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REVIEW ARTICLE

Ocular pathology in shaken baby syndrome and other forms of infantile non-accidental head injury

Jakob Matschke · Klaus Püschel · Markus Glatzel

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Abstract Retinal and optic nerve sheath hemorrhages are an essential part of the various forms of inflicted neurotrauma in infants, especially in its most common variant, the shaken baby syndrome (SBS). Clinically, ophthalmologists play an important part for the diagnosis and further management of patients with SBS. For the forensic pathologist, a thorough understanding of the basic principles behind the morphological phenomena commonly encountered in the orbita is required. This review summarizes the present knowledge of ocular pathology in inflicted infantile neurotrauma, focusing strongly on SBS. Furthermore, a review of the most recent literature on the subject is given.

Keywords Forensic neuropathology · Shaken baby syndrome · Non-accidental head injury · Ocular pathology

J. Matschke (✉)
Forensic Neuropathology,
University Medical Centre Hamburg-Eppendorf,
Martinistrasse 52,
20246 Hamburg, Germany
e-mail: matschke@uke.de

J. Matschke · K. Püschel
Institute of Legal Medicine,
University Medical Centre Hamburg-Eppendorf,
Martinistrasse 52,
20246 Hamburg, Germany

J. Matschke · M. Glatzel
Institute of Neuropathology,
University Medical Centre Hamburg-Eppendorf,
Martinistrasse 52,
20246 Hamburg, Germany

Introduction and historical background

The appearances of child abuse are protean, encompassing physical, sexual, or emotional abuse as well as neglect [23]. Although the first description of subdural hemorrhage (SDH) in association with child abuse originated in the late nineteenth century [78, 112], it was not until 1962, when Henry Kempe et al. published their seminal paper on the "battered child syndrome" [63], that abuse and maltreatment of children came to the attention of the medical community. Another 10 years later, John Caffey—pursuing previous studies by Ommaya and Yarnell [92], Guthkelch [52], and himself [14]—described the shaken baby syndrome (SBS) [15]. SBS is nowadays widely accepted as a "serious and clearly definable form of child abuse" [2], classically combining SDH and an acute encephalopathy with retinal hemorrhages (RH) and only sparse or absent signs of external injury. It occurs almost exclusively in children younger than 2 years of age—although very rare cases of older children or even adults are on record [16, 98]—often occurring in a context of inadequate or inconsistent history as provided by the caregiver. Although the exact biomechanics and pathophysiology behind the grave clinical picture with a mortality of up to 30% are still not completely understood, most researchers agree that the shaking of an infant produces severe shearing and rotational forces to cause the intracranial and ocular pathology deemed typical for this syndrome [38, 75, 88]. SBS can be considered as a common part in the broader spectrum labeled "non-accidental head injury" (NAHI), encompassing further, albeit rarer, forms of inflicted infantile neurotrauma (shaken-impact syndrome, pure blunt head trauma, crush injury, etc.). For the pathologist asked to give written or oral expertise in court, more than a basic knowledge and understanding of the

entire spectrum is absolutely mandatory—even if the scientific basis of this condition is only poorly understood [111]. The present review aims at summarizing the present knowledge and most recent scientific data of ocular pathology in SBS and other forms of NAHI for the specific scope of the forensic practitioner.

Early ophthalmologic work

Since the medical discovery of NAHI and SBS in infants, ocular pathology, mostly in the form of RH and optic nerve sheath hemorrhages, has been recognized as an essential part of the syndrome. Some 1–5% of cases present initially with ocular symptoms which stresses the role of the ophthalmologist both in helping to establish the diagnosis and aid in its further management [34, 39, 84, 91]. Paralleling the discovery of the "battered child" [63], first isolated case reports on ophthalmologic findings in these patients began to appear in the early 1960s. Kiffney described retinal detachment, which was probably bilateral, in a 7-month-old female suffering from the "battered-child-syndrome", in 1964 [64]. Three years later, Gilkes and Mann wrote in a letter to Lancet that they had been "impressed by the extreme degree and the persistence of the retinal hemorrhages [...] in the physically abused child" and called for "a definite study of these cases" [41]. Yet it took nearly another 5 years before a first scholarly review paper appeared on the subject, intended to heighten the general awareness of the "battered child syndrome" in the ophthalmologic community [34]. More comprehensive case series were soon to follow [53, 86, 87]. Since then, the number of papers has steadily risen over the years, with the emphasis and focus of the literature becoming continuously diverse, stressing the need for this topical review for the forensic pathologist [1, 5, 45, 65, 66, 70, 72, 84].

