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Investigating the possibility and probability of perinatal subdural hematoma progressing to chronic subdural hematoma, with and without complications, in neonates, and its potential relationship to the misdiagnosis of abusive head trauma

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ABSTRACT

The high incidence of subdural hematoma (SDH) from birthing was first identified with MRI by Looney in 2007 and was then more accurately determined by Rooks in 2008. Rooks screened 101 "normal" deliveries and demonstrated that 46% of the babies in her series and by inference, approximately 46% of the 4 million born normally in the US have SDH that formed in the perinatal (birthing) period during labor. Both metabolic strain and physical forces exerted on the head damage the capillaries within the dura (the intradural capillary bed), which is the source of the blood in the SDH that results from labor and delivery or at times from labor alone. While child abuse pediatricians relying on Rooks, maintain that no permanent complications result, her study was limited to 101 subjects and the sole criteria for resolution was the resolution of the SDH as seen on follow-up MRI. In fact, Rooks did have one patient (1%) who had complications that lead to symptoms and findings often associated with abuse. The purpose of this article is to explore if there is a complication rate for perinatal (PSDH) that supports that 1% of complications that are definable by different criteria. Next, if there are complications, how many of the roughly 2,000,000 cases of perinatal acute subdural hematoma every year in the United States will suffer them? Then, what are the clinical manifestations of the complications if they occur? Lastly, do the complications cause or mimic some or all of the findings that are offered by board certified child abuse pediatricians as evidence of child abuse? The article argues that a small percentage, but significant number of neonates, suffer birth related complications and findings secondary to the development of chronic subdural hematoma CSDH) that are often misdiagnosed as abusive head trauma.

1. Introduction

The high incidence of subdural hematoma (SDH) from birthing was first identified by MR by Looney in 2007 [1] and then more accurately determined by Rooks in 2008 [2] when she screened 101 "normal" deliveries. Dr. Rooks' data suggests therefore that 46% of the 4 million neonates born "normally" in the US were, in fact, not normal. Instead they had SDH that formed in the perinatal (birthing) period from both metabolic strain and physical forces exerted on the head, specifically the dura; the most external of the 3 layers of the meninges. The dense capillary bed within the dura (the intradural capillary bed), is the source of the blood in the SDH, results from labor and delivery or at times from labor alone. While Rooks wrote that no permanent complications resulted, her study was limited to 101 subjects and the sole criterion for resolution was the resolution of the SDH as seen on follow-up MRI done on only 18 of the 46 positive MRI's done at birth. In fact, Rooks did have one patient (1%) who had complications that lead to findings often associated with abuse. The purpose of this article is to explore if there is a complication rate for perinatal subdural hematoma (PSDH) that supports that 1% or complications that are definable by different criteria. Next, if there are complications, approximately how many of the roughly 2,000,000 cases of perinatal acute subdural hematoma every year in the United States will suffer them? Then, what are the clinical manifestations of the complications if they occur? Lastly, do the complications cause or mimic some or all of the findings that are offered by board certified child abuse pediatricians as evidence of child abuse?

2. The pathophysiology of perinatal subdural hematoma

PSDH starts with intradural capillary damage during labor. It may occur during contractions from direct external pressure on the dura, hypoxic (low oxygen) metabolic stress throughout the cranium [3], transient venous channel occlusion in the cranial vault due to compressive forces deforming the skull, and/or activation of complex neurologically mediated vasomotor control systems triggered by intracranial pressure changes [4].

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Any augmentation to labor, (pitocin or instrumentation forceps or vacuum extraction), or forceful pushing combined with any bony dystocia (too small a pelvis) or malpositioning of the fetus, increases the forces on the head during contractions. Each of these factors may increase the probability of PSDH and the amount of bleeding that occurs. In fact, Rooks found that 84% of neonates with cephalohematoma (molding of the head due to bleeding beneath the scalp) had SDH. She also found 18% of all c-sections had PSDH and 75% of those had been in labor and had pitocin drips before c-section. The newly identified epidemic of vitamin D deficiency [5] and calcium deficiency [6], in some studies affecting more than 85% and 43% of mothers at birth respectively, and passed on to their neonates in roughly equal measure, weakens the structural integrity of the skull [7] in utero, causing increased deformation of the skull of fetuses during labor, and likely contributes to intracranial damage and PSDH as well.

When the capillaries fail, blood first fills the intradural space with blood forming an intradural hematoma (IDH). The falx, dividing the halves of the brain and partially protected from trauma by its location, shown below in these example cases, seems to be particularly vulnerable to metabolic strain and often bleeds first, primarily due to hypoxic stress [8] (see Fig. 1A–C). The dura around the brain is subject to the wider spectrum of insults, as mentioned above, but SDH is the primary detectable finding as shown in Rooks.

