




REVIEW ARTICLE

Thrombosis is not a marker of bridging vein rupture in infants with alleged abusive head trauma

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Abstract

Aim: Thrombosis of bridging veins has been suggested to be a marker of bridging vein rupture, and thus AHT, in infants with subdural haematoma.

Methods: This is a non-systematic review based on Pubmed search, secondary reference tracking and authors' own article collections.

Results: Radiological studies asserting that imaging signs of cortical vein thrombosis were indicative of traumatic bridging vein rupture were unreliable as they lacked pathological verification of either thrombosis or rupture, and paid little regard to medical conditions other than trauma. Autopsy attempts at confirmation of ruptured bridging veins as the origin of SDH were fraught with difficulty. Moreover, microscopic anatomy demonstrated alternative non-traumatic sources of a clot in or around bridging veins. Objective pathological observations did not support the hypothesis that a radiological finding of bridging vein thrombosis was the result of traumatic rupture by AHT. No biomechanical models have produced reliable and reproducible data to demonstrate that shaking alone can be a cause of bridging vein rupture.

Conclusion: There is no conclusive evidence supporting the hypothesis that diagnostic imaging showing thrombosed bridging veins in infants correlates with bridging vein rupture. Hence, there is no literature support for the use of thrombosis as a marker for AHT.

KEYWORDS

abusive head trauma, bridging veins, cerebral venous thrombosis, child abuse, subdural haematoma

1 | INTRODUCTION

Diagnosis of bridging vein thrombosis in infants has become more common in recent years. Controversy has arisen as to whether the

diagnosis of thrombosis can be used as a marker for traumatic bridging vein rupture. Specifically, the radiological diagnosis of thrombosis has been suggested as a marker of abusive head trauma (AHT), the so-called lollipop or tadpole signs on magnetic resonance imaging

Abbreviations: AHT, abusive head trauma; BEH, benign external hydrocephalus; CT, computed tomography; ICP, intracranial pressure; ICU, intensive care unit; MRI, magnetic resonance imaging; SDH, subdural haematoma; SSS, superior sagittal sinus; SWI, susceptibility-weighted imaging.

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(MRI) or computed tomography (CT).¹⁻³ Bridging veins are part of the superficial cerebral venous system, draining blood from the cerebral cortex into the large intradural venous sinuses. The cortical veins traverse the subarachnoid and dural compartments and act as the 'bridge' between the intracranial venous circulation and the systemic circulation of the dura.⁴ When haemorrhage into the subdural compartment is found, damage to the bridging veins is often assumed to be the cause, and therefore, inspection of the veins on imaging has played an increasingly important role in the diagnosis of suspected AHT.

In this review paper, we will briefly study the embryology, anatomy and clinical significance of bridging veins, then critically appraise the existing literature on pathology, radiology and biomechanics regarding thrombosis as a marker of ruptured bridging veins.

2 | METHODS

This was a non-systematic review of literature regarding the various aspects of bridging veins, especially concerning AHT. The review was based on non-structured search in PubMed, secondary reference tracking and the authors' own article collections. No publication date limit was chosen, but search ended on 20 November 2020. Articles in English, German and French were considered. Important search terms were as follows: abusive head trauma; shaken baby syndrome; cortical vein thrombosis; bridging veins; and/or subdural hematoma, among others.

3 | RESULTS

3.1 | Bridging veins

3.1.1 | Embryology

The early connective tissue which later forms the meninges contains a vascular meshwork that evolves into a more distinct vasculature as the brain and skull grow. Initially, the plexus of embryonic vessels divides into deep and superficial layers; the superficial layer becomes the dural vessels while the deeper layers invest the brain to become the leptomeningeal vessels. This primitive network of vessels separates into a distinct venous drainage pattern through the gradual process of venous cleavage. During this process, the number of brain-to-dural venous connections is reduced: many of the veins connecting these early layers are resorbed while a few grow in length and width to become the bridging veins which are more or less fully developed by the end of the first trimester.⁵

