

Retinal Hemorrhages in Children Following Fatal Motor Vehicle Crashes

A Case Series

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Objective: To demonstrate the severity of ocular findings in young children who died of injuries due to motor vehicle crashes.

Methods: Case series of 10 children younger than 3 years who were fatally injured in motor vehicle crashes between January 1, 1994, and December 31, 2002. All children underwent autopsy that included eye examination. All available medical and autopsy records, pathology slides and photographs, and police and traffic department reports were reviewed for each case.

Results: Eight patients had retinal hemorrhages, which extended into the periphery in 13 eyes and were bilat-

eral in 7 patients. Three patients had elevated circular retinal folds. Six patients had hemorrhages below the internal limiting membrane, but no patients had deeper splitting of the retina. Nine patients had optic nerve sheath hemorrhages.

Conclusion: The association of extensive, sometimes severe, ocular hemorrhages with fatal accidental trauma, compared with previous reports of accidental trauma with no or few hemorrhages, indicates the severity of injury required to cause hemorrhages of this magnitude.

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INNUMERABLE SEVERE, MULTILAYERED retinal hemorrhages that extend to the far periphery are commonly associated with non-accidental head injury in infants and young children.^{1,2}

While there are many other causes of retinal hemorrhages, few result in hemorrhages of this severity. When a child is assessed for abusive neurotrauma, it is important to note the size, shape, number, and distribution of the hemorrhages. In addition, it is helpful to be aware of the types of force required to produce hemorrhages with a similar distribution and degree of severity that result from accidental traumatic mechanisms where the injury is known.

METHODS

The cases reported herein were collected by the Department of Ophthalmology at the Medical College of Wisconsin from January 1, 1994, through December 31, 2002, and were taken from autopsies performed by the Medical Examiner of Milwaukee County on children younger than 3 years who died in motor vehicle crashes (MVCs). Autopsies, which may include the eyes, are performed by medical examiners in patients who died of sudden, unexpected, or suspicious circumstances, according to Wisconsin law. Enucleations are

performed from behind, inside the skull, unroofing the orbital plates and dissecting from behind. The orbits are not exenterated or examined for signs of trauma. In cases of MVCs, the decision to remove the eyes is left to the individual pathologist.

Institutional review board approval for the study was obtained. All available medical and autopsy records, pathology slides and photographs, and police and traffic department reports were reviewed for each case. Many of these records are in the public domain. In addition to documentation of the specific type and location of the retinal hemorrhaging, details were also recorded regarding each child's injuries and the traumatic event leading to the injuries.

RESULTS

During the study period, 10 children underwent autopsies that included the eyes. Details of the incidents and the patients' findings are summarized in the **Table**. Six other children underwent autopsies without eye examinations and were not included.

Improper restraints and direct blows in automobile-pedestrian accidents contributed to the severity of the injuries in 6 patients. All patients had severe head injuries, but 3 did not have skull fractures. Autopsy findings did not raise any suspicion of earlier abuse of the children. Eight patients had retinal hemorrhages, which extended into

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Table. Incident Details and Autopsy Findings in 10 Children Fatally Injured in Motor Vehicle Crashes^a