The leaking blood from the dural capillary plexus, studied by Squier et al., [4 or 19] when sufficiently copious, overflows the intradural space into the subdural space easily dissecting off the arachnoid layer of the meninges from the dura. The arachnoid is loosely adherent to the dura's inside surface, the dural border cell layer (DBCL), and when dissected off by leaking blood, an anatomical disruption occurs. The DBCL is made up of loosely adherent cells, capillaries, no extracellular collagen, and enlarged extracellular spaces. The blood that collects between the dura and the arachnoid forms the conventional SDH, which is contiguous with the DBCL. In some cases of minimal intradural bleeding, IDH can be seen without SDH as shown in the Fig. 2A.

One of the first manifestations of this PSDH is an immediate disruption of the cerebrospinal fluid (CSF) absorption function of the dural capillary plexus. This occurs when the intradural space is flooded and enveloped in blood (see Fig. 5). There appears to be an obstructive component to this intradural hemorrhage (IDH) and a resultant increase in intracranial pressure occurs. This increased pressure immediately forces the skull bones to push outward. Due to the fact that the brain is relatively incompressible and the skull bones relatively mobile due to growth areas between bones that are cartilage (called sutures) over time (hours to weeks) the intracranial (IC) pressure moves the bones out and a space between the brain and the skull forms. The space that forms is abnormal, enlarged, fluid filled and is called a hygroma. Hygroma is a generic term for any abnormal fluid filled space. In the cranium it is also commonly referred to as extra-axial space (space outside the brain tissue) among many other terms listed in Table 1 below. The space that forms around the brain and the fluid within the hygroma space is distinct from the subdural fluid. The presence of some degree of extra space is a quite common incidental finding in the post natal period and its possible relationship to PSDH is discussed below. In a study of neonatal intensive care unit (NICU) graduates, a group vulnerable to PSDH, 44% left the NICU with moderate to extreme enlargement of the subarachnoid (SA) space [9]. Maximum growth of the space in these babies was noted between 3 and 12 months, a period in which abuse is most commonly diagnosed [9]. The overall prevalence of smaller degrees of post PSDH hygroma has not been studied or quantified as of yet but may be common in absolute numbers as Rooks demonstrated in her study where 1% of the patients had increased subarachnoid space at 5 months (the "example neonate"). While the number may be smaller, that 1% would project to 40,000 babies per year

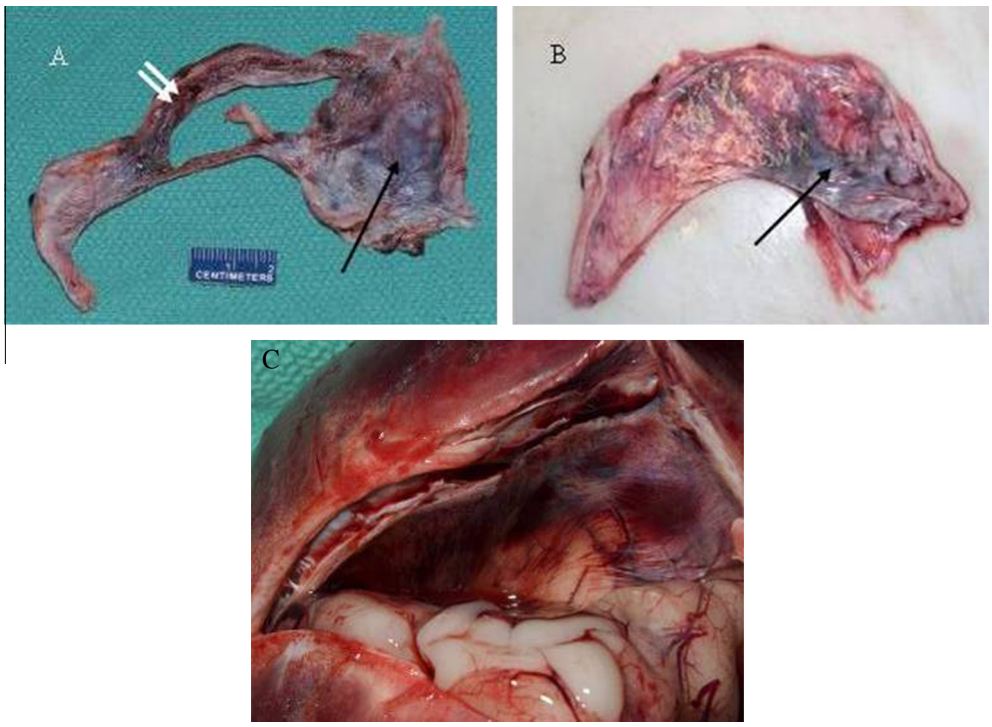

Fig. 1. (A and B) Widespread interhemispheric intradural hemorrhage (IDH) in a neonate. IDH in the falx and tentorium from two neonates. Note the blood filling the intradural space in both specimens of falx (single arrows pointing to larger collections, but blood is widely distributed). Note the blood in the upper falx in A near to the superior sagittal sinus (double arrows) where the capillary bed is most dense (see also Fig. 3). (Images from author's files). (C) Intra-falcine hemorrhage in situ in a nonabused neonate. Intra-falcine hemorrhage (arrow and throughout) in situ at autopsy in an 11-day-old neonate found dead in bed; no anatomic cause of death evident. No allegations of abuse (Images from author's files).