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Perimacular Retinal Folds Simulating Nonaccidental Injury in an Infant

Ophthalmologic abnormalities are important in the evaluation of infants suspected of being abused. A common form of child abuse is shaken baby syndrome (SBS), in which an infant is violently shaken, producing rapid, abrupt acceleration and deceleration of the cranium. Retinal hemorrhages are frequently found in children with SBS. A particularly severe form of retinal injury in SBS, traumatic retinoschisis, is characterized by a dome-shaped cavity in the macula with elevated perimacular folds at the periphery of the cavity.[1]

The etiology of perimacular folds has been the subject of debate. Massicotte et al[2] reported persistent attachment of the vitreous to the internal limiting membrane at the apices of these folds and suggested that this finding might constitute evidence of violent shaking. A recent report, however, described similar retinal findings in a 13-month-old infant who sustained a skull fracture and intracranial hemorrhage after a television fell on the infant's head.[3] We describe a child with perimacular folds and retinoschisis following a severe crush injury.

Report of a Case. A 4-month-old boy was on the floor when a 12-year-old, 63-kg child fell while running backward over an adult who was changing the infant's diaper, transmitting her entire weight through her buttocks directly to the infant's head. The infant was immediately unresponsive. Evaluation at the hospital revealed a large, comminuted, displaced parietal bone fracture; subdural and intraventricular hemorrhage; and brain herniation. Ocular examination revealed no visual responses and fixed, dilated pupils. Fundus examination revealed a large vitreous hemorrhage in the right eye. In the left eye, there was a macular retinoschisis cavity with elevated perimacular folds (**Figure 1**) and diffuse, 4-quadrant, multilayer retinal hemorrhage. Evaluation by the child protection team revealed no other physical findings or historical information suggestive of abuse. The child was found to be brain dead and he died. Two adults who witnessed the event were interviewed separately shortly after the incident and reported identical details. Forensic investigators determined that the incident was accidental. Histopathologic evaluation of the eyes revealed retinoschisis of the right eye with blood dissecting between the outer nuclear layer and the inner segment and extensive retinal hemorrhage of the left eye with multiple foci of hemorrhage involving all layers of the retina (**Figure 2**).

Comment. Retinal findings similar to our patient's have been described in adults with Terson syndrome. Keithahn et al[4] postulated that this results from a rapid rise in intracranial pressure being transmitted through the optic nerve, resulting in extravasation of fluid from intraretinal vessels and separation of the internal limiting membrane. We postulate a similar mechanism in our patient, who had a very rapid, massive increase in intracranial pressure, resulting in hemorrhage within

Figure 1. Fundus photograph of the left eye demonstrating elevated perimacular fold.

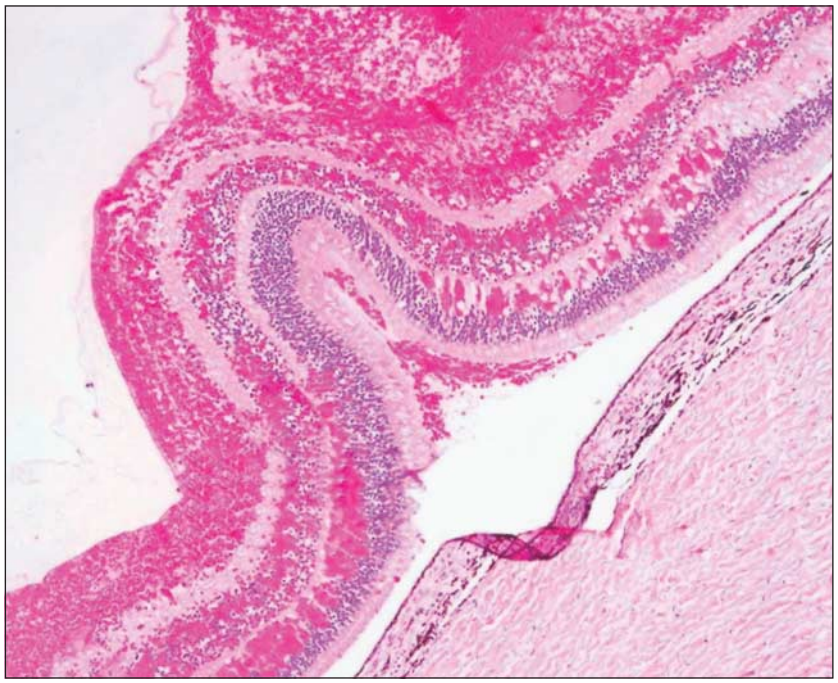

Figure 2. Postmortem photomicrograph of the left eye demonstrating perimacular fold in which the ganglion cells are obscured by severe hemorrhage. Hemorrhage is present in the intraretinal and subretinal layers (hematoxylin-eosin, original magnification ×20).

the perineural sheaths and separation of the retinal membranes at the outer nuclear layer.