While the bridging veins are formed early, the venous structures of the dura undergo modifications throughout gestation and early life. These adjustments in the intradural network are necessary to accommodate the rapid cerebral growth during this period. The configuration of the intradural blood vessels and dural venous sinuses continues to evolve throughout the first year of life, and the major

Key Notes

- This is a non-systematic review of the literature regarding the hypothesis that thrombosis of bridging veins is a marker of abusive head trauma.
- The hypothesis lacks pathological verification, has not been verified biomechanically and does not consider other aetiologies of bridging vein thrombosis.
- There is no evidence for the claim that radiologically detected thrombosis is a marker for bridging vein rupture, and hence, abusive head trauma.

dural sinuses do not attain their adult configuration until well after birth.^{6,7}

3.1.2 | Anatomy

As reviewed by Mortazavi et al.⁸ the bridging veins are typically found in three anatomical regions: cerebellar, temporal and anterior frontal cortical bridging veins. From a surgical point of view, the bridging veins pose a risk for venous infarction if disrupted or damaged. As personally experienced by the senior author [KW] during craniotomy, any manipulation of the bridging veins easily causes oozing of blood from the dura, at the entry points of the veins.^{9,10} As the bridging veins appear unharmed, this bleeding likely comes from the dural capillary bed.

In a post-mortem radiological study, Ehrlich et al.¹¹ found an average of 17 bridging veins (range 9-31) on the brain, reportedly either few of wide diameter or many smaller ones. Cases were all ages ranging from two months to 96 years, with a mean age of around 50 years old. However, a thorough autopsy study of infants found a mean of 54.1 bridging veins per case.¹² Why the reported numbers vary so much remains unknown, but method of investigation (dissection), age and cohort sizes could play a role.¹² In infants, the mean bridging vein diameter in a series was 0.93 mm (range 0.05-3.07 mm).¹²

The wall of the bridging vein consists of collagen bundles arranged circumferentially, elastin fibres and smooth muscle cells.^{13,14} The bridging veins enter the superior sagittal sinus (SSS) in various ways; some, typically found posteriorly, enter at retrograde angles,¹⁵ meaning the blood flows in an anterior direction before entering the SSS. Han et al.¹⁶ found that most bridging veins (97%) entered the SSS in this direction, so one would expect that forces in the postero-anterior direction would be particularly likely to cause stretching tension on these bridging veins. Vignes et al.¹⁷ found that the lumen of the bridging veins narrowed at the junction with SSS, with abundant smooth muscle cells in the vein wall, resembling a sphincter. Physiological narrowing of this sphincter when intracranial pressure (ICP) is increased has been demonstrated in human and animal studies.^{18,19}

The dural portions of the bridging veins are thought to be particularly fragile compared with the subarachnoid portion, giving rise to the belief that a bridging vein would rupture preferentially into the dural compartment rather than into the subarachnoid space.² Though Yamashima and Friede describe very variable wall thickness in the subdural part of the bridging veins (10–600 μm , vs. 50–200 μm in the subarachnoid part), their data are drawn from frontal bridging veins from only four adult patients aged 53–85 years. Moreover, the authors did not directly address the precise dural anatomy; there is no true subdural space in which to measure the wall thickness of bridging veins; rather the subdural compartment is a dissection phenomenon created after disruption of the 8-micron thick tissue layer (dural border cell layer) between the fibrous dura and the arachnoid barrier membrane.

3.2 | Ruptured bridging veins

Ruptured bridging veins are often assumed to be the cause of SDH in infants.^{14,20} In AHT, the presumed tear is believed to be caused by blunt trauma to the head, shaking or a combination of the two.^{20,21} Indeed, presumed rupture of bridging veins has become an important criterion of AHT.^{2,22,23} Rupture of bridging veins can be investigated from different perspectives by neuroimaging, by autopsy and by biomechanical studies:

3.2.1 | Neuroimaging

A case report of two infants with suspected AHT-related SDH reported the use of susceptibility-weighted imaging (SWI) in MRI.²⁴ Signal loss was found on SWI, thought to represent clot formation on bridging veins. No signs of venous infarction were found. Whether these findings were verified, by surgery or autopsy, was not reported.

