Acute subdural hematoma from bridging vein rupture: a potential mechanism for growth

Clinical article

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Most acute subdural hematomas (ASDHs) develop after rupture of a bridging vein or veins. The anatomy of the bridging vein predisposes to its tearing within the border cell layer of the dura mater. Thus, the subdural hematoma actually forms within the dura. The hematoma grows by continued bleeding into the border cell layer. However, the venous pressure would not be expected to cause a large hematoma. Therefore, some type of mechanism must account for the hematoma’s expansion.

Cerebral venous pressure (CVP) has been demonstrated in animal models to be slightly higher than intracranial pressure (ICP), and CVP tracks the ICP as pressure variations occur. The elevation of CVP as the ICP increases is thought to result from an increase in outflow resistance of the terminal portion of the bridging veins. This probably results from a Starling resistor model or, less likely, from a muscular sphincter.

A hypothesis is derived to explain the mechanism of ASDH enlargement. Tearing of one or more bridging veins causes these vessels to bleed into the dural border cell layer. Subsequent ICP elevation from the ASDH, cerebral swelling, or other cause results in elevation of the CVP by increased outflow resistance in the intact bridging veins. The increased ICP causes further bleeding into the hematoma cavity via the torn bridging veins. Thus, the ASDH enlarges via a positive feedback mechanism.

Enlargement of an ASDH would cease as blood within the hematoma cavity coagulates. This would stop the dissection of the dural border cell layer, and pressure within the hematoma cavity would equalize with that in the torn bridging vein or veins.

Key Words • • • acute subdural hematoma • bridging vein rupture • vascular disorders

Abbreviations used in this paper: ASDH = acute SDH; CVP = cerebral venous pressure; ICP = intracranial pressure; SDH = subdural hematoma; SSS = superior sagittal sinus.
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Course deep to the lateral lacunae, if both are present, and empty into the SSS separately. They tend to have a short intradural course prior to opening into the SSS. The Starling resistor, first described in 1912 by Knolw and Starling, has been promoted as a control of venous outflow resistance preventing the collapse of superficial cortical veins. However, Vignes et al. described positive staining for smooth muscle actin in the venous walls and adjacent SSS and hypothesized that smooth muscle fibers act as a muscular sphincter to help maintain venous pressure proximally. Thus, 2 possible physiological mechanisms have been proposed for flow resistance from the bridging vein to the SSS, a passive Starling resistor and a muscular sphincter. Magnetic resonance imaging has demonstrated narrowing of the outflow segment of bridging veins in humans with increased ICP of various causes supporting this area as one cause of venous outflow resistance.

Pang et al. demonstrated that cerebral bridging veins in pigs have an “outflow cuff segment” at the junction of the vein and the SSS. In this cuff area the collagen fibers become circumferential, rather than longitudinal, with a smaller diameter and a thickening of the wall, and such changes were interpreted as controlling venous outflow and intracranial pressure (ICP). Animal studies have demonstrated decreased flow through the “lacunar portion” of the cerebral veins, and a decrease in the diameter of the “cerebrovascular bed” proximal to the sinus that might occur in the bridging vein. Therefore, the anatomy of the terminal portion of the bridging vein is compatible with this region acting as a flow resistor.

Perfusion pressure in an organ is determined by the difference in arterial and venous pressures. Because cerebral venous pressure (CVP) is not readily ascertainable, ICP is used to determine cerebral perfusion pressure. Normal ICP is less than 15 mm Hg, with pressure varying from 5 to 10 mm Hg. Cerebral venous pressure and ICP track together, with the CVP being slightly higher.

This tracking has been demonstrated with supratentorial morbidity imaging has demonstrated narrowing of the outflow segment of bridging veins in humans with increased ICP of various causes supporting this area as one cause of venous outflow resistance.

Subdural Hematoma Anatomy

Cushing reported that it was possible to “dislodge the villous tufts from their pockets between the cross-hatched mesh of dural fibers of the parasinoidal zones, with a minimum of bleeding or with no bleeding whatsoever, if the manipulations are carefully conducted, except in those cases when they are associated with a vessel bridging from the cerebral cortex to sinus or dura.” In a later publication, Cushing’s drawings depicted a well-defined subdural space. Thus, Cushing was familiar with bleeding from bridging veins, and his published works depicted an actual subdural space.

However, the concept of a subdural space has evolved since the time of Cushing. Leary and Edwards described peeling off the inner layer of dura and found the dural lining largely made up of fibroblasts. These authors thought that the dura was lined by fibroblasts, while the arachnoid was covered by a continuous layer of flattened cells of ectodermal origin. Leary and Edwards wrote, “The formation of the subdural space embryologically is perhaps due to the separation of the surface covered by these cells from a layer of mesenchyme which becomes the inner layer of dura.” They evaluated the dura in multiple species. They described the dural inner surface as layers of flattened cells similar to flattened fibroblasts and referred to these as “dural border cells.” In the small mammals studied, they found no tight junctions between the dural border cells; therefore, the virtual subdural space was in a fascial plane. In macaque monkeys, however, there were enlargements of intercellular spaces filled with a “fuzzy material” within the dural border cell layer. Frederickson reported that separation of the dura and arachnoid was easily performed in a guinea pig model. He found that before separation the dura and arachnoid were in contact and after separation the arachnoid’s outer surface was irregular with cytoplasmic debris and disrupted cell membranes. The subdural space was reported to occur in a specific cell layer of flattened fibroblasts. Frederickson found that the number of cells varied with location and were consistent with the dural border cells described by Nabeshima et al. Haines described perioistial and meningial dural layers.

