Idiopathic Subarachnoid Hemorrhage and Venous Drainage: Are They Related?

OBJECTIVE: In the past, several possible explanations for idiopathic subarachnoid hemorrhage (ISAH) have been proposed; however, neuroimaging studies have never provided conclusive data about the structural cause of the bleeding. The aim of this study is to determine whether there are anatomic differences in the deep cerebral venous drainage in patients with ISAH compared with those with aneurysmal subarachnoid hemorrhage (ASAH) and those without intracranial hemorrhage.

METHODS: We reviewed the venous phase of carotid digital angiograms of 100 consecutive patients who had a final diagnosis of ISAH. We also analyzed the angiograms of a control group of 112 patients with ASAH and the angiograms of a nonhemorrhagic group of 25 patients having incidental aneurysms. The anatomic variants of the basal vein of Rosenthal (BVR) on both sides were classified into the following types: Type A (normal continuous), in which the BVR is continuous with the deep middle cerebral veins and drains mainly into the vein of Galen; Type B (normal discontinuous), in which there is discontinuous venous drainage, anterior to the uncal vein and posterior to the vein of Galen; and Type C (primitive), which drains mainly to veins other than the vein of Galen. We calculated the proportions to analyze the differences in the type of venous drainage between patients with ISAH, patients with ASAH, and patients without hemorrhage. χ² statistics were used to search for differences.

RESULTS: Types A and C venous drainage were present in 23.8 and 32.3%, respectively, of patients with ISAH compared with 58.7 and 15.4%, respectively, in the ASAH group and 57.5 and 5%, respectively, in the nonhemorrhagic group (P < 0.001). A primitive variant was present in at least 1 hemisphere in 38 patients with ISAH (41.8% of the cases) compared with 24 patients with ASAH (21.4%) and 2 patients (8%) in the nonhemorrhagic group (P < 0.001).

CONCLUSION: In patients with ISAH, deep cerebral venous drainage more commonly drains directly into dural sinuses instead of via the vein of Galen compared with patients with ASAH and patients without intracranial hemorrhage. The way in which this venous configuration might influence bleeding remains unknown.

KEY WORDS: Idiopathic, Spontaneous, Subarachnoid hemorrhage, Venous drainage

In nearly 15% of patients with nontraumatic subarachnoid hemorrhage (SAH), no obvious source of hemorrhage can be demonstrated by high-quality, 4-vessel cerebral angiography (1, 4, 5, 8, 10, 17, 20, 21, 25). There is evidence that patients with so-called idiopathic SAH (ISAH) have a benign clinical course and a much better final outcome than patients with aneurysmal SAH (ASAH). In the former, the rates of rebleeding and ischemia are comparatively lower and are approximately 6 and 13%, respectively (1, 10, 21, 22).
In 1985, van Gijn et al. (24) described a subtype of ISAH characterized by an accumulation of subarachnoid blood predominantly around the midbrain and the absence of an aneurysm or other identifiable source of bleeding on angiography. They called it nonaneurysmal perimesencephalic SAH (PMSAH). However, in the ISAH group, there are patients with a normal computed tomographic (CT) scan (diagnosed by lumbar puncture) and patients showing an aneurysmal pattern on the initial CT scan. Irrespective of the CT pattern, patients in these subgroups also have a benign clinical course and a better prognosis than patients with ASAH (Fig. 1) (14, 17, 19, 22, 24).

In the past, several possible explanations for ISAH have been proposed, including temporary thrombosis of an aneurysmal sac, permanent destruction or self-repair of a microaneurysm or microangioma at the time of hemorrhage, changes in blood flow caused by vasospasm that could prevent aneurysm filling on angiography, bleeding from perimesencephalic or deep veins, or hemorrhage from basilar trunk dissections (3, 6, 9, 12, 13, 16). However, neuroimaging studies (digital angiography, magnetic resonance imaging, CT angiography) have never provided conclusive data about the structural cause of the bleeding.