Choudhary et al.²⁵ used MR venography to study 45 children with assumed AHT based on a retrospective chart review. In 31 (69%) of the children, they found a mass effect on venous sinuses and cortical veins from the nearby hematoma or swollen brain. They also coined the term 'lollipop sign', to describe an imaging finding which they thought was due to a disrupted vein with an associated blood clot. The lollipop sign was found in 20 (44%) of the children in their study. Based on the pre-existing assumption of abuse, the authors concluded that the finding of susceptibility artefact associated with the veins on MRI in the setting of a subdural fluid collection could be viewed as 'evidence of direct trauma to the veins'. They found it unlikely that the venous susceptibility could have been a thrombosis unrelated to trauma. Known causes of cerebral venous thrombosis in children are many, including infections, perinatal complications, haematological disorders and dehydration.^{26,27} Trauma is reported as a rare etiological factor.²⁸

Hahnemann et al.¹ investigated 29 cases of SDH or subdural hygroma in infants with assumed AHT, using CT and MRI. In 11 cases



FIGURE 1 Left-sided subdural haematoma (arrow) in a 7-month-old child as seen on computed tomography. Surgery confirmed rupture of an ipsilateral frontal bridging vein

(40%), they found radiological signs of bridging vein thromboses. In eight of these patients, neuroimaging showed a structure thought to represent a thrombus partly outside of and partly inside a torn bridging vein. It had an oval to round body and a bent tail; hence, the 'tadpole sign' was described.

A more recent MRI study reported a remarkable mismatch between primary MRI diagnoses of bridging vein thrombosis, the tadpole sign, on the axial images compared with coronal high-resolution SWI.²⁹ The authors concluded that the tadpole sign on axial images did not reliably predict thrombosed veins. Instead, they proposed that the signal alteration indicated a traumatic deformation of the vessel, basing their conclusion on 'vessel wall irregularities' detected on the coronal SWI. The limitations of their study included small sample size, lack of pathologic correlation, possible artefacts induced by volume averaging effects and the fact that altered SWI signal cannot differentiate between slow flow and thrombosis.

Adamsbaum and Rambaud reported several cases of allegedly confessed AHT with subdural haematomas and thrombosed bridging veins visible on both CT and MRI.² The authors state that thrombosed bridging veins as seen on neuroimaging are evidence of ruptured bridging veins, which in turn must be caused by a head trauma, in itself suggestive of AHT. The images in that article showed hypodense fluid collections within the subdural compartment. Whether or not the confessions of abuse were consistent with the presence of chronic SDH or correlated with the ages of the SDH on scan was not reported. None of the images in their paper show large volume acute SDH, hyperdense on CT, and the authors did not attempt to explain why no significant acute bleeding was present despite the assumption that recent trauma had caused an acute rupture of multiple macroscopic bridging veins. Our Figure 1 shows what an SDH following acute bridging vein rupture can look like, as confirmed by surgery.

Similar doubts arise regarding an article from Ronning et al.³ They reported 99 infants with SDH, most of them with assumed AHT, fewer with accidental head trauma. The authors found that most children with AHT had parasagittal vertex clots on CT, thought to represent thrombosis, whereas very few of the children with accidental trauma had this CT sign. The images presented in the article, however, do not show SDHs, but rather subdural fluid/hygroma. Furthermore, no pathological investigation or explanation is presented, and evidence on which the diagnosis of abuse rests is not given.

A survey on diffusion-weighted MRI found four patients with venous infarction in relation to assumed ruptured bridging veins in 33 children with alleged AHT.³⁰ The authors did not consider the possibility that the thrombosis and venous infarction may have been unrelated to trauma.