Retinal hemorrhages and optic nerve sheath hemorrhages

It is generally held that retinal hemorrhage and optic nerve sheath hemorrhages form an essential part of the NAHI/SBS complex, but most researchers agree that both are neither necessary nor sufficient for diagnosis [5]. For victims of child abuse in general, frequencies of RH have been reported between 40% and 61% [34, 100, 101]. Variations in case definitions, and/or detection techniques used, cause the wide range in reported frequencies of 50–100% in SBS/NAHI patients [50, 81, 95]. The "classic" RH in NAHI/SBS are bilateral, symmetric, preretinal, subretinal, or intraretinal, as well as subhyaloid or submembranous, and mostly located at the posterior pole and/or the mid-periphery near the ora serrata (see Figs. 1 and 2) [50, 77, 81, 95]. Their shape is often reported as dot-, blot-, or flame-like [95], or, more rarely, as white-centered [62], although there has been no consensus on any particular pattern of SBS/NAHI-associated RH when compared with RH of other causes [95]. It comes as no surprise that examination by an experienced ophthalmologist is strongly recommended, since non-ophthalmologists have been shown to miss some 12–29% of all RH in SBS/NAHI patients [65, 82]. RH in NAHI/SBS is typically bilateral, but unilaterality does occur in roughly 15% of cases and should not be suggested as evidence against charges of child abuse in courtroom settings [22, 55, 116]. In a recent analysis of unilateral RH in SBS patients, Arlotti et al. found the unilaterality of RH and SDB in 100% to be ipsilateral to one another [3], which is in contradiction to a paper by Morad et al. from 2002, in which no such correlation was discerned [81]. Thus, this issue remains unresolved.

Pathophysiology and pathology

Although ocular pathology in NAHI/SBS has been recognized for nearly half a century, the pathogenesis of most characteristic findings is still only poorly understood, and, as one recent reviewer has put it, "we do not really know how RHs form in cases of alleged non-accidental injury" [45]. Most likely and intuitively compelling is a pattern of repeated severe acceleration–deceleration injury to the vitreous, paralleling that believed to be responsible for the intracranial injuries [99]. The resulting stress forces cause vitreoretinal tractional trauma to the retina and its blood vessels and thus lead to RH [50, 103, 123], retinoschisis [28, 51], and retinal folds [28, 77]. In adults, similar acceleration–deceleration mechanisms in various circumstances, ranging from emergency aircraft ejection [74] to bungee jumping [17, 21, 31, 58, 59, 109, 118], have been reported to cause RH. Furthermore, vitreous hemorrhage and retinal detachment have been reported in severely retarded children repeatedly beating their heads against walls or other hard objects [89]. Since the vitreous is tightly attached to the retina at the posterior pole and near the ora serrata, RH is often located in these regions, showing a pattern of relative equatorial sparing, thus further supporting the shearing theory [45, 50]. Other, most probably concurrent, factors include a sudden rise in intravenous pressure due to thoracic compression during shaking attempts [114] as well as the transmission of blood from the intracranial compartments to the optic nerve and the retina, as seen in adult Terson's syndrome [121].