Modified fibroblasts with no organized junctional complexes were seen beneath the meningeal layer. This layer in the normal state was continuous externally with the dura and internally with the arachnoid with no subdural space. Later, Haines et al. reported that dural border cells were present in humans as well as in animals and that within this layer the cells have irregular patterns, creating extracellular spaces of various sizes and shapes. These extracellular spaces were filled with an amorphous, nonfilamentous material consistent with the “fuzzy material” described by Nabeshima et al.

The dura mater and arachnoid mater, in addition to being attached via the dural border cell layer, are connected to the inner skull surface and the pia mater, respectively. Since the dural border cells can be separated, Haines et al. proposed that a shearing force may tear a vein, allowing blood to enter into the dural border cell layer to form a subdural hematoma (SDH). Yamashima and Friede reported that the subdural bridging vein thickness ranged from 10 to 600 μm and the subarachnoid portion from 50 to 200 μm. The collagen fibers were denser in the subarachnoid portion, and these fibers tended to be circumferential, which would resist distention but less so traction. In addition, the bridging veins have a short intradural course. Thus, the bridging veins may anatomically be predisposed to shear in the dural border cell layer causing an SDH (Fig. 1).
ASDH and the Underlying Brain

An ASDH commonly results from tearing of one or more bridging veins. This lesion also occurs from hemorrhagic contusions or intracerebral hematomas and from injury to a cortical artery or vein. Nontraumatic ASDHs can originate from a ruptured arteriovenous malformation or intracranial aneurysm. Intracranial pressure may be higher on the side of a hematoma than in the opposite hemisphere, which has been demonstrated in animals and humans. Pathophysiological abnormalities of the brain occurred with ASDHs in a rat model, with the cerebral cortex having ischemic damage. This model documented early massive glucose hypermetabolism in both hippocampi and around the ischemic area, which normalized by 4 hours after blood injection into the “subdural space.” Excitatory amino acids are released in the cortex beneath the hematoma and in the hippocampus. There is occlusion of the microvasculature, which begins in the cortex beneath the hematoma and in the hippocampus. This is found with nonaneurysmal perimesencephalic subarachnoid hemorrhage that is associated with a thin layer of blood on CT scanning and attributed to a venous origin. However, if a bridging vein is torn within the dural border cell layer as proposed by Haines et al., there would be some disruption of this layer, and the evolving hematoma would be separated from the subarachnoid space. The dural border cell layer is predisposed to separation between its cells because of extracellular spaces filled with amorphous, nonfilamentous material and a lack of cellular junctional complexes. As blood flows into the dural border cell layer, the resultant hematoma would be expected to cause an increase in the ICP, which, in turn, would result in a parallel rise in the intracranial venous

A Potential Mechanism of ASDH Growth

Bridging veins may rupture within the dural border cell layer or the subarachnoid space. One would expect minimal blood loss if the rupture occurred into the subarachnoid space or the vein retracted from the dural border cell layer into the subarachnoid space, since the pressures inside and outside the vein would equalize. This is found with nonaneurysmal perimesencephalic subarachnoid hemorrhage that is associated with a thin layer of blood on CT scanning and attributed to a venous origin. However, if a bridging vein is torn within the dural border cell layer as proposed by Haines et al., there would be some disruption of this layer, and the evolving hematoma would be separated from the subarachnoid space. The dural border cell layer is predisposed to separation between its cells because of extracellular spaces filled with amorphous, nonfilamentous material and a lack of cellular junctional complexes. As blood flows into the dural border cell layer, the resultant hematoma would be expected to cause an increase in the ICP, which, in turn, would result in a parallel rise in the intracranial venous

Fig. 1. Anatomical depiction of the formation of an ASDH. Left: Coronal section taken at the level of the SSS demonstrating tearing (solid arrow) of a bridging vein (BV) after forceful impact to the skull. Right: Detail of bridging vein tear (curved arrow) and accumulation of blood within the dural border cell layer (straight arrows). ABC = arachnoid barrier cell layer; AT = arachnoid trabeculae; CC = cerebral cortex; D = dura; DBC = dural border cell layer; EV = emissary vein; F = falx; P = pia; VL = venous lacunae. Copyright Remi Nader. Published with permission.
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pressure.\textsuperscript{21,40,41} This rise in venous pressure could result either from a passive, mechanical valve like a Starling resistor or from a myogenic sphincter.\textsuperscript{24,29,33,40,58,61} The increased venous pressure would include increased pressure in the bridging vein or veins that have ruptured into the dural border cell layer with continued bleeding into the evolving ASDH. Thus, the ASDH would enlarge from the increasing venous pressure via a positive feedback mechanism (Figs. 2 and 3). Other factors that elevate ICP, such as cerebral ischemia and swelling or formation of an intracerebral hematoma, would be expected to result in increased ICP and bridging vein pressure with more bleeding into the ASDH cavity. The same mechanism of escalating CVP as ICP increases has been proposed as a factor in cerebral edema formation.\textsuperscript{41}