Orman et al.³¹ published MRI findings of an infant (Figure 7 in their article) with typical neuroimaging findings compatible with benign external hydrocephalus (BEH). These figures purportedly show 'hypointense bridging vein thromboses' without discussion of the chronicity of the findings or whether a predisposing condition increased the risk of damage to the vein resulting in thrombosis without significant trauma.

To summarise, several neuroimaging studies report signs of apparent thrombosis which are presumed to reflect traumatic damage to the bridging veins. Three problems, however, emerge from these studies: first, as the findings are based solely on radiological investigations, the physiological/pathological correlates remain obscure. Even if imaging does show thrombosis, bridging vein rupture is not proven, and the studies do not discuss how a ruptured vein would result in a large subdural fluid collection (rather than a large collection of acute blood). Second, the fundamental assumption in AHT cases, that the presence of SDH in children reliably indicates that they were shaken or beaten, is controversial. As reviewed by Lynøe et al.³² the scientific evidence behind the shaken baby syndrome/AHT theory is very limited. Third, statements such as thrombosis is 'evidence of direct trauma' create an impression of certainty implying high-quality evidence behind these findings. None of the studies describe how they have excluded other conditions or diseases, which are more common than trauma as aetiology of bridging vein thrombosis (Figure 2).

3.2.2 | Pathology

Several surveys have investigated deaths of infants with SDH. Identification of the source of subdural bleeding at autopsy is commonly recognised as technically difficult, as the bridging veins are easily damaged during the procedure of opening the skull and dura.^{12,33}

Cheshire et al.¹² reported 48 autopsies of small children (<2 years of age) where the bridging veins were studied. Of these children, three were classified as AHT cases where the bridging veins seemed engorged with congested blood, and when they were pressed from

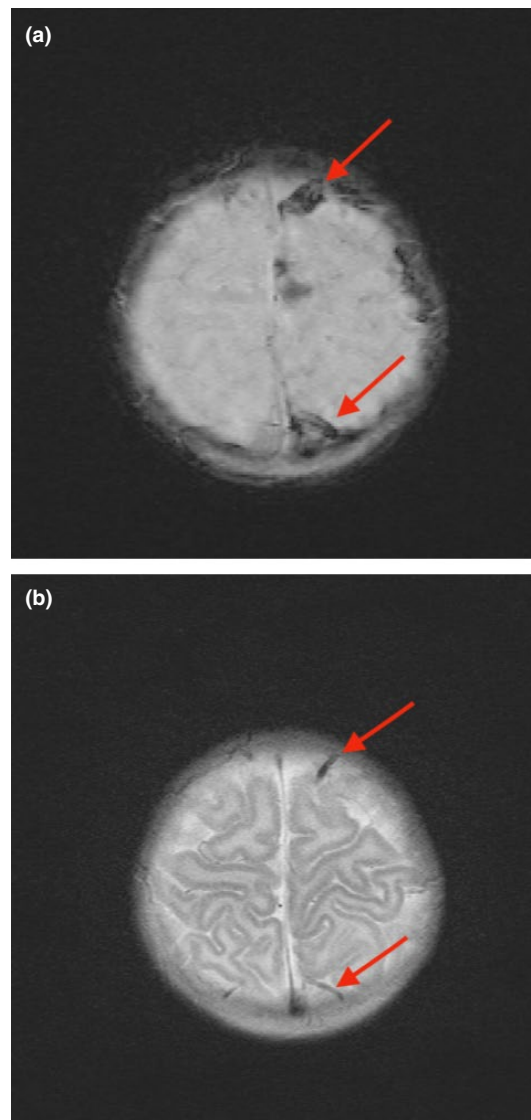


FIGURE 2 (A and B)Magnetic resonance imaging (MRI) of a 26-day-old infant with small volume subdural haematoma and fluid. Seizures developed in the hospital and MRI showed clotted cortical veins (arrows) and evolving non-haemorrhagic infarction. Abuse was initially suggested, but after full evaluation, the charges were dismissed and the child returned to the parents

the outside, they did not blanch. The significance of this observation is not discussed. These veins were not examined microscopically, hence, thrombosis could not be confirmed, nor were the dural sinuses examined to explain the cause of congestion. The authors also found fewer bridging veins in autopsies of children with assumed AHT than in children with no known head trauma. Whether this was due to elastic recoil of small calibre broken veins or veins being obscured from view by the presence of SDH could not be determined.