Recently, some authors have studied the relationship between the occurrence of ISAH and the presence of some variants of deep venous drainage in small series of patients, suggesting a possible link between abnormal drainage and bleeding (23, 26). However, other authors have not been able to demonstrate that link (7).

The aim of this work is to determine whether there are anatomic differences in the deep cerebral venous drainage as shown on digital angiography in patients with ISAH compared with those with ASAH and those without hemorrhage.

**PATIENTS AND METHODS**

We retrospectively reviewed the venous phase in lateral projection of carotid digital angiograms of 100 consecutive patients admitted to our department between 1996 and 2004 who had a final diagnosis of ISAH. Nine patients were excluded because adequate bilateral angiogram projections could not be retrieved from history files; thus, there were 91 patients in the study. We also analyzed the angiograms of a control group of 112 patients randomly selected from our ASAH database and the angiograms of a nonhemorrhagic group composed of 25 patients having incidental aneurysms. The anatomic variants of the basal vein of Rosenthal (BVR) on both sides were carefully inspected and classified independently by 2 neurosurgeons (JFA, AL) and 2 neuroradiologists (JC, FB), according to Watanabe et al. (26), into the following types: Type A (normal continuous), in which the BVR is continuous with the deep middle cerebral veins and drains mainly into the vein of Galen; Type B (discontinuous), in which there is discontinuous dual drainage, anterior to the uncal veins and posterior to the vein of Galen; and Type C (primitive), which drains directly into dural sinuses instead of the galenic system (Fig. 2).
we considered 1 missing side to calculate the total number of cases and controls (Table 1), but when considering all patients as a whole (with 2 sides), we considered the missing side as being the same type as that of the known side (Table 2).

κ statistics were used to assess the degree of agreement in the interpretation of the venous findings between observers. We also calculated proportions to analyze differences in the type of venous drainage among patients with ISAH, patients with ASAH, and patients without hemorrhage. χ² statistics were used to search for differences in the presence of anomalous venous patterns among the different study groups.

We defined 3 possible types of deep venous drainage in each patient depending on the configuration on both the left and right sides: normal drainage (AA), discontinuous drainage when Type B was seen on one of the sides (AB or BB), and primitive drainage when there was a Type C on any of the sides (AC, BC, or CC). In cases with only 1 side of venous drainage available, we considered the contralateral one as being of the same type; thus, drainage is categorized as primitive when Type C drainage is observed in one of the sides (AC, BC, or CC). In the 18 cases in which there was disagreement between the neuroradiologists and neurosurgeons about the type of drainage, we reached a final consensus.

**RESULTS**

Interobserver reliability between the neurosurgeons and neuroradiologists was acceptable when considering Type A and B drainage as normal and Type C as a primitive one (κ statistic = 0.70).

The accumulated percentages of the different types of cerebral deep venous drainage in patients with ISAH (cases), ASAH, and nonhemorrhagic controls are presented in Table 1. We could not classify the venous drainage in 18% of the 182 possible types of drainage (2 sides × 91 cases) in the group of patients with ISAH because no appropriate x-rays were found in the files; likewise, the type of venous drainage could not be classified in 11.2% of patients with ASAH and in 20% of patients without hemorrhage. Types A and C venous drainage were present in 23.8 and 32.3% of patients, respectively, with ISAH compared with 58.7 and 15.4%, respectively, in the group of patients with ASAH and 57.5 and 5%, respectively, in the nonhemorrhagic group (P < 0.001). We could not find differences between both control groups (ASAH and nonhemorrhagic) (P = 0.11).

Table 2 shows the distribution of the types of venous drainage in patients with ISAH and ASAH and patients without hemorrhage. A primitive variant was present in at least 1 hemisphere in 38 patients with ISAH (41.8% of the cases) compared with 24 patients with ASAH (21.4%) and 2 patients without bleeding (8%) (P < 0.001). We could not find differences between both control groups (ASAH and nonhemorrhagic) (P = 0.21).