Patient No./ Age	Details of Crash	Cause of Death	Head Injury	Restraint	Optic Nerve Findings	Retina Findings
1/13 wk	Head-on collision; car of drunk driver flipped onto patient's car; another child in car also killed, not examined	Brain laceration, hemothorax	2 Depressed skull fx, midbrain parenchymal laceration	Forward facing in car seat meant to be rear facing only	Bilateral sheath hemorrhage	R eye: extensive, innumerable RHs to ora serrata at all levels, multiple sub-ILM hemorrhages; L eye: normal
2/9 mo	Patient's car struck utility pole; patient struck windshield	Brain injury, liver laceration, pulmonary hemorrhage	Multiple linear skull fx, brain contusions	None; sitting on parent's lap in front seat	Bilateral sheath hemorrhage	R eye: circular retinal fold, confluent RHs at all levels to ora serrata, dome-shaped sub-ILM hemorrhage and vitreous hemorrhage; L eye: innumerable RHs at all levels to ora serrata; bilateral extensive RHs at all levels to ora serrata, sub-ILM hemorrhage
3/18 mo	Automobile-pedestrian; struck and dragged	Brain injury, liver laceration, pulmonary hemorrhage	Severe cerebral edema, SDH, IVH, no skull fx	NA	Bilateral sheath hemorrhage	Bilateral extensive RHs to ora serrata and sub-ILM hemorrhage
4/2 y	3150 kg of cargo from oncoming truck struck patient's car, forcing it backward; driver also killed	Cerebral edema	SDH; no skull fx	Correctly used forward-facing car seat in back seat	Bilateral sheath hemorrhage	R eye: peripapillary, a few peripheral intraretinal RHs; L eye: peripapillary intraretinal RHs only; no sub-ILM RHs in either eye
5/6 mo	Patient's car struck at 90° with intrusion by pickup truck of drunk driver; driver also killed	Cerebral edema	Skull fx, IVH, SDH, SAH	Rear, in car seat, direction facing unknown	Bilateral sheath hemorrhage	Bilateral extensive RHs, confluent in some areas, at all levels to ora serrata, sub-ILM hemorrhage in both eyes
6/5 mo	Nearly head-on collision; both cars traveling > 88 km/h; drunk driver of other car crossed meridian	Head injury, splenic tear	Multiple skull fx, cerebral contusions and lacerations	Correctly strapped, rear facing in middle of rear seat	Bilateral sheath hemorrhage	Bilateral extensive RHs to ora serrata; no sub-ILM hemorrhage in either eye
7/15 mo	Rollover crash; single-car accident	Head injury	L parietal lobe hemorrhage, diffuse cerebral edema, IVH, no skull fx	Unknown; was ejected from car	Bilateral sheath hemorrhage	Bilateral extensive RHs at all levels to ora serrata, sub-ILM hemorrhage; L eye: circular retinal fold
8/4 mo	Patient's car struck at 90° with 30.5-cm indentation by car of drunk driver	Head injury, grade V splenic laceration, lung contusions	Depressed skull fx with extrusion of brain, contusions and lacerations of brain, SDH extending into spinal cord	Forward facing in car seat meant to be rear facing only	Bilateral sheath hemorrhage	R eye: circular retinal fold; bilateral confluent RHs to ora serrata, sub-ILM hemorrhage, vitreous hemorrhage
9/7 mo	Pedestrian hit by illegally drag-racing car	Head injury	Multiple skull fx, cerebral avulsion, laceration	NA	L eye: sheath hemorrhage	No retinal abnormalities
10/22 mo	Ran into street; struck by car leaving curb	Head injury	Basilar skull fx, R frontal lobe laceration	NA	None	No retinal abnormalities

Abbreviations: fx, fracture; ILM, internal limiting membrane of the retina; IVH, intraventricular hemorrhage of the brain; L, left; NA, not applicable; R, right; RHs, retinal hemorrhages; SAH, subarachnoid hemorrhage of the brain; SDH, subdural hemorrhage of the brain.

^aThe types of force in all cases were impact and acceleration/deceleration injury.

the periphery in 13 eyes and were bilateral in 7 patients. Two children had no hemorrhages, and another had only a few peripapillary hemorrhages in 1 eye. Nine patients had optic nerve sheath hemorrhages, which were bilateral

in 8. Three patients had elevated circular retinal folds. Six patients had dome-shaped hemorrhages just below the internal limiting membrane (ILM), but no patients were found to have deeper splitting of the retinal layers.