When comparing microscopic appearances of dura from 50 infants without head trauma with three infants with suspected AHT, Geddes et al.³⁴ found intradural haemorrhage in 72% of the non-trauma cases. They hypothesised, based on the findings of both intradural and subdural haemorrhage in the suspected AHT cases, that

this could be caused by a cascade reaction of hypoxia, plus brain oedema, increased intracranial and central venous pressure, finally leading to bleeding from intradural and bridging veins because of immaturity and hypoxia-related vascular fragility. Although intriguing, this theory primarily shows that several experts in the field have realised that the origin of bleeding into the subdural compartment is uncertain. Indeed, the publication of this hypothesis ignited a rather intense debate, demonstrating the profound disagreement.³⁵⁻³⁷ The striking finding of intradural haemorrhage in 72% of infants without head trauma is important to keep in mind.³⁴ Subsequent studies have confirmed that intradural bleeding is a common finding in very young infants who undergo autopsy and is associated with hypoxic-ischaemic insult.³⁸

When a cerebral vein is thrombosed from any cause, it is distended by a clot and the vessel upstream/proximal of the clot becomes dilated, tortuous and varicose. The cellular reactive changes in the vein wall include proliferation of endothelial lining cells which grow into the clot within the lumen and begin to form new vessels as part of the process of recanalisation. Also, in the early stages, the vein wall becomes leaky, and it is possible to identify red blood cells passing between the cells of the vein wall (diapedesis) leading to haemorrhage into the surrounding tissues, which in the case of cortical veins leads to subarachnoid bleeding (Figure 3). Diapedesis from thrombosed dural veins can lead to intradural bleeding. This small volume haemorrhage may explain the radiological observation of tadpoles and lollipop and does not depend on traumatic tearing of the vein wall, but is the result of venous congestion.

In her review of bridging veins in AHT, Rambaud described histological investigation of ruptured bridging veins.³⁹ She found surrounding inflammation, siderophages indicating bleeding, partial or total thrombosis, and neovascularisation. According to her, dating of the trauma should be possible by examining the thromboses, although she did not explain how this timing could be done. Again, a statement such as 'bilateral bridging vein rupture confirms violent shaking' is unsupported by evidence. No clear aetiology for trauma is presented in the cases described in the article, and she did not consider natural causes of venous thrombosis.

Radiological autopsy

Some authors have reported results of post-mortem radiological investigation of bridging veins.^{11,23,40} Maxeiner²³ used a method where he injected contrast into the SSS in an attempt to produce retrograde filling of the cerebral bridging veins. If contrast appeared on X-ray outside bridging veins, an assumption of premortem traumatic tearing of the veins was made. In his study of infant bridging vein rupture, he found 'typically no significant subdural bleeding despite multiple bridging vein ruptures in the majority of these cases'.⁴¹

Stein et al.⁴⁰ described a technique using direct injection into the SSS through the posterior fontanel in infants who died non-traumatic deaths. Using this method, the authors successfully injected contrast into the sagittal sinus in 8 of 11 infants and determined that it may provide useful information regarding potential bridging vein ruptures. Neither Maxeiner nor Stein et al controlled for injection pressures, addressed post-mortem autolysis as a

