal findings, or retinoschisis (kappa = −0.127–0.104). Signs of possible increased intracranial pressure were not correlated with any retinal abnormality (kappa = −0.03–0.073). There was no correlation between evidence of impact trauma to the head and retinal hemorrhages (kappa = 0.058). Total Cranial Trauma Score and Total Retinal Hemorrhage Score, both indicating the severity of injury, were correlated (P = .032).
• CONCLUSIONS: Our study supports previous observations that the severity of retinal and intracranial injury is correlated in SBS. We cannot support the suggestions that in most children with SBS retinal bleeding is caused by sustained elevated intracranial, elevated intrathoracic pressure, direct tracking of blood from the intracranial space, or direct impact trauma. The correlation in severity of both eye and head findings may suggest, however, that retinal abnormalities are the result of mechanical shaking forces. (Am J Ophthalmol 2002;134:354–359.

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FIRST DESCRIBED BY CAFFEY IN THE 1970S AS "WHIPLASH shaken infant syndrome," the shaken baby syndrome (SBS) is a form of child abuse caused by violent shaking of an infant or young child, usually manifested as some combination of brain, ocular, and skeletal injury.[1] Infants and young children are particularly vulnerable to violent shaking because of their relatively large head and weak cervical musculature. These factors, together with incompletely fused sutures and relatively large volumes of cerebrospinal fluid, allow for greater movement within the cranial vault, resulting in potentially severe damage to the immature incompletely myelinated brain.

Retinal hemorrhages may be found in 50% to 100% of shaken infants[2,3] and, when accompanied by other evidence of nonaccidental head trauma, are regarded by many as diagnostic for child abuse.[2,4,5] Hemorrhages may be subretinal, intraretinal, or preretinal and vary enormously in both size and severity from a few small flame-shaped nerve fiber layer hemorrhages to extensive hemorrhages, obscuring underlying retinal structures.[2]

Different theories have been proposed to explain the

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development of retinal hemorrhages including an increase in ocular arterial and/or venous pressure secondary to either intracranial hemorrhage, elevated intracranial pressure, increased intrathoracic pressure (caused by the perpetrator's forceful compression of the child's chest), and/or strangulation occluding one or both of the carotid arteries.[2,6–8] Another theory regards the eye and its orbit as a single unit, damaged directly by the mechanical process of shaking, with resultant damage to the retina and its blood vessels.[2,9] Others have postulated a role for direct tracking of blood from the intracranial subdural or subarachnoid spaces, through the optic nerve sheath either directly into the eye or causing rupture of bridging vessels within the optic nerve sheath.[2,10] Eye and head injury may occur with or without blunt impact injury to the head.[2,11]

Correlation between the severity of ocular and intracranial injury has been described[12] and the presence of retinal hemorrhage was suggested as a predictor for more severe neurologic damage.[13] Although correlation between the anatomic site of intracranial and retinal lesion has been suggested, the evidence for this is not conclusive, largely due to small sample sizes.[14–16] The goal of this study was to examine for correlations between intracranial and intraocular findings in SBS. Finding correlation, or the absence of correlation, may have important forensic implications and might help in better understanding the mechanisms by which intraocular manifestations of this syndrome occur.

METHODS

WE CONDUCTED AN OBSERVATIONAL CASE SERIES OF 75 children representing all children with apparent nonaccidental head trauma consistent with SBS treated at The Hospital for Sick Children, Toronto, Canada, between January 1993 and December 1999. The study was approved by the Hospital Research Ethics Board. The diagnosis was made by the multidisciplinary Suspected Child Abuse and Neglect Program based on evidence of apparent nonaccidental head trauma consistent with SBS with at least two of the following criteria: (1) abnormal findings on neuroimaging, (2) skeletal injury, (3) ocular injury, and (4) history of child abuse that included shaking with or without blunt head trauma or an inadequate history to explain the observed injuries. We excluded children that were believed to suffer from other forms of child abuse or had a history of shaking by a caretaker, but did not exhibit the characteristic signs of SBS.

All children underwent complete physical examination, neuroimaging by computed tomography (CT) or magnetic resonance imaging (MRI) or both methods, and skeletal radiographic survey. All victims who died underwent a complete autopsy examination including ocular histologic examination. Complete ophthalmologic examinations were done by one of us (A.V.L.) and included dilated indirect ophthalmoscopy. We collected data regarding intracranial findings by reviewing the CT/MRI films and pathologic examination records. Ophthalmologic findings were recorded from the written record and from review of fundus photographs when available.