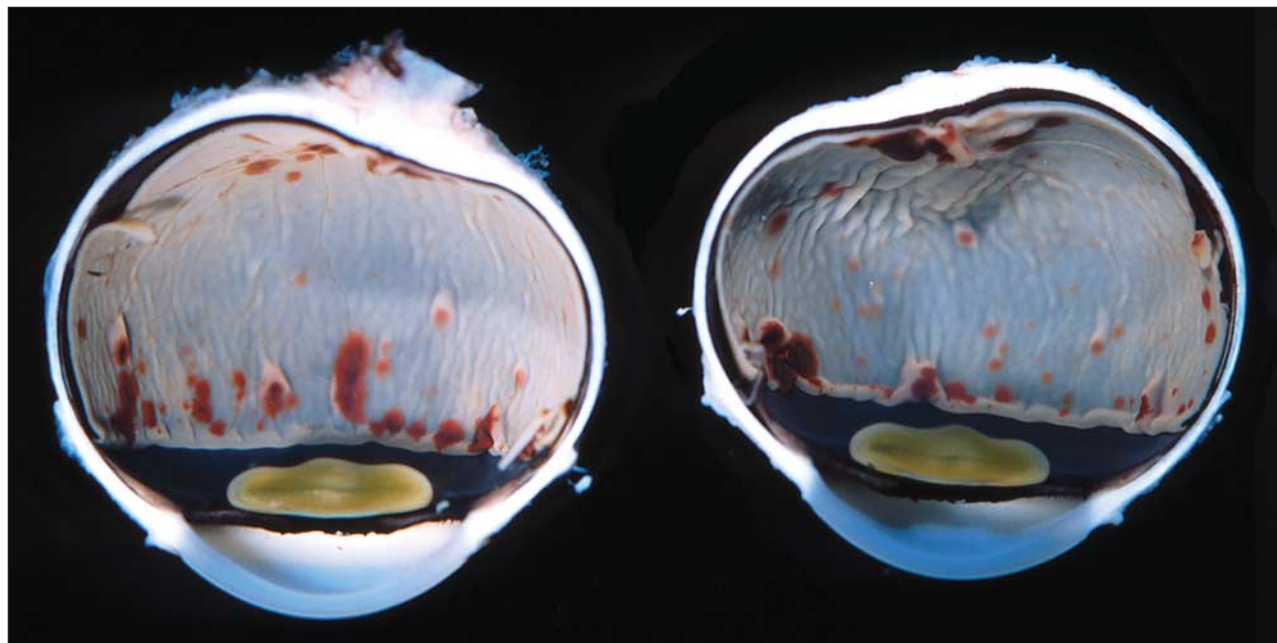


Figure 1. Patient 3, right eye (2 views). Many nonconfluent hemorrhages extend to the ora serrata.

COMMENT

The most common traumatic mechanism associated with retinal hemorrhaging of any severity—but particularly the most severe forms where the hemorrhages extend to the ora serrata—is rotational acceleration/deceleration type injury. This is the mechanism involved in nonaccidental childhood neurotrauma (also known as *shaken baby syndrome*).

Multiple reports including children of all ages have demonstrated the infrequency of retinal hemorrhaging with known accidental trauma of various mechanisms, even with skull fractures. In prospective, possibly overlapping, studies of accidentally injured nonnewborn children, only 69 of at least 776 children examined (8.9%) had retinal hemorrhages.²⁻³² Retrospective studies have shown similar results.³³⁻³⁹ Other than the certainty that the injury was accidental, details have been sparse regarding the type of accident, mortality, and the number, extent, and laterality of the retinal hemorrhages.

Of patients described in the literature, 38 had mild hemorrhages that did not extend to the ora serrata.^{2,5-8,10,11,13,14,17,21} Reports concerning an additional 23 patients did not include enough description of their hemorrhages to determine whether the hemorrhages were mild or severe.* The 7 patients known to have hemorrhages extending to the ora serrata died of their injuries.^{4,7-9,21} Three patients died of high-energy, rotational forces similar to those seen in nonaccidental childhood neurotrauma.^{8,21} Four other patients died of crush injuries to the head caused by toppling television sets, the fall of another child onto the head of the victim, or an MVC.^{4,7-9} Plunkett³⁸ retrospectively reported 4 children with retinal hemorrhages who died of playground injuries. Their hemorrhages were found by a neurosurgeon or by pediatric intensivists⁴⁰; details regarding the extent, type, and

number of hemorrhages are uncertain compared with the autopsy findings reported herein. In each investigation, a best estimate was that the injuries were accidental rather than inflicted, although some were not witnessed, particularly by an adult. That the accidents were fatal indicates the severity of the forces delivered to the children's heads; that two-thirds of those examined had retinal hemorrhages is consistent with severe injury.

