Retinal Findings in Children with Intracranial Hemorrhage

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Purpose: To identify the incidence of Terson's syndrome in children.
Design: Prospective, observational case series.
Participants: Fifty-seven consecutive children with known intracranial hemorrhage from nonabuse causes.
Methods: Dilated fundus examination to detect intraretinal hemorrhages or other abnormalities.
Main Outcome Measures: Presence or absence of intraretinal hemorrhages or other abnormalities.
Results: Fifty-five patients (96%) had no evidence of intraretinal or vitreous hemorrhage. Two patients had abnormal retinal examinations. One patient had a single dot hemorrhage associated with presumed infectious white retinal lesions. The second patient had three flame and two deeper dot intraretinal hemorrhages after a motor vehicle accident (1.5% incidence of retinal hemorrhage).
Conclusions: Retinal hemorrhage is uncommon in children with intracranial hemorrhage not resulting from shaken baby syndrome. The maximal incidence of intraretinal hemorrhage in children with nonabuse intracranial hemorrhage is 8%. *Ophthalmology 2002;109:1472–1476 © 2002 by the American Academy of Ophthalmology.*

Terson's first reported the association of vitreous hemorrhage after intracranial hemorrhage in an adult in 1900.[1] The condition was thought to be rare, with only 16 cases reported in the literature by 1952, all in adults. However, a 1954 retrospective review showed a 20% incidence in 225 adult patients with subarachnoid hemorrhage (SAH).[2] Other studies and case reports conducted over the next several decades confirmed that the syndrome was more common in adults than initially suspected.[3–6] The incidence is now estimated at 16% to 27%, based on two prospective studies of adults with SAH.[7,8] Today, the definition has been expanded to include all forms of intracranial bleeding with vitreous or retinal hemorrhage, or both.[9–11] Of the 16 affected patients examined in these two studies, 15 cases were the result of intracranial aneurysms and one was the result of trauma.

To our knowledge, there are no studies prospectively evaluating the occurrence of Terson's syndrome in children. Two observational case report articles describe retinal hemorrhages in eight eyes of six children with intracranial hemorrhage.[5,12] Although there were no investigations reported to rule out nonaccidental trauma in these six children, child abuse was likely in five (F. Kuhn, personal communication, April 1998).[10] In the sixth child, the cause of the intracranial hemorrhage remains unknown. It may have been the result of infection or hyponatremia, which may have contributed independently to the retinal findings.[5]

Several studies have examined the incidence of retinal hemorrhages after pediatric head trauma with and without intracranial hemorrhages.[10,13] These studies consistently indicate that retinal hemorrhages are rare even in severe accidental pediatric head trauma, but the sample populations often contain a small or unknown number of children with intracranial hemorrhage. Based on these studies, the incidence of Terson's syndrome in children cannot be assessed fully. Intracranial hemorrhage occurs in 5% to 23% of pediatric victims of nonaccidental trauma.[14–16] In cases where abuse is considered, skeptics of that diagnosis may argue that observed intraretinal hemorrhages could have been caused independently by the intracranial hemorrhage (Terson's syndrome), presumably from causes other than possible abuse.

We undertook a prospective study to examine children with intracranial blood from any cause other than child abuse to quantify the association with intraretinal hemorrhage.

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Materials and Methods

All children 6 weeks of age or older with intracranial hemorrhage documented by computerized tomography (CT) from any cause other than abuse were considered eligible for the study. Exclusion criteria were age less than 6 weeks or duration of intracranial hemorrhage more than 72 hours. The former was designed to eliminate the possible confounding effect of retinal hemorrhage resulting from birth.[10,17] The latter was to avoid accidental exclusion of a patient whose hemorrhages had cleared quickly or who had another intervening cause. All cases of suspected nonaccidental trauma were excluded even if the multidisciplinary child abuse team later cleared the case. None of these cleared cases had intraretinal hemorrhages.

The study began on October 1, 1996 and ended on May 30, 1998. Patients were identified by daily telephone contact with the neurosurgical, critical care, and neuroradiology services. An ophthalmologic examination, including indirect ophthalmoscopy after pharmacologic dilation of both pupils was performed on each child by at least one author (SS, PM, JRB, AVL). One author (AVL) reexamined all patients whose findings were not normal. Our primary outcome was the presence or absence of intraretinal hemorrhage. Secondary outcome measures were the presence of any other abnormalities on ophthalmoscopy. An ophthalmic examination was established as routine care for patients with intracranial hemorrhage in our hospital because of the frequent association of eye problems with severe head trauma, including papilledema, pupillary abnormalities, orbital fracture, and direct globe trauma. As a result, approval of the research ethics board was not required.

