

Bridging veins and autopsy findings in abusive head trauma

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Abstract Bridging veins are crucial for the venous drainage of the brain. They run as short and straight bridges between the brain surface and the superior sagittal sinus in the subdural compartment. Subdural bleeding is a marker for a traumatic mechanism (i.e., acceleration/deceleration, rotational and shearing forces due to violent shaking) causing rupture of the bridging veins. Demonstration of bridging vein rupture allows the unequivocal diagnosis of a traumatic mechanism and should therefore be a routine part of the postmortem in cases of subdural hemorrhage.

Keywords Abusive head trauma · Non-accidental injury · Subdural hemorrhage · Children · Bridging veins · Autopsy

Introduction

Shaken baby syndrome, now called abusive head trauma or non-accidental head injury, is the result of violent shaking with or without impact, which produces acceleration/deceleration and rotational and shearing forces. Diagnosis is based on the combination of a severe acute encephalopathy and subdural or subarachnoid hemorrhage. Rotational movement of the brain within the cranium creates shearing or tearing of the bridging veins on its surface and thus bleeding into

the subdural compartment [1]. Bridging veins are crucial for venous drainage of the brain, carrying blood from the surface of the brain to the dural venous sinuses.

Retinal hemorrhages are found in 80% to 90% of cases, and while their absence does not rule out the diagnosis, their presence strengthens it. The lack of a reliable or appropriate explanation for the symptomatology is common and should be included in the diagnostic criteria [2].

Mortality is high – as much as 20% to almost 30% in some studies [3, 4]. There are two distinct groups of non-accidental head injury cases: those who will die and those who will survive. This depends on the severity of the neurological damage; the cause of death is traumatic encephalopathy. Subdural hemorrhage is the marker for trauma (i.e., acceleration/deceleration, rotational and shearing forces) that ruptured the bridging veins. Bleeding, by itself, is not necessarily fatal to the infant. Indeed, recent MRI studies have shown that up to 46% of apparently healthy, asymptomatic term neonates have subdural bleeding after an uneventful birth [5]. That subdural bleeding is different than that caused by violent shaking, however; it is located in the posterior half of the calvarium and its precise etiology is unknown. As Rooks et al. [5] speculate:

“Perhaps compressive force from the uterus (during labor) is a causative factor (...) in that there may not only be increased prolonged propulsive forces but also increased molding and overlapping of sutures, which may lead to failure of tensile strength of the stretched vessels. Increased pressure during the labor process may augment the intracranial venous pressures, which also may be an additional factor leading to subdural hemorrhage (...) The overall birth weight of neonates with subdural hemorrhage was also significantly higher, which may have resulted in increased circumferential pressure forces from the birth canal.”

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In any case, most of this subdural bleeding resolves on its own within a month [5].

The amount of blood at the surface of the brain in the infants with non-accidental head injury who die may be minimal. First, because the bleeding is venous, and therefore slow, and second because the encephalopathy causes so much brain swelling that it presses the ends of the ruptured vessels against the skull [6].

Demonstration of ruptured bridging veins at autopsy is essential, as it confirms that the mechanism behind the bleeding is traumatic (acceleration/deceleration and rotational and shearing forces).

Anatomy

The dura mater is the thick, outermost layer of the meninges. Not simply a fibrous covering, the dura is “a complex, vascularized and innervated structure” [7]. As described by Adeeb et al. [8]:

“It is composed of three layers: the endosteal (i.e., periosteal) layer, the meningeal layer, and the border (limiting) cells layer. The endosteal layer, the outermost layer, forms the periosteal lining of the inner surface of the calvarium, to which it is strongly adherent, mainly at the base of the skull (...) The dural sinuses are venous channels that run between the endosteal and meningeal layers of the dura...They receive blood from the cerebral veins through the bridging veins (...) The superior sagittal sinus [SSS] occupies the upper fixed margin of the falx cerebri. (...) The border cells layer is directly applied to the arachnoid mater and with no or few recognizable junctions between them.”

Nor is there any space between them.

According to Haines [9]:

“The arachnoid consists of an outer part, the arachnoid barrier cell layer, and an inner portion, the arachnoid trabeculae which bridge the subarachnoid space. Arachnoid barrier cells are (...) closely apposed to each other, and joined by many cell junctions; in this layer there is little extracellular space and essentially no intercellular material (...). There is no evidence of an intervening space between the arachnoid barrier cell layer and the dural border cell layer that would correlate with what has been called the subdural space.”

The latter is therefore better called the subdural compartment.