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**Table 1. Percentages of the different types of cerebral deep venous drainage in patients with idiopathic subarachnoid hemorrhage (cases), patients with aneurysmal subarachnoid hemorrhage (controls), and patients without hemorrhage (nonhemorrhagic controls)**

<table>
<thead>
<tr>
<th>Type of venous drainage</th>
<th>No. of cases (% of cases)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total ISAH cases</td>
</tr>
<tr>
<td>A</td>
<td>36 (19.5)</td>
</tr>
<tr>
<td>B</td>
<td>66 (36)</td>
</tr>
<tr>
<td>C</td>
<td>47 (26)</td>
</tr>
</tbody>
</table>

**Missing cases**

|                         | 33 (18)     | —                        | 26 (11.2)   | —                       | 10 (20)                | —                                       |

**Total no. of sides**

|                         | 182 (91 × 2) | 224 (112 × 2) | 50 (25 × 2) |

*ISAH, idiopathic subarachnoid hemorrhage; ASAH, aneurysmal subarachnoid hemorrhage. Two sides for each patient were considered. χ² statistics were used for differences between the 3 groups (P < 0.001); between ASAH controls and nonhemorrhagic controls (P = 0.11); between ASAH controls and ISAH cases (P < 0.001); and between nonhemorrhagic controls and ISAH cases (P < 0.001).*

**Table 2. Type of bilateral cerebral deep venous drainage in patients with idiopathic subarachnoid hemorrhage (cases), patients with aneurysmal subarachnoid hemorrhage (controls), and patients without hemorrhage (nonhemorrhagic controls)**

<table>
<thead>
<tr>
<th>Type of venous drainage, no. of cases (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal (AA)</td>
</tr>
<tr>
<td>ASAH controls</td>
</tr>
<tr>
<td>ISAH cases</td>
</tr>
<tr>
<td>Nonhemorrhagic controls</td>
</tr>
<tr>
<td>Total</td>
</tr>
</tbody>
</table>

*AA, normal drainage; AB, discontinuous drainage when Type B was seen on one of the sides; BB, discontinuous drainage when Type B was seen on one of the sides; AC, primitive drainage when there was a Type C on any of the sides; BC, primitive drainage when there was a Type C on any of the sides; CC, primitive drainage when there was a Type C on any of the sides; ASAH, aneurysmal subarachnoid hemorrhage; ISAH, idiopathic subarachnoid hemorrhage. χ² statistics for differences between the 3 groups (P < 0.001); between ASAH controls and nonhemorrhagic controls (P = 0.21); between ASAH controls and cases (ISAH) (P < 0.001); and between nonhemorrhagic controls and cases (ISAH) (P < 0.001).*
Because of these changes in the developing venous pathways, the mesencephalic veins from the lateral ventricles are diverted from the lateral atrium and join the telencephalic veins to enter the primitive transverse sinus. Similarly, the primitive lateral cortical veins, which drain the telencephalic and diencephalic veins, are deflected into the dural sinuses. As the cerebral hemispheres expand posterolaterally and the tentorium, which contains the transverse sinus, moves caudally, the tentorial sinus becomes more prominent. As the stem of the anterior dural plexus decreases in size, the tentorial sinus becomes more prominent. The deep venous system of the brain is composed of the internal cerebral vein, the great vein of Galen, the BVR, and their respective tributaries. The BVR begins just anterior to the midbrain near the anterior perforated substance, travels laterally to this structure, and terminates posteriorly, usually into the great vein of Galen, although it may drain into the straight sinus or the internal cerebral veins. Padget (18) demonstrated that the BVR is a complex vessel that formed when the embryo was approximately 80 mm long and arises secondarily as a longitudinal anastomotic channel resulting from the union of several regional embryonic veins. Particularly important in the embryological development of the venous system (26). The deep venous system of the brain is composed of the internal cerebral vein, the great vein of Galen, the BVR, and their respective tributaries. The BVR begins just anterior to the midbrain near the anterior perforated substance, travels laterally to this structure, and terminates posteriorly, usually into the great vein of Galen, although it may drain into the straight sinus or the internal cerebral veins. Padget (18) demonstrated that the BVR is a complex vessel that formed when the embryo is approximately 80 mm long and arises secondarily as a longitudinal anastomotic channel resulting from the union of several regional embryonic veins. Particularly important in the development of the cerebral venous system, the primitive telencephalic and diencephalic veins drain into the stem of the anterior dural plexus. They later join the newly formed primitive transverse sinus via the tentorial sinus. As the stem of the anterior dural plexus decreases in size, the transverse sinus becomes more prominent. As the cerebral hemispheres expand posterolaterally and the tentorium, which contains the transverse sinus, moves caudally, the tentorial sinus, which drains the telencephalic and diencephalic veins, becomes elongated and circuitous. Similarly, the primitive mesencephalic vein is stretched as it passes under the temporal and occipital lobes to enter the primitive transverse sinus. Because of these changes in the developing venous pathways,