This is similar to a hypothesis proposed by Osterholm, who theorized that ICP elevation above 20 mm Hg would result in SSS collapse, with subsequent elevation of venous pressure and more bleeding from the torn bridging vein.\textsuperscript{45} However, Osterholm’s hypothesis of SSS collapse is not likely in the 20–mm Hg pressure range because of the sinus edges tethered by the dura attached to the skull on each side and the falx inferiorly. A rise in ICP has been demonstrated experimentally to result in variable SSS pressure changes.\textsuperscript{59} In some animals, SSS pressure may decline with a concomitant rise in CVP because of a decrease in cerebral blood flow, and pressure in the SSS does not begin to increase until the ICP reaches 30 mm Hg.\textsuperscript{57} Eventually, rising pressure can produce compression of both the SSS and the bridging vein outflow segment.\textsuperscript{59} Thus, Osterholm’s proposal of SSS compression is probably not a factor, at least early in the formation of an ASDH. Initially, the most likely cause of ASDH enlargement is positive feedback of venous pressure from the terminal vein acting as a flow resistor and torn bridging veins bleeding into the dural border cell layer.

Cessation of ASDH Enlargement

There is evidence of active bleeding into an SDH on contrast-administered CT and MRI scans when obtained very early after trauma.\textsuperscript{16,56} Such bleeding has been found at craniotomy from bridging veins, cortical arteries, or from contused brain tissue.\textsuperscript{16,56} An autopsy series of patients who died of “pure” SDHs was reported by Maxeiner and Wolff.\textsuperscript{24} Interestingly, the volumes of “pure” ASDHs from venous or arterial origin did not significantly differ.\textsuperscript{24} In the absence of a mechanism that would cause a progressive increase in venous pressure, one might expect hematomas from arterial bleeding to be larger than those of venous origin. However, a positive feedback mechanism as proposed above would cause progressive enlargement of an ASDH from torn bridging veins.

As the subdural cavity enlarges, the pressure needed to expand the hematoma will increase because the blood is trapped in a confined space between the hard skull and the increasingly less pliable compressed brain tissue. In addition, concurrent coagulation should be occurring in the presence of fresh blood. This would, in turn, make the available cavity around the bleeding vein smaller, thus requiring more venous pressure for continued enlargement. Such a small cavity of liquid blood surrounded by clot is suggested by early CT scanning.\textsuperscript{56} Eventually, bleeding would cease as pressure inside and outside the bleeding vessel equalizes, or a solid clot forms at the vessel opening. Outcomes are worse in patients with AS DHs and coagulopathy.\textsuperscript{1} However, Bershard et al. did not demonstrate that traumatic ASDHs were larger in patients with coagulopathy; other factors, such as delay in surgery, could be responsible for the worse outcomes. Nontraumatic ASDHs have been shown to have greater thicknesses in the presence of coagulopathy.\textsuperscript{4} This might suggest that in patients without coagulopathy, coagulation of blood in the dural border cell layer is occurring and restricting the size of the hematoma. There has been no exclusive study published that discusses the coagulation mechanism implicated in modulating the expansion of ASDHs. It has been shown that the fibrinolytic system is defective in cases of chronic SDH.\textsuperscript{20} Chronic SDH fluid analysis implicates coagulation via the extrinsic clotting pathway.\textsuperscript{54} Since a chronic SDH starts initially as an acute hematoma, the same clotting mechanism may be occurring in acute hematomas as well. Therefore, an extrinsic pathway is likely involved in clot formation restricting the size of the ASDH.

Conclusions

Acute subdural hematomas frequently arise from tearing of bridging veins. These veins are torn within the dural border cell layer and result in blood flowing into a potential space within the dura mater. Bleeding may continue via a positive feedback mechanism that causes the CVP to increase as the ICP is elevated. This subsequently results in continued dissection of the border cell layer as the hematoma enlarges. This continues until blood in the periphery of the ASDH begins to coagulate, stopping the border cell layer dissection, and pressure within the ASDH cavity rises to equal that in the torn bridging vein or veins.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Miller. Acquisition of data: Miller. Drafting the article: both authors. Critically revising the article: Miller. Reviewed submitted version of manuscript: both authors. Approved the final version of the manuscript on behalf of both authors: Miller.
**Fig. 3.** Upper: Depiction of tearing of a bridging vein (Vein 2) in its transit through the dura. This tends to occur preferentially within the dural border cell layer because the cells of this layer are easily separated. If there is enough force during separation of the dural border cells, the traversing vein may be torn with blood filling the potential space within the dural border cell layer. Lower: With increasing ICP, there is constriction of veins within the subarachnoid space (see Vein 1) and elevation of cerebral venous pressure to maintain venous patency. This increase in venous pressure results in more blood flowing through the tear in Vein 2 with growth of the ASDH. Copyright Remi Nader. Published with permission.

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