CASE REPORT

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Periadventitial Extracranial Vertebral Artery Hemorrhage in a Case of Shaken Baby Syndrome

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ABSTRACT: We report a case of a two-month-old boy who became unresponsive in the sole custody of his father. Resuscitation efforts on route to the hospital were able to restore the infant's heart beat. However, neurologic function never recovered. Autopsy revealed massive cerebral edema, recent subdural, and subarachnoid hemorrhages, bilateral retinal hemorrhages, and cervical spine ligament hemorrhages. Separation of individual cervical vertebrae showed extensive, bilateral, periadventitial vertebral artery hemorrhages between C1 and C4, with corresponding luminal compression of the vertebral arteries. The importence of this previously unreported phenomena of periadventitial vertebral artery hemorrhage in the setting of shaken baby syndrome is discussed.

KEYWORDS: forensic science, shaken baby syndrome, periadventitial hemorrhage, extracranial, vertebral artery, postmortem technique

Fatal shaken baby syndrome encompasses a clinical history of whiplash shaking of an infant's head, and autopsy findings of cerebral edema, subdural and/or subarachnoid hemorrhage, retinal hemorrhages, and diffuse axonal injury (1–3). Associated postmortem findings may include fracture or dislocation of the cervical spine, hemorrhage in the spinal ligaments, and finger tip contusions of the shoulders, arms, chest, and abdomen. To our knowledge, periadventitial vertebral artery hemorrhage has not been previously reported in shaken baby syndrome. We describe such a case, propose an autopsy technique to check for this finding, and discuss its mechanism.

Case Report

This 2-month-old male infant was at home in the sole custody of his father. According to the father, after the baby was fed a bottle of milk and placed in his crib, "he vomited and suddenly became unresponsive." Emergency medical services were summoned and found the baby a reflexive and with fixed and dilated pupils. He was intubated and cardiopulmonary resuscitation was begun. On arrival to a local hospital his temperature was 96°F (per rectum), his pulse was 78, and his blood pressure was 44/doppler. He had no spontaneous respirations. Physical examination identified a bruise in his right clavicular area. His body was flaccid and unresponsive to stimuli. Laboratory coagulation time and platelet count studies were in the normal range. He was administered mannitol, epinephrine, and dopamine. A head CT scan showed severe cerebral edema with complete obscuration of gray/white matter demarcation and subdural hemorrhage. Ophthalmologic exam demonstrated bilateral retinal hemorrhages. The clinical diagnosis of whiplash shaken infant syndrome was strongly suspected by hospital staff. After he was stabilized, he was transferred to a tertiary care hospital. For four days, he was maintained on mechanical ventilation and medications for blood pressure support and was then pronounced dead. His mother consented to donation of his organs, and his heart, lungs, liver, small bowel, and pancreas were procured.

Autopsy revealed massive cerebral edema, recent subdural, subarachnoid, and intracerebral hemorrhages, hypoxic/ischemic brain injury, cervical to lumbar spinal cord necrosis, bilateral recent retinal hemorrhages, C1 verterbral ligament and adjacent posterior neck muscle hemorrhages, C1 ligamentous laxity, and a superficial contusion over the right clavicle.

Separation of individual cervical vertebrae showed extensive, bilateral, periadventitial vertebral artery hemorrhages between C1 and C4 (Fig. 1). Microscopic examination confirmed the above finding and identified associated luminal compression of the vertebral arteries within the transverse foramina (Fig. 2). Small, periadventitial vessels were torn, however, the vertebral arteries themselves were completely intact. No vertebral artery thrombus, aneurysm, or dissection was identified.

Discussion

The autopsy findings of acute subdural, subarachnoid, and retinal hemorrhages, and cerebral edema support the clinical suspicion of shaken baby syndrome in this case. Further postmortem evidence of whiplash shaking included the C1 vertebral ligament and adjacent posterior neck muscle hemorrhages and the C1 ligamentous laxity. The clavicular contusion was possibly a result of the perpetrator's firm grasp on the infant while he shook his son's head and neck back and forth. The absence of scalp contusion or laceration, epidural hemorrhage, and skull fracture affirmed that blunt head trauma played a minimal role if any in the infant's death. The

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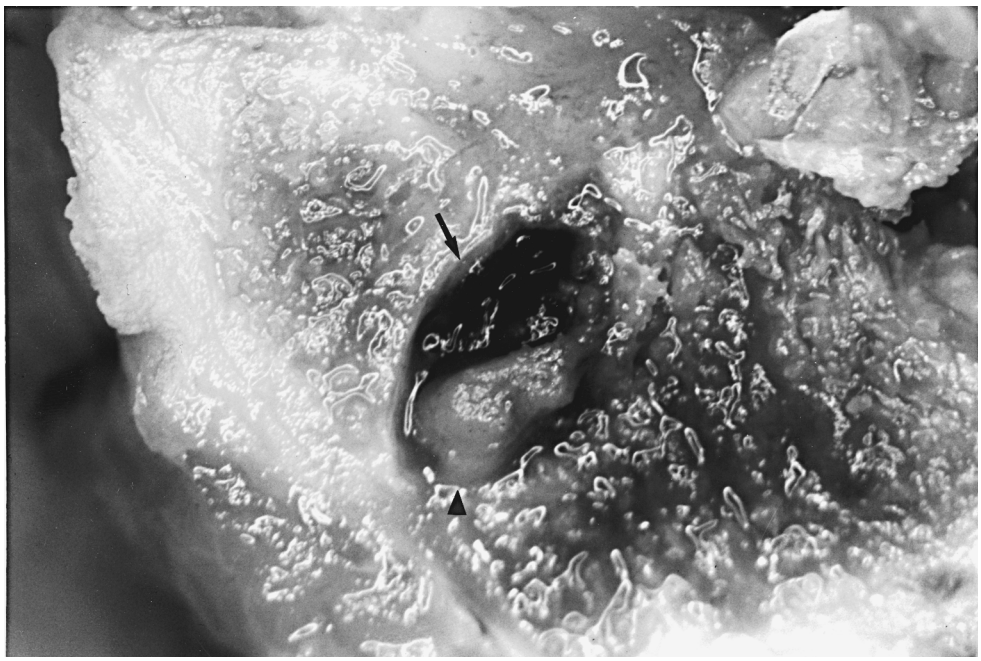