Table 3 shows the distribution of venous drainage in the 3 subgroups of patients with ISAH as related to the pattern of bleeding seen on the initial CT scan (normal CT, aneurysmal pattern, or perimesencephalic pattern). Bilateral Type A was present in only 7.9% of patients with a perimesencephalic pattern compared with 25% of patients with a normal CT scan ($P < 0.05$).

**DISCUSSION**

Although many attempts have been made to define the origin of bleeding in patients with ISAH, it remains to be determined. Some authors have attributed the hemorrhage to anomalies in ventriculostriate and thalamoperforating arteries (2) and other vascular abnormalities (27). In this respect, no consistent abnormalities were described in the initial report by van Gijn et al. (24) after careful examination of the venous phases of 13 patients with PMSAH. The anterior segment of the BVR drained into the superior petrosal sinus through the lateral mesencephalic vein in 2 patients, and the petrosal vein drained into the vein of Galen in 1 patient, leading the authors to postulate that extravasation of blood might originate from normal vascular structures, such as the BVR or one of its tributaries.

Watanabe et al. (26) suggested that an anomalous configuration in the BVR could be responsible for the bleeding in 6 patients presenting with ISAH. As we did in this study, they classified the variations in the BVR into 3 types and analyzed the venous phases of the angiograms in 6 patients with PMSAH and in 102 controls with ASAH. They identified a primitive type (Type C) of venous drainage in 7 (58%) of the 12 possible drainages in the group of patients with PMSAH compared with only 22% of those presenting with ASAH. In addition, none of the 6 patients with PMSAH presented a bilateral Type A drainage.

More recently, van der Schaaf et al. (23) compared the venous drainage of the midbrain in 55 patients with PMSAH and 42 patients with ASAH by using CT angiography. A primitive variant was present in 1 or both hemispheres in 53% of patients with PMSAH and in 19% of ASAH patients. A bilateral normal continuous drainage (bilateral Type A) was present in only 7.3% of the PMSAH patients. They also reported that in all 16 patients with unilateral primitive drainage having noncontrast CT scans, blood had extravasated on the side of the primitive drainage or symmetrically; in addition, there were no patients with unilateral primitive drainage and extravasated blood mainly distributed on the contralateral side.

In our series, we included all patients with ISAH, i.e., those with PMSAH, those showing an aneurysmal CT pattern of bleeding (all of them with 2 negative angiograms), and those with a normal CT scan who were diagnosed by lumbar puncture. After analyzing the available data, we believe that all of those cases represent different presentations of the same pathological entity, which carries a good outcome in 94 to 100% of the cases. Although some authors have reported a worse outcome in patients with an aneurysmal pattern on CT scans, the complications responsible for the poorer result may be attributed to the presence of more blood on the initial CT scan (14). Therefore, we think that the origin of bleeding may be the same in all of these cases, with the only difference being the total amount of blood filling the subarachnoid space.

The results of our series are very similar to those reported by van der Schaaf et al. (23). In their study, 41.8% of patients with ISAH had at least 1 Type C drainage, which was seen in only 21.4% of the patients in the control group. In keeping with these findings, we observed normal bilateral drainage in only 13% of our ISAH patients.