Unfortunately, there is a reluctance of most clinical and some forensic pathologists to remove the eyeballs of an

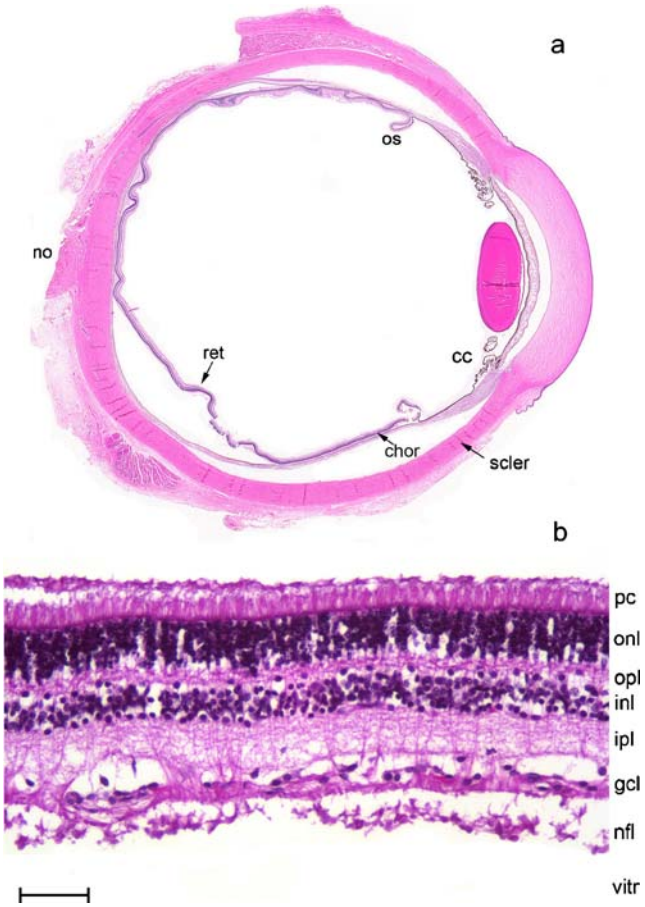

Fig. 1 Anatomy of the eyeball and the retina. **a** Whole mount section through the eyeball of a 7-month-old male infant with a diagnosis of SIDS with anatomical designations: sclera (*scler*), chorioidea (*chor*), retina (*ret*), ciliary body (*cc*), ora serrata (*os*), nervus opticus (*no*) (H&E stain). **b** Section through the retina (detail from **a**) with anatomical designations: photoreceptors (*pc*), outer nuclear layer (*onl*), outer plexiform layer (*opl*), inner nuclear layer (*inl*), inner plexiform layer (*ipl*), ganglion cell layer (*gcl*), nerve fibre layer (*nfl*), vitreous (*vitr*). (H&E stain, scale bar indicates 50 μm)

infant during autopsy even in cases of suspected child abuse. This may explain the relative dearth in postmortem case–control studies. The posterior approach for removal of the globes is strongly recommended since it allows a larger segment of the optic nerve to be obtained. Most pathologists prefer horizontal sections of the globes in the pupil–optic nerve plane after fixation in buffered formalin for a minimum of 3 days. Some researchers have suggested an anterior approach, allowing better correlation with premortem pictures [42]. The difficulties in comparison of various reports of ocular findings are due to widespread use of very limited descriptions, such as "RHs were present" [45]. To overcome this, structured and detailed protocols have been designed [46], but unfortunately are far from becoming widely accepted or even known. Usually, standard staining procedures such as hematoxylin and eosin (H&E) and Masson's trichrome will show even slight RH and optic nerve sheath hemorrhages. Turnbull's stain will detect hemosiderin as evidence for repeated hemorrhages or in cases with >48 h survival. Although the clearance or persistence of hemosiderin following neonatal RH is poorly studied, in general, it can be assumed that if hemosiderin can be detected in the retina of infants more than 2–3 months old, that this is due to RH after birth [113]. Caution is warranted in histological reports of retinal folds, since confusion with the inevitable postmortem artifacts due to processing of specimens cannot be ruled out with certainty (Fig. 3), although folds due to shaking are said to arch over the macula, which is in contrast to the artificial folds typically originating at the optic nerve head [28, 77]. Postmortem morphometrical analysis of retinal hemorrhages has been performed, reporting RH in up to 73.2% of the entire retinal area in victims of SBS [8].