TABLE 1. Retinal Hemorrhage Score*

Number of hemorrhages	
≤10	1
>10	2
Extent of hemorrhages	
One zone	1
Two zones	2
All zones	3
Type of hemorrhages	
Preretinal	1
Intraretinal	1
Subretinal	1

*Each eye scored separately. Final score is sum of scores for both eyes.

Retinal hemorrhages were graded using the retinopathy of prematurity retinal zone method (zones I, II, and III).[17] We designed a retinal hemorrhage score and a cranial trauma score (Tables 1 and 2). Each eye and cerebral hemisphere was scored separately and a final score was obtained by adding the scores for both sides in each patient.

Correlations between retinal and intracranial abnormalities were analyzed using the McNemar test. The Spearman correlation test for nonparametric data were utilized for analysis for correlation between the retinal hemorrhage score and the cranial trauma score.

RESULTS

OUR STUDY GROUP INCLUDED 75 CHILDREN DIAGNOSED AS victims of SBS. Average age at the time of diagnosis was (mean ± SD) 10.6 ± 10.4 months (range 2–48 months). Forty-six (46/75, 61%) were males. In only 37 cases (37/75, 49%) was any head trauma reported on admission. Signs of direct impact head injury (scalp swelling and/or skull fracture on neuroimaging or postmortem evidence) were noted in 30 children (40/75, 40%). Table 3 provides neuroimaging findings (CT, MRI, or both) on admission. Neuroimaging was abnormal in all cases. The most common finding was subdural hematoma (SDH), found in 93% (70/75) of the children. Subdural hematoma was bilateral in 48 of the children (48/70, 69% of cases). Cerebral edema (33/75, 44%) and subarachnoid hemorrhage (12/75, 16%) were also relatively frequent: 67% (22/33) and 42% (5/12) of them were bilateral, respectively. Other less frequent findings including parenchymal contusion, epi-

TABLE 2. Cranial Trauma Score*

Direct impact trauma	
Scalp swelling	1
Skull fracture	1
Subdural hemorrhage	
Small (=5 cm)	1
Widespread (>5 cm)	2
Epidural hemorrhage	
Small (=5 cm)	1
Widespread (>5 cm)	2
Subarachnoid hemorrhage	
1–2 cistern	1
More than 2 cisterns	2
Intraparenchymal blood	
Small (=3 cm)	1
Widespread (>3 cm)	2
Cerebral edema	
Focal around hemorrhage	1
Entire hemisphere	2
Causing midline shift	3
Vascular infarction	
Localized (= one lobe)	1
Widespread (>one lobe)	2
Parenchymal contusion	
Localized (= one lobe)	1
Widespread (>one lobe)	2

*Each hemisphere was scored separately. Final score is the sum of scores for both hemispheres.

TABLE 3. Cranial CT and/or MRI Abnormalities*

	Total	Unilateral
Subdural hemorrhage	70 (93%)	22 (29%)
Cerebral edema	33 (44%)	8 (11%)
External scalp swelling	17 (23%)	12 (26%)
Skull fracture	18 (24%)	10 (13%)
Subarachnoid hemorrhage	12 (16%)	5 (9%)
Vascular infarction	9 (12%)	3 (4%)
Parenchymal contusion	6 (8%)	5 (9%)
Intraparenchymal blood	6 (8%)	5 (7%)
Intraventricular blood	2 (3%)	1 (2%)
Epidural hemorrhage	1 (1%)	1 (2%)
Total number of patients	75	

*Number in parentheses is percentage of the total number of patients. Some children have more than one abnormality.

dural hemorrhage, and vascular infarction were usually focal and mostly unilateral.

Sixty-four (64/75, 85%) children had retinal abnormalities (Table 4). Most of these children (53/64, 83%) had multiple confluent hemorrhages, 46 (46/53, 87%) of which were bilateral. Hemorrhages were usually found at three levels: subretinal, intraretinal, and preretinal (47/64, 74%). Only ten (10/64, 16%) patients had ten or less scattered hemorrhages. Traumatic retinoschisis was seen in 24 patients (24/75, 32%), and was more often unilateral (15/24, 63%). Papilledema was relatively rare (4/75, 5%). Retinal detachment was not seen.