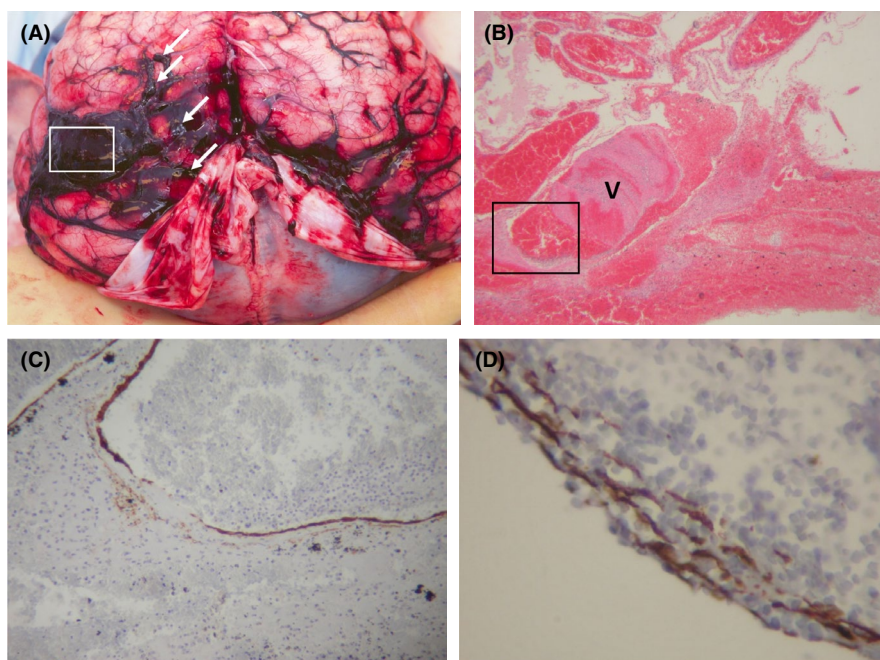


FIGURE 3 (A) Macroscopic view of a large mass of varicose/dilated veins with surrounding subarachnoid blood and local bridging vein thrombosis (white arrows). A section from the area marked with a white box is shown in 3B). (B) Thrombosed vein (V) in the subarachnoid space with surrounding subarachnoid haemorrhage (haematoxylin and eosin stain). (C) Same vein as in B). A defect in the vein wall is seen in the lower left (smooth muscle actin stain, counterstained with haematoxylin and eosin). (D) Higher magnification of vein wall stained with smooth muscle actin (muscle cells are brown, red blood cells are blue). Red blood cells are seen passing between the muscle cells of the vein wall (diapedesis) (smooth muscle actin stain, counterstained with haematoxylin and eosin)

potential contributor to bridging vein disruption after injection or accounted for the presence of the intradural plexus, which would fill with retrograde injection of the sinus. Filling of the plexus would give the impression of contrast outside the sinus and bridging veins which could consequently be mistaken for rupture of the veins.

Neither method has been widely adopted.

3.2.3 | Biomechanics

Through the years, several researchers have tried to investigate the physical properties of bridging veins and their role in SDH formation. Animal studies, finite element studies and cadaveric studies have all been published.

Ommaya et al found that bridging veins ruptured in rhesus monkeys subjected to angular acceleration (shaking), but later stated that these forces were too strong to be achieved by manual shake.^{42,43} In a classical study, Duhaime et al.⁴⁴ used infant-like dolls with attached accelerometers and subjected them to various shaking and impact episodes. Based on tolerance limits from primates, they found that shaking alone would not create enough force to cause SDH, suggesting that blunt impact to the head was necessary to generate such damage. Their technique was later refined in a study with similar results,⁴⁵ but was critically reviewed and questioned by others.⁴⁶ As stated by Jones et al in 2015, 'no study has to date demonstrated that shaking alone, without an associated impact, exceeds the injury thresholds associated with SDH'.⁴⁷ A recent physics calculation found that a low-level fall yielded greater angular acceleration to a 6-month-old infant than shaking.⁴⁸ Similarly, one article, using a doll model, found that the head movements during normal play in an infant were similar to previously published studies on violent shaking in a model, and that shaking movements could not reach angular accelerations regarded as necessary for SDH.⁴⁹

Roth et al.⁵⁰ created a finite element head model and found that the bridging veins underwent equal maximum strain for shaking and impact, concluding that both inflictions could cause SDH. However, this study has several limitations: it is not a validated model, the parameters are not from infants, bridging veins are modelled as linear springs which lack viscoelastic properties and the modelled impact can be compared with a short fall of half a metre.