As clinical experience suggests, most studies indicate a relationship between the extent of RH and the clinical picture: in one study, the severity of RH based on type and size of the bleeding and the extent of fundus involvement significantly correlated with the severity of acute neurologic injury [122], and in another study including 75 SBS/NAHI children, a correlation between the severity of retinal and intracranial injury (as manifested as a high index in both a "total cranial trauma"- and a "total retinal hemorrhage-score") was shown [81]. Thirdly, a strong correlation between central nervous system (CNS) and ophthalmologic trauma could be shown following forensic pathological study of 23 children who died of NAHI [50]. In a comprehensive study of 110 SBS/NAHI patients examined by an ophthalmologist, the presence of any RH increased the risk of dying with an odds ratio of 5.12. Furthermore, the authors described a significant trend with the severity of RH from no RH (10% dying), unilateral RH (23%) to bilateral RH (38%) [65]. Yet, caution in the courtroom for extrapolating an applied force from resulting pathology in individual cases is strongly advised [45]. Furthermore, cases with RH unaccompanied by intracranial hemorrhages with good recovery have been published [55, 114] and a correlation between the occurrence of Terson's syndrome with adverse outcome in adults with intracranial hemorrhage has been reported [108, 121]. Finally, one report was unable to find any correlation between the extent of RH and the severity of brain injury [95] (Fig. 3).

It should be kept in mind, that in most pathological reports, optic nerve sheath hemorrhages (Fig. 4) are more frequent than RH [11, 123], making detailed histological study of the separate optic nerve mandatory. In postmortem studies, RH of varying degree and/or optic nerve sheath hemorrhages can be detected in up to 100% of NAHI/SBS cases [8, 11, 50, 85, 123]. Optic nerve sheath hemorrhages can be found in the subarachnoid or the subdural space, but tend to be most prominent in the latter [11]. A detailed

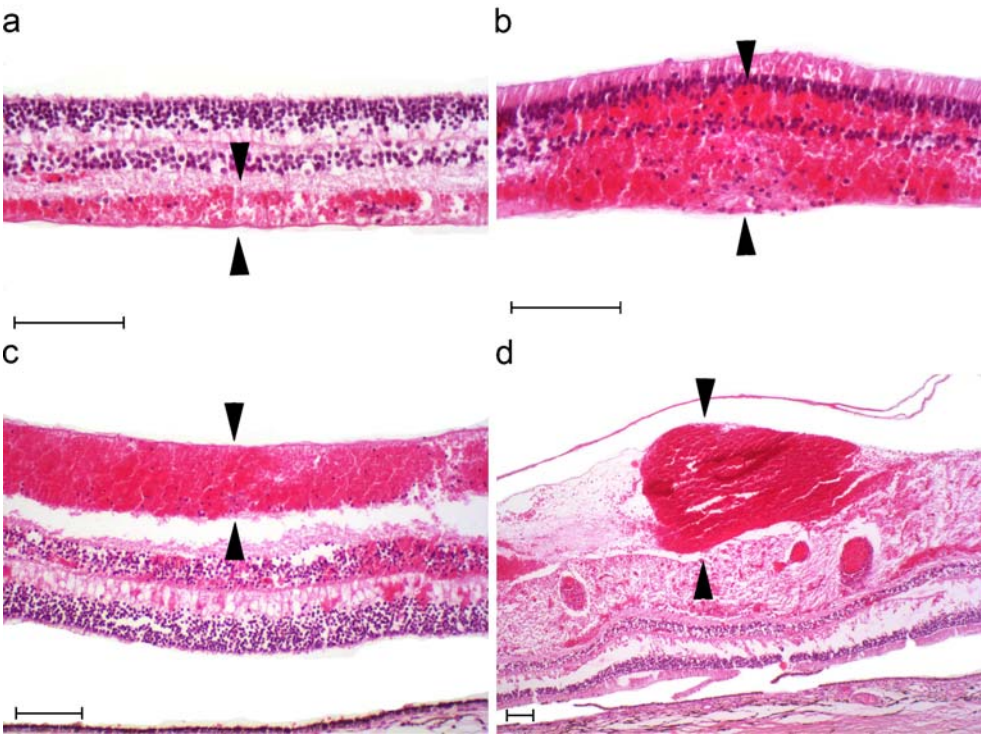

Fig. 2 Retinal hemorrhages. Various forms of retinal hemorrhage (between *arrowheads*) in a case of a 10-month-old female infant with a diagnosis of SBS after being admittedly shaken: **a** subretinal, **b** intraretinal, **c** submembranous (still covered by the internal membrana limitans), and **d** subhyaloid (H&E staining; scale bar 50 μm)