TABLE 4. Retinal Abnormalities in the Study Group (n=75)*

	Total (Bilateral and Unilateral)	Unilateral
Retinal hemorrhages	64 (84%)	12 (16%)
Asymmetric bilateral hemorrhages	21 (28%)	
Multiple hemorrhages (>10)	53 (71%)	6 (8%)
Few hemorrhages (=10)	10 (13%)	4 (5%)
Hemorrhages in zone I only	15 (20%)	4 (5%)
Hemorrhages in zones I–III	46 (61%)	6 (8%)
Sub, intra, and preretinal hemorrhages	44 (59%)	4 (7%)
Intraretinal hemorrhages only	5 (7%)	2 (3%)
Preretinal hemorrhages only	5 (7%)	2 (3%)
Pre and intraretinal hemorrhages	6 (8%)	4 (5%)
Retinoschisis	24 (32%)	15 (20%)
Papilledema	4 (5%)	1 (1%)
Retinal detachment	0	0
Total number of patients	75	

*Numbers in parentheses are percent from total number of patients. Some children had more than one abnormality. Retinal zones are according to the retinopathy of prematurity staging system.[17]

In each patient, we compared the laterality (left, right, or bilateral) of each of the head findings listed in Table 3 with the laterality of the retinal findings. No association was found (kappa = −0.026–0.106). Thirty-nine patients (39/75, 52%) were found to have asymmetric cranial findings (18 strictly unilateral findings and 21 bilateral, but asymmetric cranial findings). Retinal findings were asymmetric in 21 cases (21/64, 33%, strictly unilateral in 12 cases and bilateral, but asymmetric in nine). When evaluating the overall tendency towards unilaterality or asymmetry there was no association between the intracranial and intraocular findings. Moreover, there was no association between the actual side (right or left) of findings (kappa = −0.040 and −0.099, respectively). Patients who had unilateral scalp swelling and/or skull fracture, or unilateral subdural hemorrhage, cerebral edema, or subarachnoid hemorrhage, did not have a greater chance of having unilateral retinal findings on the ipsilateral or contralateral side (kappa = −0.043–0.059).

No correlation was found between the occurrence of any of the intracranial findings described in Table 3, including signs of direct impact trauma, with any of the following retinal findings: normal or abnormal retinal examination, multiple hemorrhages (>10) or few hemorrhages (<10), symmetric or asymmetric retinal findings, or retinoschisis (kappa values between −0.127–0.104).

We identified 29 patients who had one or more of the following signs of possible increased intracranial pressure: papilledema, widespread bilateral cerebral edema, unilateral edema with a midline shift, and large intracranial hemorrhage that compressed the brain tissue. We could not find any correlation between these findings and any of the retinal findings (kappa = −0.030–0.073). Total cranial trauma score and total retinal hemorrhage score in each patient were found to be correlated ($P = .032$).

When evaluating the results of the skeletal surveys that were completed in all patients, we identified eight children (10%) who suffered from rib fractures. Five of these patients had bilateral retinal hemorrhages, two had normal eye examination, and one had unilateral retinal hemorrhages. Due to the small number of children with fractures, further statistical analysis could not be performed.

DISCUSSION

OUR STUDY DESCRIBES A LARGE POPULATION OF CHILDREN diagnosed with SBS in which both eye and head findings were compared for correlation. The 85% incidence of retinal hemorrhages found in our study group is well within the range of 50% to 100% reported by various authors.[2,18] Intracranial findings in our study were also compatible with previous reports [2,14,18,19]

Retinal hemorrhage score (Table 1) and cranial trauma score (Table 2) were found to be correlated in our study. This supports the reports by Wilkinson and associates[13] and Kivlin and colleagues[18] that found the same correlation clinically. Green and associates found a comparable correlation in autopsies.[12] This correlation may be important for forensic evaluation of SBS victims. We believe this evidence shows that more severe mechanical injury caused by vigorous shaking will cause more severe intracranial and ocular damage. We also examined our data to test various theories of retinal hemorrhage pathogenesis.

• **DIRECT TRACKING OF BLOOD THROUGH THE OPTIC NERVE:** Unilateral retinal hemorrhages are well recognized in SBS.[2,18,20] Recently, Paviglianiti and associates[15] and Clarke and associates[16] reported SBS patients that had asymmetric ipsilateral retinal and intracranial bleedings. This might be interpreted as bolstering the theory that retinal hemorrhages result from direct tracking of blood from the intracranial space into the subdural or subarachnoid spaces of the optic nerve, with secondary direct tracking of blood into the eye or compression of the central retinal vein or rupture of bridging vessels in the optic nerve sheath.[21] However, the observations in their small samples could easily have occurred by chance alone.