This case demonstrates that a child may develop elevated perimacular folds from a severe head crush injury. The differential diagnosis of this abnormality includes SBS, but confusion with SBS can be eliminated by the corroborated history of severe crush head trauma and supportive physical findings.

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Financial Disclosure: None reported.

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Peripheral Lacquer Cracks as an Early Finding in Pathological Myopia

Lacquer cracks are uncommon findings in the posterior pole of highly myopic eyes. Herein, we report a case of a young man with an unusual localization of the lesion in the midperiphery of the eye.

Report of a Case. An 18-year-old man had a history of progressive myopia since childhood. No history of ocular trauma was obtained. The best-corrected visual acuity was 20/20 OU. The refractive error was −6.50 diopter (D) sphere OD and −2.75 D sphere OS (spherical equivalent). Binocular indirect ophthalmoscopic examination revealed myopic configuration of the optic nerve head and marked peripheral chorioretinal atrophy. In the temporal midperiphery, at approximately 2 disc diameters posterior to the vortex veins, 2 fine, arc-shaped lacquer cracks, about 1 disc diameter apart in the midportion and merging at the extremities, were present in the right eye, extending from the 9-o'clock to 1-o'clock positions (**Figure**).

Fluorescein angiography demonstrated early pseudofluorescence with no intraretinal or subretinal leakage of dye and faint hyperfluorescence in the late phase. Indocyanine green angiography showed 2 linear hyperfluorescences above the superotemporal vascular arcade (Figure insert). Central visual field examination showed no contraction.

Axial A-scan length measurements were 29.50 mm OD and 27 mm OS. A 25-mm transverse diameter of both globes at the anatomical equator was measured from the temporal to the nasal side, indicating a moderate equatorial scleral enlargement (normal value, 23.50 mm).

Comment. Lacquer cracks are uncommon findings in the posterior pole of highly myopic eyes with a prevalence ranging from 4.3% to 9.2%.[1] They appear to be caused by stretching of the coats of the eyeball with increasing axial myopia.[1] Although no direct statistical correlation is demonstrated with increasing axial lengths,[1] the highest incidence was found for values of 31.5 to 32.4 mm. This lesion is most probably associated with a preceding subretinal hemorrhage[2] and is often found within a posterior staphyloma. It involves young adults, the youngest patient to exhibit this change in 1437 eyes examined by Curtin et al[1] being a 19-year-old man. The eyes of men have a 2-fold incidence of lacquer cracks as compared with those of women.[1] Microscopically, a healed rupture in the retinal pigment epithelium–Bruch membrane–choriocapillaris complex has been observed.

Lacquer cracks have only been observed in the posterior pole of highly myopic eyes. In the present case, they were present in the midperiphery of the eye, a location that has never been described for these lesions to our knowledge. A possible explanation for the absence of peripheral lacquer crack descriptions could be related to their early occurrence in the disease course or to their rarity. Additionally, the reduced pigmentation of the equatorial zone of the eye as compared with the posterior pole could make lacquer cracks more difficult to identify, particularly in myopic patients with pale fundi.

A mild enlargement of the equatorial zone and a relatively well-

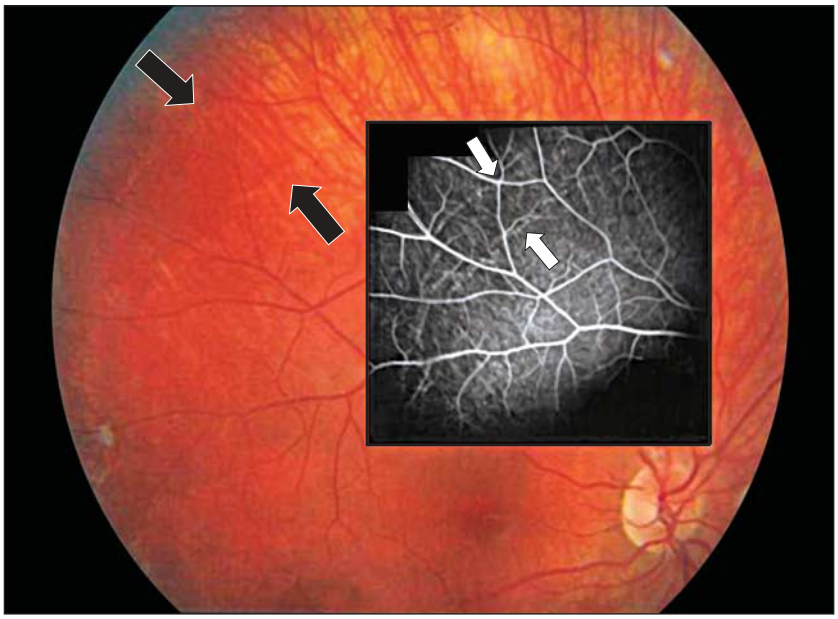

Figure. Right fundus of the patient showing 2 lacquer cracks temporal to the macula (arrows). Fluorescein angiogram demonstrates well-defined linear hyperfluorescences (arrows on insert).