histopathologic study of the orbit of 18 SBS patients revealed optic nerve sheath hemorrhaging at the anterior or posterior portions of the optic nerve, as well as hemorrhages into the orbital fat and extraocular muscles [123]. Consequently, the postmortem analysis of these tissues has been called for [84, 123].

Ocular pathology other than RH and optic nerve sheath hemorrhages

Since the first independent report on ocular pathology in SBS [64], ophthalmologists have been aware of findings other than RH, including signs of periorbital or anterior segment damage as a result of direct or penetrating trauma. These findings have so far not been the subject of comprehensive studies but instead have been published in various case reports or case series. Amongst these, there have been reports on perimacular retinal folds and hemorrhagic macular retinoschisis, both of which are considered highly specific for SBS [9, 28, 37, 51, 77]. Another relatively common finding is hemorrhaging into the vitreous: one study found that in patients younger than 18 years of age with vitreous hemorrhage, manifest or occult trauma accounted for 73.1% and SBS was the cause in 50% of the bilateral cases [110]. Other findings include macular holes with or without RH [4, 94], intrascleral hemorrhages [28], retinal and vitreous detachment [10, 120], hemorrhages in orbital fat and extraocular muscles [123], complex findings simulating congenital glaucoma

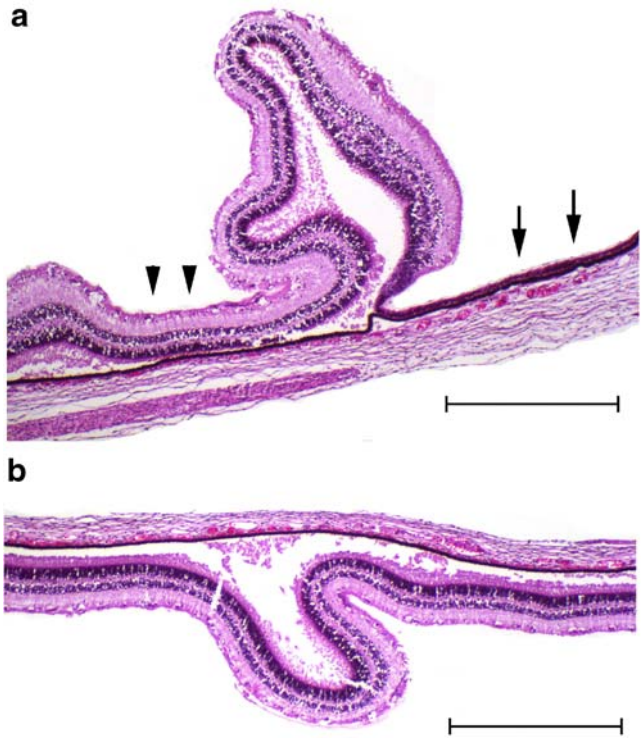

Fig. 3 Anatomy of the retina (details). **a** Section through the ora serrata (detail from Fig. 1) at the transition between the multi-layered photosensitive pars optica of the retina (*arrowheads*) and the single layer of non-pigmented epithelium of its non-photosensitive pars caeca (*arrows*). (H&E stain, scale bar 500 μm). **b** Artificial tearing of the retina from the underlying pigmented epithelium and the choroid (detail from Fig. 1). This postmortal phenomenon is a common and inevitable artifact of tissue processing and is easily confused with retinal folds due to shaking (H&E stain, scale bar 500 μm)