“When a tissue space is created in this general area of the meninges it is the result of tissue damage and

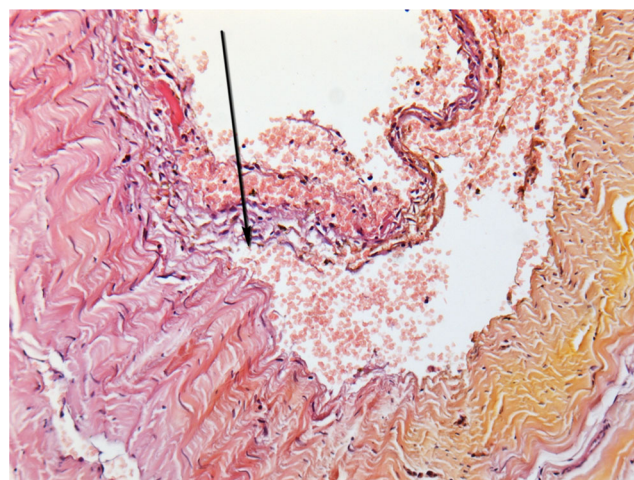


Fig. 1 Dura mater of a 3-month-old with subdural hemorrhage. There is cleaving of the dural border cell layer (arrow) by subdural bleeding. (HESX10)

represents, in most instances, a cleaving open of the dural border cell layer” (Fig. 1). Accumulation of blood in this layer is responsible for the pathology of subdural hemorrhage. “A survey of reports describing the morphology of the inner and outer capsule of so-called subdural hematomas in humans reveals that dural border cells are found in both parts of the capsule [9].”

According to Mack et al. [7]:

“Although subdural hemorrhage is frequently traumatic, there are nontraumatic conditions associated with subdural hemorrhage, and the inner dural plexus is a likely source of bleeding in these nontraumatic circumstances.”

There may also be oozing of blood from the dural end of a bridging vein with increased pressure in the vessel lumen.

Traumatic “hemorrhage results from rupture of the bridging veins, which connect the veins draining the brain into the dural sinuses. Through (their) course, these veins traverse the dural border layer and have a weak attachment with its cells, in contrast to its strong attachment to the cells of the arachnoid mater” [8].

Rupture of the bridging veins results in bleeding into these spaces, i.e., the subdural compartment. The amount of bleeding depends on whether the rupture is total or partial. Thus, subdural bleeding can be only blood within the dural border cells layer.

In a review of the literature, Mortazavi [10] states:

“An intracranial bridging vein is defined as a vessel located in the subarachnoid space that empties into the dural venous sinuses. Three types of bridging veins have

been investigated: cerebellar bridging veins, temporal bridging veins, and anterior frontal cortical bridging veins. They are most often located near the superior sagittal sinus (SSS) and anterior portion of the temporal lobe.”

There are two types of cerebellar bridging veins: the vermician/midline, which empty into the confluence of sinuses and are found along the midline, and the hemispheric/lateral groups, which empty into the tentorial sinuses and are found on the hemispheric surface of the tentorium cerebelli [10]. Though not yet demonstrated, it is possible that a rupture of these veins is responsible for infratentorial posterior fossa hemorrhage.

Again, from Mortazavi [10]: “The temporal bridging veins (travel) over the temporal lobe laterally (...) and veins can be divided into a mediodorsal group that enters into the superior sagittal sinus and a posteroinferior and middle cerebral group that opens into the lateral sinus.”

There are three types of anterior frontal cortical bridging veins: lateral convexity, medial surface and basal surface. They drain into the superior sagittal sinus [10].

Bridging veins are crucial for the venous drainage of the brain. They carry blood from the brain’s surface to the venous sinuses of the dura. They run as short, straight bridges between the brain surface and the superior sagittal sinus in the subdural compartment, nearly perpendicular to both (Fig. 2). As Yamashima and Friede [11] point out, “Between these two points, bridging veins take a straight course with no tortuosity to allow for possible displacement of brain (...) The cranial ends of bridging veins are fixed to the rigid dura mater, while the cerebral ends are attached to the movable hemisphere” (Fig. 2). “The falx protects the brain, and thus the bridging veins, from lateral displacement, but there is no protection against antero-posterior movement” [11].

In a light and electron microscopic study of the bridging veins, Yamashima [11] found that there was enormous variation in the vein wall thickness in the subdural portions, which ranged between 10 and 600 microns, while the walls in the subarachnoid portions were relative uniform in thickness, ranging from 50 and 200 microns. Electron microscopy showed that the “wall contained an abundance of collagen fibres and a variable number of elastic fibres” [11]. The collagen fiber distribution within the bridging vein wall was compact in the subarachnoid portion, whereas it was loosely woven in the subdural portion. Moreover, the bridging veins in the subarachnoid portion were tightly reinforced by arachnoid trabecular cells, while in the subdural portion there were only scattered, attenuated dural border cells.