The most commonly suggested mechanisms for retinal hemorrhages in children younger than 3 years, particularly in nonaccidental head trauma, are mechanical vitreous traction on the retina and a sudden increase in venous pressure in the retinal vessels.⁴¹ The mechanical theory suggests that traction on the retina due to the motion of the vitreous body during violent rotational acceleration/deceleration forces causes direct damage to the retina and its blood vessels.⁴¹ The attachment of the vitreous body in children is strongest at the posterior pole, along the vessels, and at the periphery, which may explain the distinctive distribution often seen in the most severe retinal hemorrhaging and retinal detachment in nonaccidental neurotrauma, similar to **Figure 1**.⁴¹⁻⁴³ The observed attachment of the ILM to the peak of the circular retinal fold⁴¹ and the finding of orbit tissue injury during autopsy examination of children with nonaccidental neurotrauma and accidental death⁵ have also been cited as evidence of mechanical forces.

Some well-documented cases of abusive neurotrauma have shown no sign of direct head impact on careful autopsy examination of the layers of the head.⁴⁴⁻⁴⁶ Perpetrator confessions have corroborated the shaking mechanism suspected.⁴⁷ The children we studied had severe acceleration/deceleration injuries consistent with the mechanical theory for retinal hemorrhages. Patient 7 had an elevated retinal fold but did not have skull fractures.

Most elevated retinal folds reported in children have been associated with abusive neurotrauma.^{41,44,48,49} These

*References 3, 8, 16, 20, 21, 23, 25, 27, 30, 32, 39.

children had a higher mortality rate, indicating that they had more severe injury. Three children who died of accidental crush injuries to the head were reported to have elevated retinal folds.^{3,4,9} The occurrence of these folds in an uncertain number of patients with nontraumatic Terson syndrome has raised the possibility that the hemorrhage itself rather than traction of the vitreous on the retina plays a role,^{50,51} but this possibility has been questioned by Levin.⁴¹ In at least 1 trauma patient, a very large, dome-shaped sub-ILM hemorrhage has been found spanning the macular region encompassed by the circular retinal fold.⁴ The 3 children reported herein to have elevated circular retinal folds did not have this large sub-ILM hemorrhage (patients 2, 7, and 8) (**Figure 2**). Emerson and coauthors⁵² have proposed that sustained venous stasis and leakage from retinal vessels can lead to elevated circular retinal folds in non-accidental neurotrauma. Our patients demonstrated that abrupt injury can cause these folds as well.

A sudden increase in venous pressure in the eye is the other primary mechanism proposed to cause retinal hemorrhages in nonaccidental neurotrauma. Less evidence supports this, however, because Terson syndrome is rarely seen in children. A study of the eyes in children with intracranial hemorrhage of various causes found only 5 small hemorrhages in 1 patient who had been in an MVC.¹³ Increased venous pressure secondary to Valsalva maneuvers or chest compression⁵³⁻⁵⁵ does not seem to be a likely cause of intraocular hemorrhage because these processes cause very few or no retinal hemorrhages in children.⁵⁶⁻⁶¹ Crush injuries may cause retinal hemorrhages because of increased intracranial and intraocular venous pressure. However, 9 patients who died of their crush injuries did not have retinal hemorrhages or had only a few posterior hemorrhages,^{5,7} and 11 survivors did not have any hemorrhages.⁷

