H116 Optic Nerve Sheath Hemorrhages Associated With Non-Traumatic Subdural and Subarachnoid Hemorrhage: A Case of Undiagnosed Congenital Hydrocephalus

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After attending this presentation, attendees will be able to recognize congenital hydrocephalus and understand the direct relationship between rapidly increased intracranial pressure and ocular and optic nerve sheath hemorrhages.

This presentation will impact the forensic science community by demonstrating the direct relationship between rapidly increased intracranial pressure and intraocular and optic nerve sheath hemorrhages and by reinforcing the need for thoroughness in the investigation and postmortem medical examination of all sudden childhood deaths.

Ocular and optic nerve sheath hemorrhages in children are frequently attributed to inflicted traumatic brain injury; when seen in conjunction with subarachnoid hemorrhage, they are often mistakenly considered pathognomonic of Shaken Baby Syndrome. A case of non-traumatic subarachnoid hemorrhage with optic nerve sheath hemorrhage (Terson Syndrome) from Miami-Dade County, FL is presented.

A 4-month-old, ex-35-week gestation male infant was born by cesarean section on April 1, 2014, as part of a dichorionic, diamniotic twin pregnancy, which was complicated by oliguria and pre-eclampsia. He had episodes of apnea and bradycardia, following birth, and was noted to have a large head. A cranial ultrasound revealed "minimally" dilated lateral ventricles. His pediatrician and neurologist's notes indicate that he was "normocephalic," but his head circumference increased from 34 centimeters at birth to 37 centimeters on April 25, 2014, to 38 centimeters on May 5, 2014, to 40 centimeters on May 12, 2014, and to 41 centimeters on June 2, 2014, which was recorded as a change from the 54th percentile to the 94th percentile from the first to second postnatal month without correction for prematurity. When corrected for prematurity, his head circumference is at the 98th percentile at birth and rises from there. A follow-up ultrasound on May 19, 2014, revealed an increase in the size of the lateral ventricles from 3mm to 5mm.

On July 27, 2014, the child was fussier following a nap, began to have difficulty breathing, and became non-responsive. He was found to have cerebral edema from anoxic/ischemic brain injury and eventually progressed to brain death. The medical notes indicate that this was due to a cardiac arrest, but there is no record of him ever suffering cardiac arrest or a period of hypoxia. Therefore, the only explanation for the anoxic/ischemic brain injury is an intrinsic issue within the brain where the intracranial pressure had increased and reduced cerebral perfusion. He had craniomegaly with progressively increasing hydrocephalus, which when untreated, eventually leads to elevated intracranial pressure and reduction of cerebral blood flow. The finding of retinal hemorrhage was said to be the result of non-accidental trauma, but anything that increased intracranial pressure can cause retinal hemorrhage.

Initially described in the early 20th century, Terson Syndrome referred to vitreous hemorrhage associated with subarachnoid hemorrhage. Today, the definition includes any degree of intraocular hemorrhage associated with intracranial hemorrhage and rapid elevations in intracranial pressure. Although some of the findings in this case mimic those described in cases of inflicted traumatic brain injury, this illustrates the importance of first excluding a natural disease process and thorough examination in all pediatric cases.

Intraocular Hemorrhages, Congenital Hydrocephalus, Terson Syndrome