In terms of collagen fiber arrangement, “there was always a tendency for circumferential fibres to prevail compared with longitudinal ones, particularly in the subdural part. This

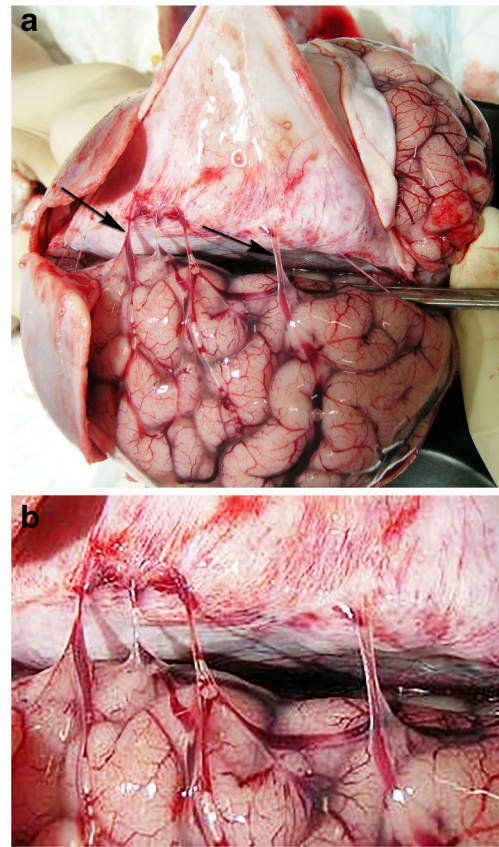


Fig. 2 Normal bridging veins of a male, 4 months old infant. **a** Bridging veins (arrows) run as short, straight bridges between the surface of the brain and the superior sagittal sinus in the subdural compartment. **b** The cranial ends of bridging veins are attached to the rigid dura mater, while the cerebral ends are attached to the movable hemisphere

pattern would render the vein’s wall more resistant to distension while reducing its resistance to traction” [11].

In Yamashima’s words,

“All these features imply that bridging veins are more fragile in the subdural portion than in the subarachnoid portion. Indeed, the differences in the structure of the two portions of the bridging vein were so striking that one is less inclined to ask: ‘Why do bridging veins rupture easily?’ but rather: ‘Why isn’t the rupture of bridging veins more common, especially in the subdural portions?’ (...) The anteroposterior acceleration or deceleration of the head can easily cause traction of the bridging veins, and they will rupture at their weakest point, that is, in the subdural space” [11].

Regarding the possible role that an enlargement of the subarachnoid space might have in facilitating bridging vein rupture, due to their stretching in this space, Raul [12] used a finite element model of a 6-month-old child’s head, varying the size of the subarachnoid space. He showed that enlargement of the subarachnoid space has

a damping effect that reduces the relative brain/skull displacement. These numerical simulations suggest that the benign enlargement of the subarachnoid space should no longer be considered a risk factor for subdural bleeding.

The number and diameter of bridging veins are variable. Ehrlich [13] used an X-ray technique to examine 350 cadavers (240 with closed head trauma and 110 without head trauma) ages 2 months to 96 years (mean age: approximately 50 years).

“During autopsy, after the skull is opened in the horizontal plane, the calvarium, together with the upper half of the brain, is cut with a long knife (...) After removing the calvarium, together with the upper half of the brain, a balloon catheter is placed in the dorsal opening of the superior sagittal sinus and blocked. About 5–15 ml of contrast medium (barium sulfate solution) is instilled into the sinus with a syringe. Axially directed filling radiographs are acquired in swift succession” [13].

A leak from a torn bridging vein is easily visible. This procedure was used to count the number of bridging veins per case and to measure their diameters. There was wide variation:

“from 9 to 31 veins, with an average of 17 bridging veins per brain. In over 60% of the cases 50–75% of the bridging veins were classified as symmetric. In conjunction with an increase in the number of bridging veins per brain, a corresponding decrease in their average diameter was determined. More than half of the brains examined had a total number of veins in line with the mid range of 13–20 and also fell within the mid range in diameters 1.9–2.5 mm. In brains with less than 12 bridging veins the average diameter, with a probability of 95%, was shown to be 2.2–2.7 mm. In those cases with more than 21 bridging veins, the average diameter was between 1.8

and 2 mm. Thus, regarding the numbers and diameters of the veins, cases could be separated in two groups: one with many bridging veins of small diameter and another with few veins of a large diameter” [13] (Fig. 3).