In several studies, human cadaver heads have been subjected to occipital impacts creating various rotational strains.⁵¹⁻⁵³ Based on post-test findings, the authors suggested threshold levels of rotational accelerations and velocities causing bridging vein rupture. Similar studies have also been performed in adult rhesus monkeys,⁵⁴ although the reliability of extrapolating such findings to human infants has been challenged.⁵⁵ The main finding in these studies is that quite substantial acceleration forces are required to damage the bridging veins, forces that are reliably created by an impact, but not by shaking alone.

Recent reviews have concluded that thresholds, based on experiments or models, used to assess shaking trauma are of low quality and questionable use.^{56,57}

Zhu et al.⁵⁸ conducted a combined autopsy and modelling study of 137 bridging veins from six adults. Based on bridging vein diameters and angles relative to the SSS, they calculated that venous thrombosis would occur more easily in wider bridging veins >1.2 mm, and when angles at the entry points were small (<65°).

One model found that the junction between the bridging veins and the SSS is stiffer than the bridging veins themselves, making this part particularly fragile and prone to rupture.⁵⁹ This is questionable, considering the previously mentioned finding of a reinforced, sphincter-like junction.¹⁷ Monea et al.⁶⁰ did mechanical testing (stress-strain) of this junction and found quite variable results, both between individuals and within the same individual.

Although biomechanical models may seem useful, the multitude of models and the variable results make it difficult to form any definitive conclusions on the role and behaviour of bridging veins in SDH formation in general, and in trauma cases specifically. Results from cadaver studies are problematic due to the use of non-vital tissue already undergoing autolysis and simplistic experiment setups. Finite element studies can provide results from various traumas, but depend on very accurate values of for instance anatomy, geometry and tissue characteristics.⁶¹ Even the intriguing use of cadavers to confirm the biomechanical predictions from a finite element model⁶² carries the risk of creating a model based on properties not found in real-life vital tissues. Biomechanical properties of different tissues are largely unknown, and the reported values differ. However, biomechanical studies are still useful as they allow us to compare different situations and traumas, for instance shaking versus a short fall.

3.2.4 | Neurosurgical considerations

Some surgical approaches to the brain involve sacrificing bridging veins. A study of 63 paediatric patients showed no signs of venous infarction on MRI following interhemispheric transcallosal surgical procedures that involved ligation of bridging veins.⁶³ A recent study, however, found changes in ICP, motor and sensory function, and histological changes, for instance haemorrhage, in mice following venous infarction induced by cutting bridging veins.⁶⁴ The changes peaked at around 12 h after surgery and seemed to resolve within 48 h.

In another animal study, an artificial increase in ICP led to dilatation and decreased blood flow velocity in cerebral bridging veins, suggesting a compensatory increase in resistance to outflow.¹⁸

4 | DISCUSSION

The tearing of bridging veins is considered an important criterion for the diagnosis of AHT. Furthermore, thrombosis of bridging veins has

been suggested as a surrogate for traumatic rupture and a certain diagnostic sign of shaking.^{1,2,22}

When reviewing the existing literature in this field, there are some overarching issues that need to be addressed.

First, many studies, both biomechanical and from autopsy, are based on adult patients, not young infants. Considering the rapidly developing cerebral venous system in fetuses and neonates, the direct comparison between adult and infant bridging veins should be done with caution.