Three accidentally crushed children were reported to have large hemorrhages and retinoschisis.^{3,4,9} In our patients, there were sub-ILM hemorrhages and no deeper splitting of the retina. In 3 patients who underwent vitrectomies for large dome-shaped macular hemorrhages associated with a retinal fold or pigmentary changes around the macula, the excised anterior wall of the cyst contained ILM.⁶² These patients included a 17-year-old and 4-year-old with Terson syndrome of unknown cause and a 5-month-old with shaken baby syndrome. A postmortem examination in a 7-month-old with shaken baby syndrome also showed a hemorrhagic cyst enclosed by ILM.⁴⁴

Many abused children with retinal hemorrhages have skull fractures that must have been caused by impact injuries but rarely to the point of classifying them as crush injuries.^{21,63} Seven of the patients reported herein had skull fractures (patients 1, 2, 5, 6, 8, 9, and 10), and 5 (patients 1, 6, 8, 9, and 10) had depressed skull fractures or fractures with brain lacerations. In the police reports, there was no indication that the children were crushed between 2 surfaces, as has been the case in patients reported to have an object or person fall on their head.^{3,4,9} Three patients had no skull fractures (patients 3, 4, and 7); their injuries largely resulted from acceleration/deceleration mechanisms. Impact and acceleration/deceleration mechanisms can combine to cause retinal

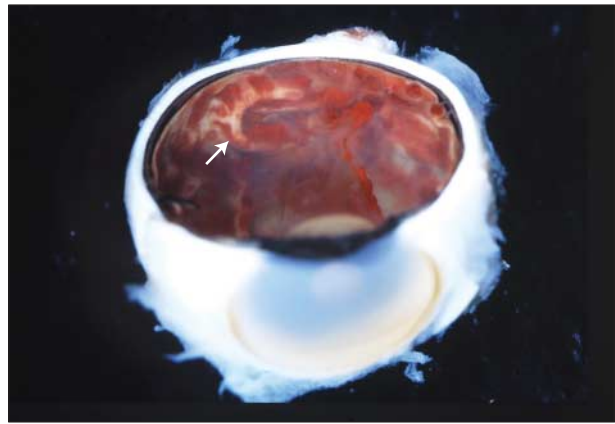


Figure 2. Patient 8, right eye. Many confluent hemorrhages extend to the ora serrata. An elevated circular retinal fold is noted in the posterior pole (arrow).

hemorrhage, as in the tin ear syndrome⁶⁴ and nonaccidental childhood neurotrauma.⁴¹

Our patients had injuries that involved acceleration/deceleration mechanisms, but they also had sudden head injuries and intracranial bleeding; thus, both proposed mechanisms for retinal hemorrhages could apply. Patient 9, who died of severe brain injury on the day of the accident, had only optic nerve sheath hemorrhage. Patient 4, who had only mild peripapillary hemorrhages in the left eye, died 7 days after the injury. Retinal hemorrhages were noted by the intensive care attending physician 1 day before death, but the extent and laterality of the hemorrhages were not noted. Some retinal hemorrhages could have resolved before death.

It is not clear why some patients who were fatally injured by accident or intent do not have retinal hemorrhages. Some of these patients died immediately, negating the possibility that hemorrhages had been present but resolved before death. The particular type of trauma and where the forces were sustained must account for the variation in retinal findings.

CONCLUSIONS

All of our patients died after being subjected to extremely severe forces involving rapid deceleration with a rotational (whiplashlike) component, similar to that described in nonaccidental childhood neurotrauma. The extent and severity of the retinal hemorrhages, when compared with the rare and mild hemorrhages in other less severely accidentally injured patients, indicate that more severe trauma causes more severe eye findings. These were all extremely high-force injury mechanisms that far exceed those involved in common (or even uncommon) household accidents. This supports previous conclusions^{2,8,21,65} that, in the absence of a known major accidental injury involving severe intracranial trauma, such as a crush injury or high-force MVC, extensive retinal hemorrhages are highly indicative of occult, severe intentional injury.

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