This may play a role in susceptibility to bridging vein rupture.

Demonstration of bridging vein rupture

Investigation for bridging vein rupture should be a routine postmortem procedure in cases of subdural bleeding. The finding of bridging vein rupture definitively identifies mechanical trauma as the cause of a child’s death, and the presence of several torn bridging veins indicates a significant degree of trauma [14]. Conversely, a finding of intact bridging veins rules out trauma as the cause of subdural bleeding.

To avoid the technique mentioned above, which has the disadvantage of cutting the brain [13, 15], Stein first performed non-contrast computed tomography (CT) of the infant’s head to document its original state, then instilled contrast agent directly via fontanel puncture into the superior sagittal sinus and repeated the scan. This method is minimally invasive and can be done quickly and conveniently on clinical CT systems, though it requires an open fontanel. It shows even very small deep and superficial cerebral veins – the bridging veins, in particular – without damaging them. Ruptures appear as extravasation of the contrast medium, which helps to locate them at autopsy [16].

Another way to diagnose ruptured bridging veins is to look for thrombosis with careful analysis of the vertex by CT or, even better, by MRI. Imaging can directly show tubular-shaped clots, which are typically suggestive of acute bridging vein thrombosis. Although these tubular clots might also represent clotted blood in the subarachnoid space, they should always be considered markers of acutely disrupted veins (Fig. 4). Though rarely mentioned in the radiologic literature, this sign is very important to recognize, given its crucial diagnostic value [17]. There is an obvious limitation to this approach, however, in that it requires that the ruptured bridging veins have a thrombus, which may take hours. Nevertheless, their detection on imaging is of high diagnostic value, as it confirms the traumatic nature of the subdural bleeding.

The demonstration of ruptured bridging veins can be and should be attempted at autopsy as well – something that is possible in most cases. The calvarium should be opened very carefully, removing only the bone and leaving the dura in place. This is easy in small infants with open sutures, which may be separated due to increased intracranial pressure. It is trickier when the cranial sutures are closed, due to the firm connections

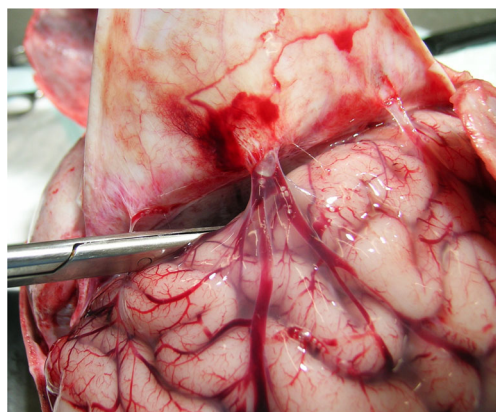


Fig. 3 Normal findings in a male, 5 months old: the photograph demonstrates a few wide bridging veins

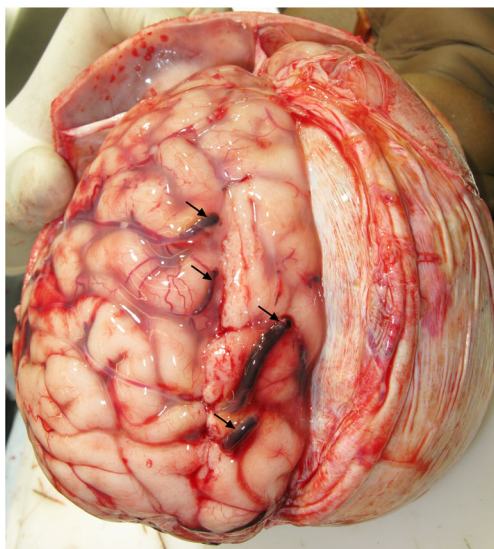


Fig. 4 A girl, 5 ½ months old who died after a 12-day stay in the intensive care unit: thrombosis of acutely disrupted bridging veins (arrows)

between the calvarium and the dura mater [8]. Once the dura matter is exposed, cutting it at its base on both sides and gently lifting it up is easy. This exposes the vertex, making it possible to look for the presence or absence of the bridging veins on either side (Fig. 5). This in situ view is particularly important in cases where severe brain swelling may interfere with subsequent neuropathological examination.

