G115 Sudden Unexpected Infant Death: Lymphocytic Meningoencephalitis With Multiple Retinal Hemorrhages

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After attending this presentation, attendees will learn how multiple retinal hemorrhages extending to the ora serrata are not diagnostically specific for abusive head trauma (shaken baby syndrome).

The presentation will impact the forensic science community by expanding the list of conditions in infants associated with multiple retinal hemorrhages that extend to the periphery of the retina.

This presentation will inform attendees of something they do not know–multiple retinal hemorrhages, involving the peripheral retina and extending to the ora serrata in infants, are not specific for abusive head trauma (shaken baby syndrome).

Numerous authors contend that specific ophthalmological findings in infants and young children with abusive head trauma (AHT) include numerous retinal hemorrhages that extend far into the periphery of the retina. Listed non-traumatic causes of retinal hemorrhages— coagulopathy, sepsis, meningitis, vasculopathies, increased intracranial pressure, and cardiopulmonary resuscitation—reportedly do not result in the multiplicity and peripheral distribution of the hemorrhages associated with AHT.

This case presents a 7¾-month-old male infant with multiple retinal hemorrhages extending to the ora serrata who died suddenly and unexpectedly from severe, diffuse lymphocytic meningoencephalitis. He had had rhinorrhea and upper respiratory congestion for about two weeks. His mother had been giving him acetaminophen every four hours. Otherwise, he had been healthy. He was placed down for a nap around 2:30 p.m. and was found unresponsive at about 3:00 p.m. Resuscitative efforts were begun immediately. A call was made to 911 at 3:05 p.m. and EMS arrived at 3:13 p.m. He was transported to the emergency department (ED) and arrived at 3:35 p.m. He was pronounced dead at 4:07 p.m. following 32 minutes of resuscitative efforts in the ED.

The medicolegal autopsy was performed 17 hours after he was pronounced dead. There was no evidence of trauma, skull fractures, intracranial hemorrhages or injury of the brain or spinal cord. Microbiological cultures of blood, trachea and lung were non- contributory. A skeletal survey did not reveal any evidence of acute or healing fractures. Subsequent toxicological analysis did not detect any licit or illicit drugs that caused or contributed to his death.

Postmortem monocular indirect ophthalmoscopy detected multiple retinal hemorrhages. The fundal hemorrhages in the left eye were over the posterior pole extending past the equator and abutting the ora serrata in all four quadrants; three small retinal hemorrhages were in the right globe.

His calvarial *dura* was smooth with areas of hyperemia and congestion of dural vessels, but no subdural extravasated blood or membranes were present. The dural venous sinuses were patent and the leptomeninges had no areas of extravasated blood. The cerebrum had a well-defined grey-white junction with no lesions in the cortex, white matter, or subcortical nuclei. The cerebral ventricles were of normal caliber and the ependymal lining of the ventricles appeared normal for age. The brainstem was normally developed with no gross abnormalities. The cerebellum exhibited normal folia, white matter, and dentate nuclei. The spinal cord had no areas of hemorrhage or exudates.

Microscopically, the cerebrum, brainstem, and cerebellum showed a multifocal lymphocytic infiltrate with numerous microglial nodules and neuronophagia. The inflammatory process involved the cerebral grey and white matter (including the basal ganglia), brainstem grey matter, and cerebellar white matter. The brainstem involvement was diffuse, with inflammatory foci in the midbrain, pons, and medulla; the spinal cord was not involved. No viral inclusions or areas of necrosis were seen. There was lymphocytic involvement of the cerebral leptomeninges and small perivascular lymphocytic collections were just deep to the ependyma. No significant ventriculitis was present. Immunohistochemical (IHC) stains for CD4 and CD8 showed a multifocal T-cell inflammatory infiltrate within the cerebral parenchyma and around blood vessels. An IHC stain for CD20 highlighted a smaller number of B-cells around blood vessels and within the parenchyma. The ICH stain for CD68 highlighted the microglial nodules and IHC stains for CMV, HSV-1, and HSV-2 were negative. The Centers for Disease Control performed IHC testing for panentero viruses and EV71 plus polymerase chain reaction (PCR) for enterovirus and parechovirus—all were negative.

Meningoencephalitis is a rare complication of common infantile viral infections. Most viral infections with central nervous system manifestations cause either meningeal involvement, namely aseptic meningitis, or a mild clinical syndrome of meningoencephalitis rather than a fatal form of encephalitis. The causative agent in

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this case was not

apparent despite IHC and PCR testing for enteroviruses and parechoviruses. Of particular interest, this infant had numerous retinal hemorrhages in the left globe distributed posteriorly, equatorially, and peripherally—a finding considered by many authors unique to AHT and indicative of repetitive acceleration-deceleration injury (shaken baby syndrome). It is imperative that forensic pathologists not equate multiple retinal hemorrhages with a peripheral distribution exclusively with AHT. Postmortem ocular findings must not be interpreted in isolation, but correlated with the circumstances of the death plus the anatomic and histopathologic findings.

Retinal Hemorrhages, Lymphocytic Meningoencephalitis, Abusive Head Trauma

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