The categorization of the type of venous drainage into the subgroups of normal continuous (Type A), normal discontinuous (Type B), and primitive (Type C) variants is based on the embryological development of the venous system (26). The deep venous system of the brain is composed of the internal cerebral vein, the great vein of Galen, the BVR, and their respective tributaries. The BVR begins just anterior to the midbrain near the anterior perforated substance, travels laterally to this structure, and terminates posteriorly, usually into the great vein of Galen, although it may drain into the straight sinus or the internal cerebral veins. Padget (18) demonstrated that the BVR is a complex vessel that formed when the embryo is approximately 80 mm long and arises secondarily as a longitudinal anastomotic channel resulting from the union of several regional embryonic veins. Particularly important in the formation of the BVR are the telencephalic, diencephalic, and mesencephalic veins. As described by Padget in her extensive work on the development of the cerebral venous system, the primitive telencephalic and diencephalic veins drain into the stem of the anterior dural plexus. They later join the newly formed primitive transverse sinus via the tentorial sinus. As the stem of the anterior dural plexus decreases in size, the tentorial sinus becomes more prominent. As the cerebral hemispheres expand posterolaterally and the tentorium, which contains the transverse sinus, moves caudally, the tentorial sinus, which drains the telencephalic and diencephalic veins, becomes elongated and circuitous. Similarly, the primitive mesencephalic vein is stretched as it passes under the temporal and occipital lobes to enter the primitive transverse sinus.

![Table 3](https://example.com/table3.png)

**Table 3.** Types of bilateral cerebral deep venous drainage in the subgroups of patients with idiopathic subarachnoid hemorrhage

<table>
<thead>
<tr>
<th>Type of drainage</th>
<th>No. of cases (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal CT scan</td>
<td></td>
</tr>
<tr>
<td>Discontinuous</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
</tr>
<tr>
<td>Normal CT bilateral</td>
<td>(25)</td>
</tr>
<tr>
<td>Discontinuous or primitive</td>
<td>(75)</td>
</tr>
<tr>
<td>Total</td>
<td>(100)</td>
</tr>
<tr>
<td>Aneurysmal pattern</td>
<td>(13.5)</td>
</tr>
<tr>
<td>Discontinuous</td>
<td>(86.5)</td>
</tr>
<tr>
<td>Total</td>
<td>(100)</td>
</tr>
<tr>
<td>Perimesencephalic pattern</td>
<td>(7.9)</td>
</tr>
<tr>
<td>Discontinuous</td>
<td>(92.1)</td>
</tr>
<tr>
<td>Total</td>
<td>(100)</td>
</tr>
</tbody>
</table>

* CT, computed tomographic.
a shorter and more direct course develops by secondary longitudinal anastomoses between the deep telencephalic, ventral diencephalic, and mesencephalic veins and the dorsal diencephalic tributary of the internal cerebral vein or the superior mesencephalic tributary of the great vein of Galen. The BVR is created in this fashion (Fig. 3). Posteromedial and superior drainage of the BVR into the galenic venous system via the anastomotic channels is phylogenetically a more advanced form and is predominant in primates, especially humans. In contrast, posterolateral and inferior drainage of the BVR into the transverse sinus via the tentorial sinus is a more primitive form (11). Therefore, the typical normal pattern of drainage into the vein of Galen is designated as Type A. In Type B, the vein drains both anteriorly (uncal vein) and posteriorly (the galenic system). Because failure of anastomosis is most frequent between the telencephalic and diencephalic segments, Type B could be considered a common variant of Type A. In Type C, the vein drains into veins other than the galenic system (the uncal, anterior, or lateral pontomesencephalic veins or tentorial sinus).

