G21 Histopathological Examination of Childhood Optic Nerve Sheath Hemorrhage

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The goals of this presentation are to identify the vasculature associated with the optic nerve sheath, discuss possible mechanisms of optic nerve sheath hemorrhage, and identify the difference between an intradural, a subdural, and a subarachnoid hemorrhage.

This presentation will impact the forensic science community by increasing awareness of the similar hemorrhage patterns of optic nerve sheath hemorrhage associated with fatal intracranial bleeding in young children from natural and traumatic causes.

Although Optic Nerve Sheath Hemorrhage (ONSH) has long been recognized as a complication of intracranial bleeding and sudden increased intracranial hypertension, the vascular source of optic nerve sheath intradural, subdural, or subarachnoid hemorrhage is unknown. Muller and Deck noted ONSH in 87% of 46 eyes examined after a sudden rise in intracranial pressure, while only 37% of the eyes had retinal hemorrhages.[1] Walsh and Hedges demonstrated that ONSH was not a direct extension from intracranial bleeding to the optic nerve and retina, but these hemorrhages occur secondarily to sudden distension of the optic nerve subarachnoid space from an acute rise in intracranial pressure, which presumably ruptures the bridging vessels in the optic nerve sheaths.[2]

Lambert *et al.* first described the presence of ONSH in Shaken Baby Syndrome (Abusive Head Trauma).[3] Subdural ONSH has since been observed in 65% to 100% of infants and young children diagnosed with Abusive Head Trauma (AHT). The most common site for AHT associated ONSH is in the immediate retrobulbar portion of the optic nerve similar to the findings in non-abusive intracranial bleeding observed by Muller and Deck and Walsh and Hedges. Lambert *et al.* suggested that an acute rise in intracranial pressure, transmitted to the optic nerve sheath, caused expansion of the sheath with rupturing of bridging veins. However, Wygnanski-Jaffe et al. suggest that orbital and optic nerve injury is more common and more severe in AHT than accidental head trauma.[4] They regard the eye and its orbit as a single unit, damaged directly by the mechanical process of shaking and surmise that there is a unique feature of repeated acceleration-deceleration injury, as seen in SBS, which leads to orbital trauma with retinal hemorrhages and ONSH. They suggest the increased frequency of hemorrhage at the anterior and posterior optic nerve sheath indicate that the junction of the globe and optic nerve and the firm posterior orbital attachments of all orbital structures create fulcrums leading to tissue damage at these locations.

Although optic nerve bridging veins have been implicated as the bleeding source of ONSH, this has not been confirmed histopathologically and does not adequately explain the increased concentration of intra-dural hemorrhage that is typically seen to communicate with subdural blood near the retro-bulbar optic nerve sheath. Returning blood from the retina and optic nerve, the central retinal vein may remain for some distance in the substance of the dural sheath before joining the superior or sometimes the inferior ophthalmic vein, or even the cavernous sinus directly.

This study hypothesized that serially sectioning and examining the retrobulbar optic nerve and surrounding sheath histologically should identify disrupted blood vessels within the intradural portion of the ONSH and thus explain the vascular source of ONSH associated with intracranial bleeding in children. Six archived autopsy cases with bilateral ONSH in children with fatal intracranial injuries were retrieved: two traumatic brain injuries from child abuse; two accidental traumatic brain injuries; and, two spontaneous non-traumatic intracranial hemorrhages.

Their ages were from 2 to 55.3 months and the male:female ratio was 1:1. Survival time from injury or incident to death varied from 2.75 to 57 hours. All had intra-cranial hemorrhage and bilateral retinal hemorrhages.

The optic nerves and surrounding sheaths were sequentially sectioned at 4 microns (thickness) with alternating Hematoxylin-Eosin (H&E) stained and unstained sections (50 sections/optic nerve: (25 H&E slides and 25 unstained slides)). All H&E histological sections were examined and assessed for intra-dural hemorrhage, subdural hemorrhage, subarachnoid hemorrhage, and perineural soft tissue hemorrhage.

A similar pattern of hemorrhage was identified in all six cases. There was no significant variation in the location of the extravasated blood in the natural, accidental, or child abuse deaths. All cases exhibited asymmetrical intradural hemorrhage that was centered on intra-dural blood vessels and all had perineural soft tissue hemorrhage. This study was unable to identify a definite disruption of the retinal artery, retinal vein, and/ or accompanying smaller vasculature with the number of sections examined. A similar pattern in these cases suggests a common source of the intra-dural, subdural, and subarachnoid blood.

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To identify the vascular source of the ONSH blood, additional serial sectioning must include the entire retrobulbar optic nerve and sheath including the entire intra-dural course of the retinal artery and vein. Careful orientation of the optic nerve segments will also facilitate assessment. Finally, application of an elastic-trichrome stain on alternating sections will assist in illustrating the intra-dural vascular walls that can be obscured by the extravasated blood.

References:

1. Muller PJ, Deck JHN. Intraocular and optic nerve sheath hemorrhage in cases of sudden intracranial hypertension. *J Neurosurg* 1974;41:160-166.
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3. Lambert SR, Johnson TE, Hoyt CS. Optic nerve sheath and retinal hemorrhages associated with the shaken baby syndrome. *Arch Ophthalmol* 1986;104:1509-1512.
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Forensic Science, Optic Nerve Sheath Hemorrhage, Retinal Hemorrhage

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