Second, studies on infants with SDH and alleged AHT are most often based on assumed or suspected head trauma, not witnessed or proven. The true mechanism behind each case is therefore obscure, and reliance on the presumption of abuse gives rise to circularity and an inherent unreliability in the subsequent conclusions drawn from that data.

Third, there is a growing understanding of the birth process as an important contributor to intracranial haemorrhages in newborns. A difficult birth is a known risk factor for developing SDH,^{65,66} but even in normal deliveries and with asymptomatic term neonates, an MRI study found that almost half of the infants had SDH.⁶⁷ Bridging vein rupture, as seen on neuroimaging, is very rarely identified in these babies and dural bleeding from the vast intradural venous plexus is a more likely source. The degree to which birth-related SDH may affect later findings and symptoms is still unknown.

Indeed, an important part of diagnostic evaluation of any patient is to consider all possible differential diagnoses. This becomes even more important in cases of suspected AHT, where allegation of abuse and legal proceedings has significant consequences for the infants and their families. Many conditions are recognised as causes of SDH in infants, such as infections, malformations, and metabolic and coagulation disorders.⁶⁸

Benign external hydrocephalus (BEH) is also a known risk factor for developing SDH.⁶⁹⁻⁷⁴ It has been assumed that the widened subarachnoid space in BEH would stretch the bridging veins, making them more vulnerable to rupture, even with minimal trauma. Surprisingly, a finite element study showed a dampening effect of the enlarged subarachnoid space, claiming that BEH would not be a risk factor for developing SDH.⁷⁵ The article, however, has one major limitation, namely that the bridging veins were not assumed to be stretched in the case of widened subarachnoid spaces. Disagreement still exists as to whether BEH is a risk factor or not.^{76,77} A recent review investigated the similarities between SDH and BEH, and presented a unifying theory of pathophysiology behind these subdural collections.⁷⁸

Few neuroimaging studies have reported findings of ruptured bridging veins, except the ones reviewed above. Findings such as the tadpole or lollipop signs lack pathologic verification of bridging vein injuries. Nevertheless, the authors of the article describing the tadpole sign claim that bridging vein thrombosis is an excellent indicator of AHT in SDH cases.¹ Similarly, it is stated that parasagittal vertex clots may be a novel predictor of AHT.³ A neuroimaging sign of bridging vein thrombosis may simply reflect either slowed flow

or venous thrombosis from natural disease unrelated to trauma and cannot be considered pathognomonic for venous injury or trauma.

A limitation of our review is that no systematic literature search was undertaken. However, there are only limited numbers of articles relevant to our specific question: is bridging vein thrombosis a marker of AHT? We believe that our study of the field has allowed us to make a thorough, albeit not systematic, review.

5 | CONCLUSION

As for neuroimaging, whether a venous injury can be identified, if it really exists, and whether it is caused by a trauma, remains uncertain. As for pathology, neither autopsy nor other examinations can prove that bridging vein ruptures or thromboses are caused by AHT only. As for biomechanics, the multitude of models is not able to show how and which traumas may lead to bridging vein rupture.

The subject of SDH and AHT in infants is a sensitive matter with strong feelings and opinions. This makes it even more important to maintain a high degree of accuracy and verifiability in the field. This review points to an alarming lack of evidence behind the investigation and interpretation of thrombosed bridge veins.

CONFLICTS OF INTEREST

Julie A. Mack has served as an unpaid expert witness in cases of alleged abuse. Cyrille Rossant is the current president of a French non-profit organisation (Adikia) providing moral support to parents facing false allegations of child abuse (unpaid activity). Waney Squier has acted as an expert witness, sometimes paid, in cases of suspected child abuse both for the prosecution and for the defence. She was reported to the General Medical Council (the governing body for the doctors in the UK) on the basis of her evidence in shaken baby cases and got her licence suspended, but restored on appeal. Knut Wester has served as a mostly unpaid expert witness for the court and the defence in a few cases of suspected abusive head injury in Norwegian courts. Sverre Morten Zahl declares no conflicts of interest.

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