FIG. 1—*Cross sectional view of C1 vertebra centered on the left transverse foramen. Within the transverse foramen, collapse of the left vertebral artery (arrow head) is due to external compression from extensive periadventitial hemorrhage (arrow).*

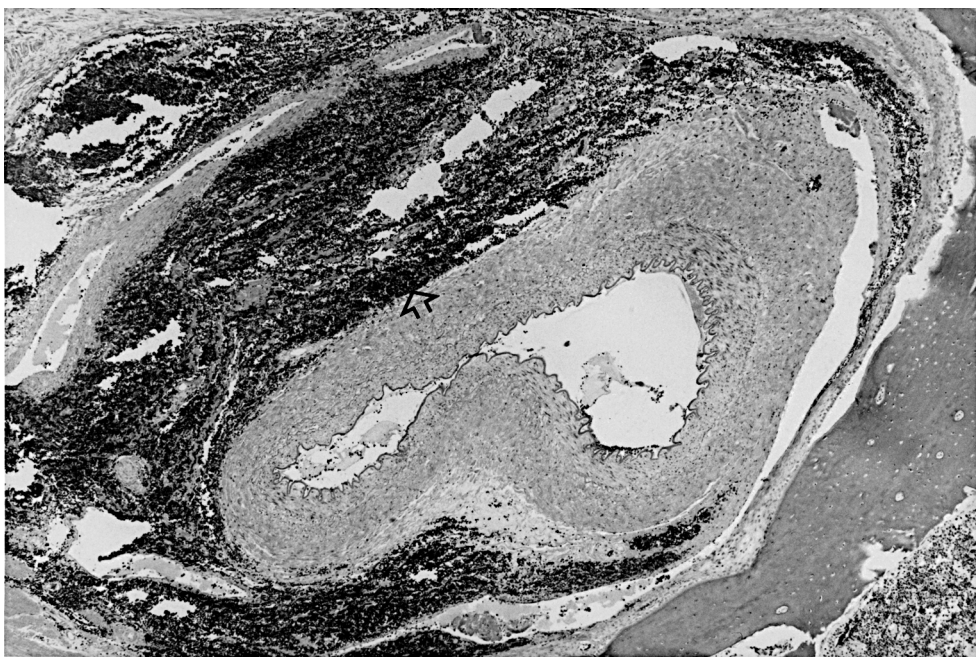

FIG. 2—*Low power microscopic view of the left transverse foramen of the C1 vertebra. Note compression of the left vertebral artery. This is due to periadventitial hemorrhage (arrow head). Hematoxylineosin, original magnification ×10.*

infant's acute intracranial and posterior neck hemorrhages were clearly of a traumatic origin, especially since he presented with normal blood coagulation and platelet values.

In this case of shaken baby syndrome, we postulate that the repetitive acceleration-deceleration of the infant's head produced stretch and shear forces on small periadventitial vessels. As a result, these vessels were torn and they bled into the periadventitial space of the transverse foramina. Because of the "tight" space that the vertebral artery occupies within the transverse foramen of the cervical vertebrae, hemorrhage in the periadventitial space caused external compression of the vessel wall and constricted the lumen of the vessel. If the periadventitial hemorrhage is extensive, col-

lapse of the lumen may occur and vertebral artery blood flow diminish. Bilateral occlusion of the vertebral arteries would likely cause ischemic injury/infarction of the anterior cervical spinal cord, brainstem, and cerebellum due to loss of collateral supply.

In our particular case, severe luminal compromise of both vertebral arteries was documented at autopsy. Although extensive necrosis of cerebellum, brainstem, and cervical spinal cord were present, it is unclear now much the vertebral artery occlusion contributed to this, because of the presence of more wide spread hypoxic/ischemic injury in the brain, most likely due to the infant's cardiac arrest.

Reports of traumatic vertebral artery injury in the pediatric population have been restricted to cases of thrombosis, distal embolization, aneurysm, and dissection (4–8). To our knowledge, vertebral artery injury has not previously been described in cases of infant whiplash shaking—so called shaken baby or shaking-impact syndrome. In one case described by Lam et al. (9), a pericallosal artery traumatic aneurysm developed from a well documented incident of whiplash shaking. They ascribed the vascular injury to shearing forces, the same mechanism we invoke in explaining periadventitial hemorrhage of the extracranial vertebral arteries in our case of shaken baby syndrome.

We suggest that in the postmortem examination of infants suspected to have incurred fatal whiplash head trauma, when the pathologist sees trauma to posterior neck ligaments or documents deep neck muscle hemorrhage, the autopsy should include examination of the individual cervical vertebrae. This is best accomplished by using a posterior approach with the cadaver in the prone position. After posterior unroofing (by incising all of the bony spinal laminae and removing them with the spinous processes) and amputation of the entire dura and spinal cord, the upper (C1 to C5) cervical vertebrae should be removed *en bloc* by disarticulation of C1 from the skull base and C5 from C6. Next, the individual upper cervical vertebrae are separated by incising their connecting ligaments. The vertebral bodies can be viewed *en face* and a cross section of the vertebral arteries may be inspected. This examination also permits close examination for vertebral fractures and dislocations.

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