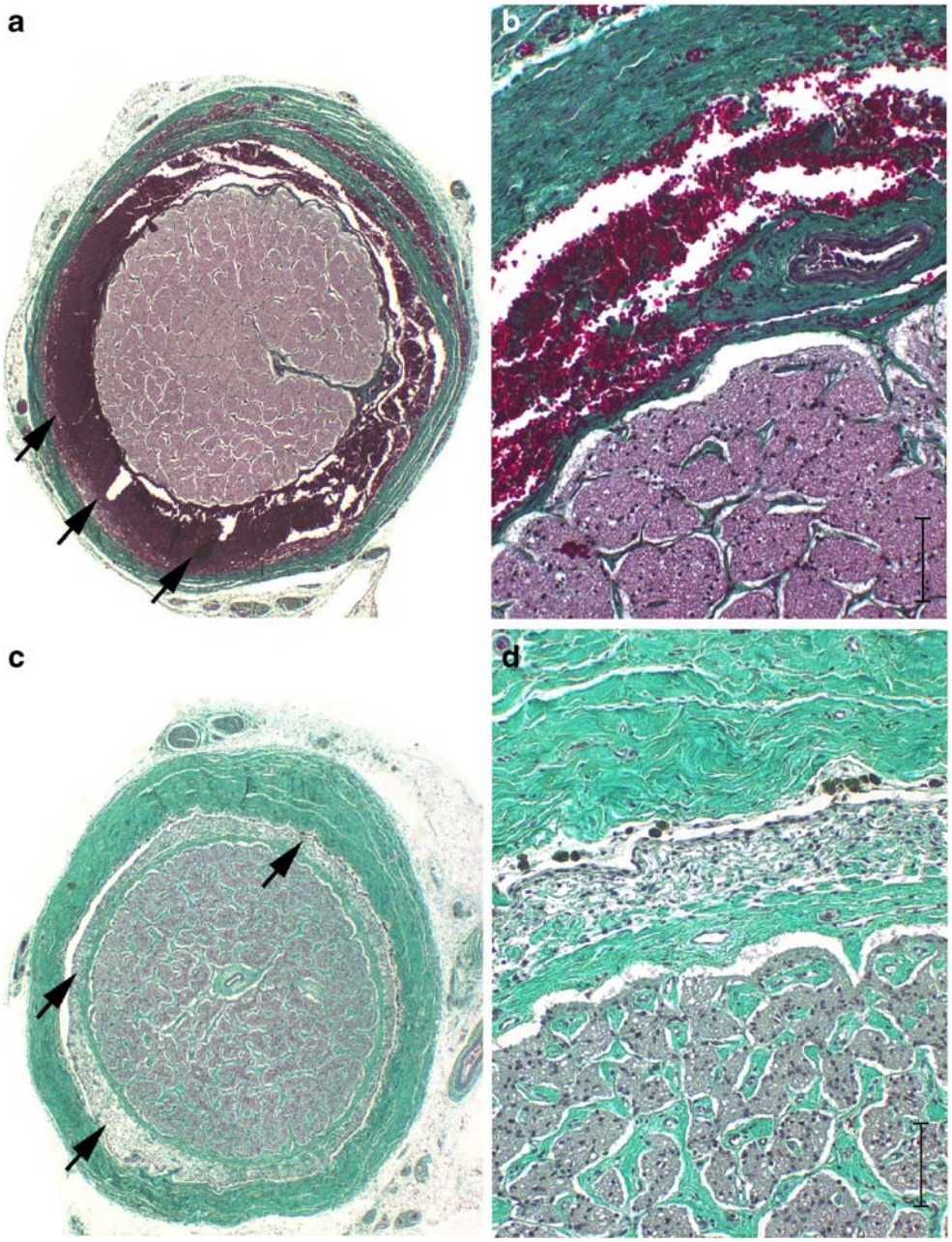

Fig. 4 Optic nerve sheath hemorrhages. **a** Whole mount transverse section of the optic nerve sheath in a 4-month-old girl with SBS showing fresh hemorrhages (*arrows*; Masson's trichrome). **b** Magnification from **a**, scale bar 100 μm (Masson's trichrome). **c** Whole mount transverse section of the optic nerve in a 3-month-old male who had been admittedly shaken 1 month prior to death showing granulation tissue in the nerve sheath (*arrows*; Masson's trichrome). **d** Deposition of hemosiderin in **c** (Turnbull's stain, scale bar 100 μm)

[115], keratoconjunctivitis [93], vertical sensory nystagmus [83], giant retinal tears [69], epiretinal membrane formation [27], and traumatic cranial nerve palsy [13, 30, 65].