A grading scale for intracranial hemorrhages was created for this study by the investigating neuroradiologists (DCA, ES), addressing both location and amount. The five locations were epidural, subdural, subarachnoid, intraventricular, and intraparenchymal. Epidural and parenchymal hemorrhages were measured using the greatest two dimensions on neuroimaging. Subdural hemorrhages were measured based on the maximum thickness of the blood from the inner wall of the calvarium. Subarachnoid blood was quantified by counting the number of involved cisterns. Intraventricular hemorrhage was graded as filling less than 50% or filling 50% or more of the ventricular volume.

The term *intraretinal hemorrhage* is used to describe specific findings in our study where hemorrhage is seen within retinal tissues. On the other hand, the less specific term retinal hemorrhages is used when describing the findings referenced in other studies, if that study does not otherwise fully clarify the exact location of hemorrhange within the retina.

Statistical analysis consisted of calculating the confidence interval for the rate of intraretinal hemorrhages in the total study population. Because the number of observed events was more than zero but still too small to allow a normal approximation to exact binomial probabilities, we solved for the confidence interval by using nonlinear equations according to the methods described by Jovanovic and Zalenski.[18]

Results

Fifty-seven patients met the inclusion criteria. The patients were 5 months to 16.1 years of age (mean, 10.3 years). Seventeen children (30%) were 4 years of age or younger. There were 30 males and 27 females.

Table 1 shows the mechanism for intracranial hemorrhage. Twenty-seven (47%) were the result of trauma. Of these, 15 (55%) were motor vehicle accidents. Sports or hobbies accounted for 6 (22%), whereas falls from a height made up the remaining 6 trauma cases (22%). Our sample size prevented us from achieving the necessary power to perform subcategory correlations such as age versus height of fall. Twenty-four patients (42%) were entered into the study after surgery, with tumor resection accounting for 20 of these (83%). Seizure control and surgery for a ventriculoperitoneal shunt accounted for the remaining 4 cases (17%).

Table 1. Mechanism of Intracranial Hemorrhage

- Trauma (n = 27)
  - Motor vehicle accident (n = 15)
  - Sport or hobby (n = 6)
  - Falls (n = 6)
    - Height
      - Five story building
      - One flight of stairs
      - Eight-foot slide
      - Six-foot ravine
      - Fall from mother's arms
      - Fall from stroller while reaching for elevator button
- Surgery (n = 24)
  - Medulloblastoma (n = 7)
  - Astrocytoma (n = 8)
  - Other tumors (n = 5)
  - Seizure control (n = 3)
  - Ventriculoperitoneal shunt (n = 1)
- Other (n = 6)
  - Arteriovenous malformation, cavernous sinus fistula, subacute bacterial endocarditis with subarachnoid hemorrhage resulting from mycotic aneurysm, spontaneous hemorrhage after heparinization in the course of treatment for the complications of immunoglobulin A nephropathy, vasculitis, idiopathic spontaneous hemorrhage

Table 2 summarizes the CT findings in our 57 patients. Several of the patients had more than one finding on CT. Eighteen patients (32%) had subdural hemorrhages ranging in size from 1 to 15 mm. There were 7 patients (12%) with epidural hemorrhages and 7 (12%) with subarachnoid hemorrhages. Thirty-three patients (57%) had intraparenchymal hemorrhages ranging in size from 1 to 3300 mm^2. Twenty-two patients (38%) had intraventricular hemorrhage: 14 with less than 50% of the ventricles filled with blood and 8 with more than 50% filled. Thirty-seven patients (65%) had either a skull fracture (n = 13) or craniotomy (n = 24).

On ophthalmic examination, 55 patients (96%) had normal retinal examinations without evidence of vitreous or intraretinal hemorrhage (Table 3). The two remaining patients with retinal findings are described below.

Ten patients (18%) had papilledema. None had intraretinal hemorrhages. All were patients with tumors, and in six of the cases, the papilledema was noted before surgery. Not all patients with elevated intracranial pressure as determined by neuroradio-

Table 2. Intracranial Findings Determined by Neuroimaging (n = 57)

Finding	Frequency (%)	Amount
Subdural hemorrhage	18 (32%)	1–15 mm
Epidural hemorrhage	7 (12%)	160–1200 mm^2
Subarachnoid hemorrhage	7 (12%)	1 cistern, n = 2; >1 cistern, n = 3; widespread, n = 2
Parenchymal hemorrhage	33 (58%)	1–3300 mm^2
Intraventricular hemorrhage	22 (39%)	<50%, 14 >50%, 8
Skull fracture	13 (22%)	
Craniotomy	24 (42%)	

Table 3. Ocular Findings

Finding	N (%)
No retinal hemorrhage	55 (97%)
Papilledema	10 (18%)
Other	
Retinal hemorrhages	1 (1.5%)
White spots	1 (1.5%)

logic examination or intracranial bolt monitoring had papilledema. Each of these surgical patients had no evidence of intracranial hemorrhage on preoperative CT.

