Forensic Sci Med Pathol
DOI 10.1007/s12024-013-9525-4

COMMENTARY

"Shaken baby syndrome" and forensic pathology

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Accepted: 26 December 2013

Dr. Byard's summary of the various features of the so-called "shaken baby syndrome" (SBS) is intended to stimulate discussion about this controversial entity [1]. In particular, he reminds us: that the various labels used for such cases involve preconceptions regarding intent (e.g., *inflicted*, *abusive* or *non-accidental* head injuries) or pathogenesis (e.g., *shaken* baby syndrome and even Byard's "lethal craniocerebral *trauma*"); that the brain injury in some cases may be the consequence of direct brainstem injury (i.e., Byard's "*cervicomedullary syndrome*"); and that we should not dismiss shaking as an etiology just because accidental and natural processes may mimic the triad of encephalopathy, subdural bleeding, and retinal hemorrhages, which are conventionally linked to a shaking etiology.

We will only address the significance of the subdural bleeding, whose etiology is most often regarded as traumatic. The characteristic pattern of subdural bleeding in SBS is of small, bilateral, non-space-occupying subdural hematomas (SDHs). In 1972 Guthkelch [2] suggested that this bleeding came from the bridging cortical veins, that the blood entered an established subdural "space," and that this conclusion fitted with the general idea of stretching and tearing of these vessels during the supposed shaking of the infant. Guthkelch's concept regarding the etiology of SDH in SBS then became widely accepted [3].

However, the significance of such SDH has come under scrutiny in terms of its etiology, location, and source. Of particular importance for assessing SDHs in the triad and suspected cases of SBS are the following points:

1. There is no evidence of a naturally occurring subdural space. Rather, the apparent subdural "space" is the result of a pathological or artifactual process that splits open the poorly cohesive dural border layer cells that form the outer layer of the dura-arachnoid interface [4, 5]. Accordingly, so-called "subdural" bleeding starts within the dural border layer.
2. The vasculature associated with the dural border layer includes the following:
   a. The bridging veins are large caliber vessels draining cortical venous blood from the brain to the systemic circulation. They are in the subarachnoid space for most of their course before they penetrate the dura, including its border cell layer, and enter the superior sagittal sinus. The walls of these "bridging" veins are markedly thinner in the dural border layer than in their subarachnoid segments because of scant collagen and the lack of arachnoid trabecular cells [6].
   b. There is a dense venous plexus in the dura that drains into the dural sinuses; these plexuses are prominent in the parasagittal location, more pronounced in infancy, and more conspicuous in the caudal region of the falx [7, 8].

These observations indicate that SDH may result from tears in the large but thin-walled segments of the bridging veins as they pass through the dural border layer or from

This comment refers to the article available at doi:10.1007/s12024-013-9514-7.

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Published online: 25 January 2014

rupture of the narrow vessels of the dural venous plexuses. Large space-occupying SDHs would be the expected result of the former whereas the latter should lead to smaller amounts of and more diffuse "non-space-occupying bleeding," particularly in the parasagittal and parafalcine regions. The pattern of the SDH in the triad, being scant, bilateral, and parafalcine, suggests bleeding from the dural plexuses as opposed to the bridging veins.

Whereas large space-occupying SDHs are an accepted consequence of blunt force head injury in children and adults, the etiology of the dural venous plexus pattern (DVP) of SDH is less certain. However, this pattern is seen in some cases of non-traumatic hypoxic-ischemic encephalopathy (HIE), particularly in young infants with complex medical illnesses [9]. These observations imply a cause and effect relationship between subdural bleeding and HIE, perhaps as a result of hypoxic injury and consequent congestion and rupture of the small vessels of the dural venous plexus. However, because not all infants with HIE present with SDH [9, 10] additional factors are likely to contribute to the DVP pattern of SDH, including blood clotting abnormalities, metabolic derangements and/or the effects of increased intracranial pressure.

These observations indicate that the SDH in the triad is the result of bleeding from the dural plexuses rather than from torn bridging veins, a pattern of bleeding that may be associated with trauma or be of natural etiology. Such bleeding alone is therefore unreliable evidence of an "inflicted" head injury or SBS.

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