Differential diagnosis

There are substantial numbers of cases reporting RH in infants due to apparently innocent causes, or in the context of a variety of medical disease such as spinal arteriovenous malformation [19], fibromuscular dysplasia [20], osteogenesis imperfecta type I [36], blood dyscrasias [76], and Coats' disease [5]. Although some of these may be associated with minor accidental trauma, stigmata of the underlying disorder allow its recognition usually rather quickly, absolving the physician from the need for further inquiry for suspected child abuse [113]. Often cited is the occurrence of RH (as well as of other stigmata of SBS/NAHI) in glutaric aciduria type I, a rare neurometabolic disorder due to a autosomal recessive deficiency in glutaryl-CoA dehydrogenase [35, 54]. Although these infants generally present with acute crisis-like encephalopathy, progressive dystonia, seizures and frontotemporal atrophy, screening of urine for increased levels of glutaric and 3-hydroxyglutaric acid in suspected SBS/NAHI cases is often recommended [35]. RH in the clinical syndrome of

apparent life-threatening events (ALTE), which has recently been reported to occur in 1.4% of patients [96], may make the differential diagnosis in such cases extremely difficult. The most difficult and controversial situation may be encountered in a court setting where the defense pleas to have shaken an infant in an alleged attempt of resuscitation after what may appear to have been an ALTE which may or not explain findings of SDH and/or RH.

Since it is often alleged that an accidental head injury is the cause of an infant's condition with SDB and RH being present, the question whether RH may occur with accidental head injury has been the object of many studies. Even when few isolated cases report RH or other ocular pathology in the setting of accidental injury [18, 33, 68], it is worthwhile to remember that these represent an extreme rarity. Accordingly, comprehensive epidemiologic studies have failed to show any significant association between RH and accidental head injury: in a study of 75 children younger than 3 years of age who sustained accidental head injury, all had normal funduscopic findings [12]. In 25 children (four of which were under 2 years of age) who had sustained accidental head injury, RH was not detected in any of the cases [26]. Ophthalmologic examination of 140 children with a median age of 4.5 years with accidental head injury found only two cases with RH; each of these had been involved in side-impact motor vehicle crash and one died [60]. Of ten patients with RH in a cohort of 100 children younger than 2 years, hospitalized for head injury, nine had NAHI. The remaining case suffered a high-speed motor vehicle crush and later died from the injuries [24]. If RH occurs in the setting of accidental injury, it is usually in high-velocity impact motor vehicle accidents, high falls, or severe crush head injury [44, 47, 60, 68, 123]. Crush injury may also cause retinal folds [68, 73]. From the forensic viewpoint, strong criticism is warranted in published case reports on RH in minor head injury, when the alleged mechanism of injury apparently has not been corroborated by independent witnesses [18].