Patient 1

A 7-year-old passenger in a motor vehicle accident was thrown approximately 100 feet and had a Glascow Coma Scale of 3. A CT scan showed midline intracranial shift with left subdural, intraparenchymal, and intraventricular hemorrhages. His right retina had three superficial flame-shaped nerve fiber layer hemorrhages and two deeper intraretinal hemorrhages, all less than 500 μm, located along the vascular arcades. There was no hemorrhage in the macula. The left fundus examination was normal.

Patient 2

An 8-year-old female with a 6-week history of immunoglobulin A nephropathy had a tonic-clonic seizure at home. Her CT revealed cortical infarcts without evidence of hemorrhage. She was hospitalized and was administered intravenous heparin and antibiotics for group B sepsis. Five days later, a 50- × 20-mm intraparenchymal thalamic hemorrhage developed. She also had intraventricular hemorrhages of more than 50% of ventricular volume involving the third, fourth, left, and right ventricles. Her ophthalmic examination revealed bilateral small white retinal lesions consistent with infection. One eye had a single dot hemorrhage in one of the white lesions.

From an incidence of 1 in 57 (1.5%), we calculated a maximal predicted incidence for intraretinal hemorrhage at 8% using an upper confidence interval of 95%. Although two patients had retinal hemorrhages, only patient 1 was not attributable to a cause other than intracranial hemorrhage. Patient 2 had hemorrhage resulting from retinal infection and was excluded from our calculations.

Discussion

We undertook this prospective study to report retinal findings in children with intracranial blood from any cause other than abuse. Although prospective studies in adults have demonstrated Terson's syndrome at a rate of 16% to 27%,[7,8,12] our study suggests that the incidence is much lower in children.

Shaking is a violent act. It is now believed that shaking alone can cause most of the intraretinal and intracranial findings.[10,19,20] Death can also occur from shaking alone. Shaken baby syndrome is characterized by the triad of skeletal injury, intracranial hemorrhage, retinal hemorrhage, or a combination thereof, usually in the absence of external signs of injury.[10,21] Some authors believe that impact must accompany the shaking to cause the syndrome,[22] but this is widely disputed.[10,19,20,23]

Retinal hemorrhages are observed in 50% to 100% of shaken babies.[10,24,25] The exact pathophysiologic mechanisms of many of these hemorrhages are unknown. Retinoschisis, when present, is the result of shearing forces on the macular retina through its connections with the vitreous, which are particularly strong in children.[10,26] Purtscher's retinopathy may also play a role in some children, because a rise in intrathoracic pressure would be expected especially when the infant's torso is grasped to such a degree that rib fractures occur. This pressure rise may be transmitted to the eye, causing disruption of venous return.[10,27] The optic nerve sheath theoretically may be affected by intracranial pressure or hemorrhage, as in Terson's syndrome. Repetitive shaking may also induce unique shearing trauma to the orbital contents and retina.[10]

We are unaware of any studies of Terson's syndrome specifically in children. To understand the pathophysiologic mechanism of the disorder, we must rely on several adult studies with proposed theories.[7] One early theory suggested that hemorrhage could track from the subarachnoid space of the optic nerve sheath directly to the retina or vitreous.[28] This was refuted by those who believe that no such anatomic continuity exists.[3,29,30] Additionally, there have been adults with intraocular hemorrhage with raised intracranial pressure (ICP) in the absence of SAH, thus implying that blood tracking is not required.[7] A second theory is that compression of the central retinal vein resulting from hemorrhage in the optic nerve sheath could cause decreased venous return.[31] This mechanism has been challenged by Heyreh's[32] experiments in primates, which showed that occlusion of the central retinal vein at its exit from the optic nerve sheath did not cause retinal or vitreous hemorrhage. In addition, the pattern of retinal hemorrhage in Terson's syndrome does not approximate that seen in retinal vein occlusions. The third theory was that a rapid increase in ICP causes retinal venous stasis resulting from decreased return to the cavernous sinus from the veins that drain the globe.[29] However, there is another outlet for drainage of the globe: the facial and pterygoid venous systems, which are not affected by increased ICP.[2]