Although a large proportion of both head and eye injuries in our study were unilateral/asymmetric (52% and 33%, respectively), no correlation was found between the presence of unilaterality/asymmetry in the eye and head regardless of side or between the sides of bleeding. Budenz and associates also found no correlation.[14] Moreover, two patients in our study had bilateral subdural or subarachnoid optic nerve sheath hemorrhages on postmortem examination. One of these patients had a normal eye examination and the other had only unilateral retinal hemorrhages. Although we cannot rule out the possibility that direct tracking of blood from the intracranial space plays a role in some individual cases, our data do not support this process in the pathogenesis of retinal hemorrhages in SBS.

• **INCREASED INTRACRANIAL PRESSURE:** Increased intracranial pressure as a cause for intraocular bleeding has been considered both for isolated cases of retinal hemorrhages and as part of the pathophysiology of Terson syndrome, which combines intracranial and intraocular bleeding. It is suggested that sudden effusion of the colony-stimulating factor (CSF) into the optic canal's subarachnoid space causes dilation of the optic nerve sheath, rupturing the bridging vessels within it. This, in turn, could compress and obstruct the retinochoroidal anastomoses and/or central retinal vein resulting in retinal bleeding.[22]

Although intracranial pressure was not measured in all of our patients, it is reasonable to assume that patients with papilledema, widespread cerebral edema, or an intracranial hemorrhage causing a midline shift had elevated intracranial pressure. Patients with these presumed signs of elevated intracranial pressure in our study did not differ in type, extent, or frequency of retinal findings when compared with the rest of the patients. Only a small minority of our patients had papilledema (four patients, 5%). This exact rate was also reported by Kivlin and associates.[18] To our knowledge retinal hemorrhage due to even severe elevated intracranial pressure in children who have not been shaken has never been reported as severe and extensive.

• **INCREASED INTRATHORACIC PRESSURE:** Increased intrathoracic pressure is the presumed cause for retinal bleeding in Purtscher retinopathy.[23] Although retinal findings in SBS usually lack the characteristic large white patches seen in Purtscher retinopathy, elevated intrathoracic pressure has been mentioned as a possible cause for retinal bleedings in SBS.[24] Eight children (10%) in our study were diagnosed with rib fractures. One can speculate that compressing the chest to such extent would indeed cause significantly increased intrathoracic pressure. Although all eight children had subdural hemorrhage and other intracranial findings, only six had retinal hemorrhages and none had the characteristic findings of Purtscher retinopathy. In addition, the absence of rib fracture in all other patients with intraocular hemorrhage may imply that elevated intrathoracic pressure has a limited role in the pathogenesis of retinal hemorrhages in

SBS. Unfortunately, the low number of children with rib fracture prevented us from doing further statistical analysis.

• **IMPACT HEAD TRAUMA:** Several researchers have suggested that direct impact trauma is necessary to generate severe injury in SBS[25,26] and an experimental model was advanced to demonstrate this concept.[27] We have found no difference in the extent, type, or frequency of retinal bleeding between patients who exhibited signs of direct impact trauma vs those who did not. Alexander and associates, in their study on 24 abused infants, also failed to find correlation between direct impact trauma and retinal bleeding.[28]

• **MECHANICAL INJURY CAUSED BY SHAKING:** There are suggestions in the literature that retinal injury may be related to the unique direct effects of shaking on the eye and the orbit.[2,29,30] The repetitive shaking movement may produce shearing forces that disrupt blood vessel integrity.[2,31] Injury to other orbital structures, such as the sympathetic and parasympathetic neurons, might also disrupt retinal vasculature autoregulation.[2,32] The effect of shaking directly on the intraocular contents may be particularly important in the infant eye with its firm attachments between the vitreous and the major retinal blood vessels, the posterior pole, and the ora serrata.[33] In fact, it is well accepted today that retinoschisis in SBS is created by the shearing forces these attachments exert on the retina.[2,34]

Our positive finding of correlation between retinal hemorrhages and intracranial injury may support the theory that mechanical shaking and its direct effect on the globe and orbit has a major role in the pathogenesis of SBS. This assumption is supported by our inability to confirm other theories for the pathogenesis of retinal hemorrhages in SBS. Further laboratory and clinical studies are underway at our center to test this theory.

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