Resuscitation attempts have been repeatedly suggested as a further possible explanation for RH in infants, as thoracic compression for cardiac massage has been shown to increase intracranial pressure [102]. As some authors hypothesized, even slapping on the back in an attempt to revive a baby that suddenly goes limp, may cause RH, allegedly due to hypoxia in combination with elevation of intrathoracic pressure [6, 119]. A critical re-evaluation of the original reports casts grave doubts on these cases: in one case there were no independent witnesses except for the persons involved in the slapping act, and in the other, the baby had suffered second and third degree burns and scalds after his 2-year-old sibling had allegedly involuntary turned on the faucet. A recent comprehensive study performed ophthalmoscopy in pediatric patients under 2 years of age, which had undergone cardiopulmonary resuscitation (CPR) for non-traumatic illnesses. None of the 43 studied patients showed RH, with the exception of one patient with small punctate bleedings [90]. Another study of 20 resuscitated children found multiple large bilateral RH in a 2-year-old developmentally retarded girl with a history of near-drowning and a single fresh RH in a 6-week-old girl immediately after termination of 75 min of resuscitation who was later given a diagnosis of SIDS (although she had been admitted with "fever, upper respiratory tract infection and possible sepsis") [48]. A further study of 45 pediatric patients in whom CPR was not preceded by a traumatic event identified only one case (2%) with RH; this patient had had severe arterial hypertension and seizures [61]. In a postmortem histopathological study of 169 childhood deaths with a mean age of 17 months, 61 cases with RH and prolonged resuscitation attempts were found, but 60 of these suffered from head injury, CNS disease, or sepsis, which are known concurrent causes of RH. The single remaining case was an "undetermined" death from a household with two prior child deaths and documented abuse [43].

A similar mechanism has been suggested for the presumed occurrence of RH following epileptic seizures with a sudden rise in retinal venous pressure, following a rise in central venous pressure secondary to a rise in intrathoracic pressure, paralleling the Valsalva retinopathy seen in some adults after coughing, vomiting, or weightlifting. Yet in one study of 32 consecutive children under the age of 2 years admitted with convulsions, none had RH [117]. Accordingly, a further, more comprehensive study in 153 children aged 2 months to 2 years, who were examined by an ophthalmologist following an epileptic seizure revealed only one 8-month-old girl with unilateral flame-shaped RH around the optic disk [80]. In a recent study of 32 children requiring hospitalization after a seizure (mean age, 30 months), none had RH, although some of the children were resuscitated [104]. No RHs were found in 100 infants with vomiting caused by hypertrophic pyloric stenosis [56]. Finally, indirect ophthalmoscopy in 100 consecutive children between 3 months and 2 years with severe persistent cough failed to come up with a single case of RH [49].

Retinal hemorrhages, which can be bilateral, can occur in up to more than 30% of newborns, albeit their incidence declines substantially within the first days [7, 29, 107] and even within the first hour [40]. Usually, most RH in newborns are completely resolved without any sequelae by 8 days after birth [29, 57, 113], whereas NAHI-associated RH will persist usually for months [79]. Association to mode of birth delivery has been reported with the highest frequency for vacuum delivery, followed by vaginal delivery with or without forceps and least often in cesarean

section [25, 29, 57, 71, 97, 107] although this still remains controversial [7]. One study found a higher frequency of RH in neonates following dinoprostone-induced labor when compared with oxytocin [106].

RH and even vitreous hemorrhages may also occur as a complication of retinopathy of prematurity (ROP) in 1–2% of preterm babies [32], but most of these can usually be differentiated by associated findings [67, 113]. For medico-legal issues, it is of importance that, contrary to the quick resolution of RH in term newborns, ROP may be more longstanding [32]. Since prematurity is considered an independent risk factor for SBS/NAHI but on the other hand, the immature vessels of ROP may predispose to RH after a shaking event [67], the differential diagnosis of RH in preterm infants can be challenging and in courtroom cases may require the additional expertise of an experienced ophthalmologist.

Terson's syndrome is the co-occurrence of RH or vitreous hemorrhage together with (in the broader definition) any kind of intracranial hemorrhage (subarachnoid, subdural, intracerebral). While not rare in adults, a study of 57 consecutive children (mean age, 10.3 years) with known intracranial hemorrhage of non-abuse cause, 55 (96%) had no evidence of RH or vitreous hemorrhage. Of the remaining two patients, one had a single dot RH, and the other had three flame-shaped and two dot RH following a 100-ft throw in a motor vehicle accident with an initial GCS of 3 [105]. Furthermore, a association between anatomic site of intracranial and retinal bleedings (which should be expected in Terson's syndrome) could not be drawn in one large study of 75 SBS/NAHI patients [81].

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