Finally, ruptured bridging veins can also be demonstrated histologically, if missed previously. Indeed, sections of bridging veins can be seen on samples taken from the frontoparietal vertex. If bridging veins are ruptured, there will be an inflammatory reaction (which

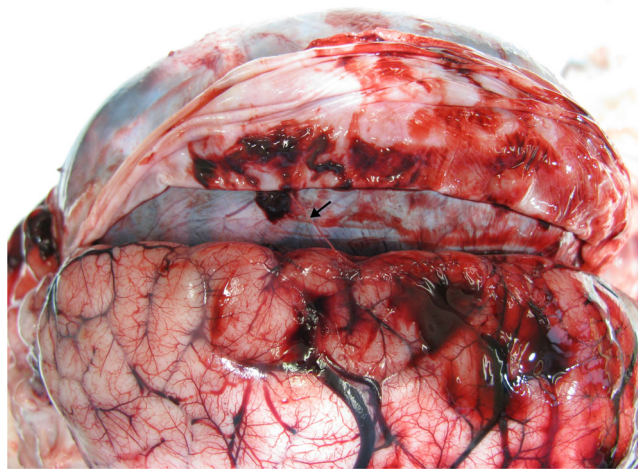


Fig. 5 A girl, 7 ½ months old infant who died after 4 days in the intensive care unit: subdural bleeding and disrupted or torn bridging veins (arrow)

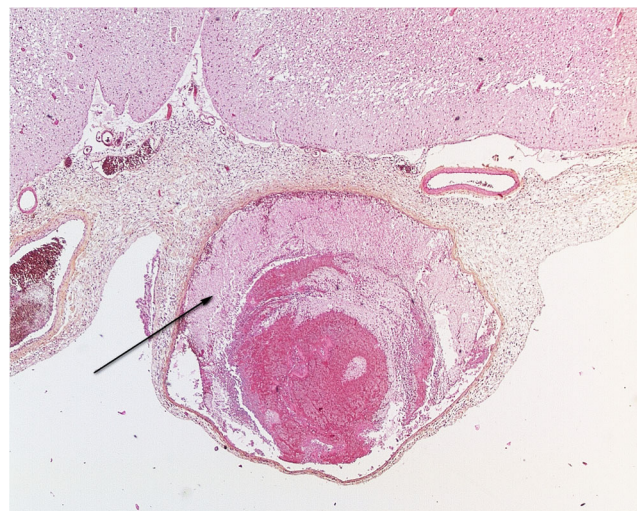


Fig. 6 A male, 12 months old infant who died after 1 month in the intensive care unit: partial thrombosis of a bridging vein (arrow) (HESX5)

helps with dating) around the venous section that increases with increasing proximity to the ruptured end. There will also be siderophages (Perl's stain) around the venous section, becoming especially numerous close to the end of the ruptured vein. Siderophages indicate bleeding – in this case, of a ruptured bridging vein. There may also be partial or total thrombosis (Fig. 6), depending on the distance between the ruptured area and the sample and the time since the rupture. The dating of the thrombosis also helps in dating the overall head trauma. The thrombosis will evolve from clot to hyalinization, and then be recanalized by neovessels (capillaries) (Fig. 7). What happens after that is not

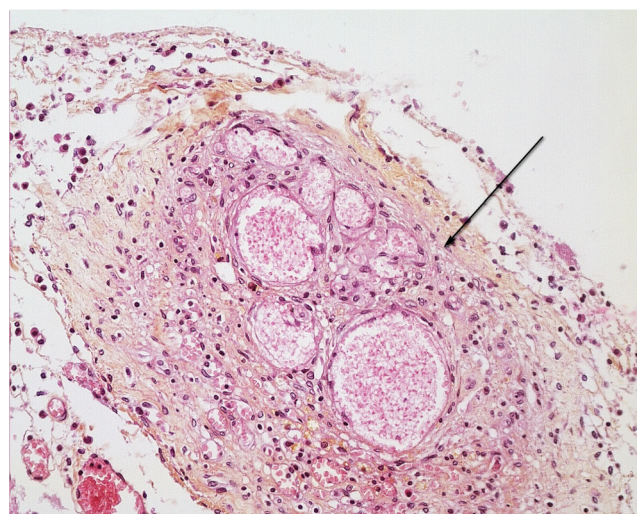


Fig. 7 Same infant as in Fig. 6: complete thrombosis of a bridging vein (arrow), composed of collagenous fibers and partially permeated by neovessels (HESX10)

known: Could new bridging veins grow from the torn vessels?

Conclusion

Examination of the bridging veins should be a mandatory part of the postmortem examination of children with subdural bleeding. Documenting a rupture is essential, as it proves the traumatic nature of the subdural bleeding; likewise, intact bridging veins rule out trauma as a cause. Bilateral bridging vein rupture confirms violent shaking. Unilateral bridging vein rupture is suggestive of unilateral trauma.

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Conflicts of interest None

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