Our study shows that deep cerebral venous drainage more commonly drains directly into the dural sinuses instead of via the vein of Galen in patients with ISAH. The way in which this venous configuration might influence bleeding remains unknown. Some authors have postulated that the direct connection of peri-mesencephalic veins with the dural sinuses may predispose to sudden increases in venous pressure, resulting in engorgement and rupture of the veins (26). Other authors have postulated that a tear in the vein where it crosses the tentorial margin might result from torsion or friction and secondary rupture (23, 24). Finally, other investigators believe that bleeding could be arterial in origin, resulting from a basilar short-segmented dissection or a capillary telangiectasia (16, 27). In summary, all the different hypotheses advanced to explain bleeding have both convincing and weak points.

Our results suggest that in most of the patients with ISAH, the venous configuration around the midbrain could play an important role in the origin of the bleeding. The direct connection of cerebral veins to the dural sinus and the fact that primitive variants lack fusion of the primitive veins during embryological development might predispose these vessels to rupture owing to an increase in venous pressure. In addition, some of the primitive veins circulate across the tentorial margin, exposing them to torsion or friction and making them even more prone to rupture (15).

The very low incidence of rebleeding in this anatomic context is striking and difficult to explain because the disposition of venous drainage is not likely to change after the initial bleeding. Matsumaru et al. (15) speculated that the spontaneous healing of the venous rupture by fibrous tissue reaction would reinforce the wall of the vein, decreasing the risk of rupture. However, this explanation is not very convincing and probably represents the weakest point of their hypothesis.

**Disclosure**

This project was supported by Grant 2007/82 from the Fundacion Mutua Madrileña and Fondo de Investigaciones Sanitarias Grant FIS PI 070152.
REFERENCES


Acknowledgment

We thank Fatima Iarlori, B.S., for help with the editing of the figures for this manuscript.

COMMENTS

A len et al. provide a closer examination of the venous anatomy of patients with ISAH versus aneurysmal subarachnoid hemorrhage (ASAH). ISAH continues to fascinate us because of our failure to define a clear-cut etiology. The authors note the differing hypotheses that have been advanced, ranging from arterial to venous sources for ISAH in the literature. I personally favor venous origin hypotheses owing to the typically limited amount of perimesencephalic blood that is observed.

The authors’ contribution does not provide any specific information regarding mechanisms, beyond the hint that the venous anatomic configuration may be a risk factor for ISAH. They are appropriately cautious in their conclusions about the mechanism of bleeding and point out the flaws and strengths of differing hypotheses that have been advanced to date.

Where can one go from here? Moving from anatomy to better characterization of physiology in the basal venous structures will likely be important. This is likely to come from advances in imaging to allow us not only to better study anatomy but also to quantitate flow in the basal venous structures in ISAH.

Bob S. Carter
Boston, Massachusetts

The authors have attempted to preliminarily examine a possible association of anatomic variation in deep cerebral venous drainage with ISAH. Specifically, they sought to examine differences in the deep cerebral venous drainage in patients with ISAH as compared with those with ASAH and those with unruptured aneurysms and no SAH. To this end, the authors studied the venous phase of 100 consecutive patients with ISAH, 112 patients with ASAH, and 25 patients with incidental aneurysms. For this analysis, they classified anatomic variants of the basal vein of Rosenthal on both sides into Type A (normal
continuous), Type B (normal discontinuous), and Type C (primitive). They found the primitive variant to be present in at least 1 hemisphere in 38 patients (42%) with ISAH, in 24 patients (21%) with ASAH, and in 2 patients (8%) in the nonhemorrhagic group. They conclude that deep venous drainage is more commonly made directly into dural sinuses (primitive pattern) rather than through the vein of Galen in patients with ISAH in comparison to patients with ASAH or those without hemorrhage.

This is an interesting study that provides a preliminary glimpse into the important issue of delineating the mechanism responsible for ISAH, which is a common clinical problem. The authors provide provocative speculation that merits future investigation. However, it is important to note that as the authors correctly emphasize, an association was identified here between a primitive pattern of venous drainage and ISAH; this in no way proves causation, and the majority of patients with ISAH did not, in fact, demonstrate a primitive venous drainage pattern.

Hian K. Yeoh
R. Webster Crowley
Aaron S. Dumont
Charlottesville, Virginia