The most likely theory was proposed in 1954 and states that increased ICP results in excess accumulation of cerebrospinal fluid within the nerve sheath communication of the subarachnoid space.[2,33] As a result, compression of the retrobulbar portion of the optic nerve can obstruct the retinochoroidal anastomoses and, perhaps to some degree, the central retinal vein as well. This theoretically would result in a decrease in venous drainage that would cause stasis and hemorrhage. Because the retinochoroidal anastomoses are located near the optic nerve and scleral junction anterior or "at the anterior limitation of" the subarachnoid space, the compression would occur only with large amounts of nerve sheath dilatation. As a result, only certain patients would experience intraocular hemorrhage.[7] Stretching of the optic nerve sheath may also shear bridging vessels that go from the dura to the pia mater.[6]

Why Terson's syndrome occurs less frequently in children is unknown. Our patients did have a wide range of mechanisms for intracranial hemorrhage, with trauma being the primary cause (47%). Traditionally, studies of adult

Terson's syndrome have included arteriovenous malformation (AVM) as a primary cause: a rare entity in childhood. Although our series had only one patient with an AVM, review of the literature also reveals that retinal hemorrhages rarely occur in children with a ruptured aneurysm.[10,34] Only 12% of our patients had SAH, another of the more common causes of Terson's syndrome in adults. None of these 12% had intraretinal hemorrhages. The incidence of SAH in children is very low with the exception of shaken baby syndrome. To direct attention specifically to the incidence of intraretinal hemorrhage in children with AVM or SAH would require a very large multicenter trial. However, if childhood Terson's syndrome was indeed more common after SAH, this should be reflected in the incidence of intraretinal hemorrhage after accidental head trauma, one of the major causes of pediatric SAH. In fact, many studies have shown that the incidence of hemorrhage after accidental head trauma is less than 3%.[10] Intraretinal hemorrhage almost always requires severe life-threatening impact injury that rarely raises concerns of child abuse, such as the motor vehicle injury in our child with intraretinal hemorrhage.[10,13] In addition, there is evidence to suggest that the very few hemorrhages seen in this child's retina, characteristic of most children who show the uncommon retinal hemorrhage after accidental trauma, may actually be the result of the extreme acceleration–deceleration shearing stress rather than the intracranial hemorrhage.[10]

Raised ICP is also common in the adult studies. Five of 21 of our patients (24%) in whom the data were available had raised ICP, several high enough to require ICP bolt monitoring. It is likely that we would find that even more of our patients had elevated ICP should the information be available to us retrospectively. Yet once again, a review of the literature shows that intraretinal hemorrhage is rare in children with raised ICP.[10]

Perhaps there is better autoregulation of retinal vasculature in children as compared with adults. Children's vessels may be more resilient in other undefined ways, offering some protection against Terson's syndrome. In support of this concept is the markedly decreased incidence of diabetic, sickle cell, and hypertensive retinopathy in children, entities that are very common in adulthood.[10,35,36] One group has proposed that the mechanism for the differences in children versus adults is that of hormonal influences.[35] If the pediatric central retinal vein and retinochoroidal anastomoses are better able to withstand the external pressures proposed in the pathophysiologic theories of Terson's syndrome, then the lesser rate of acquiring intraretinal hemorrhages would be expected. Another possibility is that the potential communication between the optic nerve sheath and intracranial space is more restrictive in young children. To our knowledge, this has not been investigated, but it may represent an opportunity for future investigation.

Less than one third of our patients were in the age range in which shaken baby syndrome occurs. If one considers that most severely injured shaken babies are younger than 2 years old, then our comparative population is even smaller. If the vasculature of children is particularly protective against the influence of intracranial hemorrhage, then one may expect that the youngest of children would be the most protected. Because of the low frequency of intracranial hemorrhage in infants and very young children, a multicentered approach would be required to determine definitively the incidence of Terson's syndrome. Although some caution should be exercised when extrapolating our findings to those of children in the shaken baby syndrome age range, our results support recent studies in the literature: severe intraretinal hemorrhages are caused by shaking.[10]

Our study suggests that intraretinal hemorrhages in children with intracranial hemorrhage from causes other than shaken baby syndrome would be expected in less than 8% of cases. A larger sample size would be needed to further reduce the 95% confidence interval for the 1.5% incidence observed in our study group. The mere presence of an intracranial hemorrhage will usually not explain the findings of intraretinal hemorrhages in a child. When the clinical picture fits with shaken baby syndrome, one may be hesitant to explain intraretinal hemorrhage solely by the concurrent finding of intracranial hemorrhage ascribed to another cause, particularly if the hemorrhagic retinopathy is extensive. Rather, the presence of intraretinal hemorrhage with intracranial hemorrhage without another clear explanation in an infant or young child should lead one to consider shaken baby syndrome as a primary diagnosis.

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