SA–CME Information

Description

Subarachnoid hemorrhage (SAH) is a medical emergency in which radiologists play an important role in diagnosis and characterization to optimize treatment. Prompt diagnosis is crucial, and knowledge of underlying pathologic processes and potential complications guides the diagnostic workup. This article reviews the imaging features and relevant clinical characteristics of SAH.

Objectives

As a result of this activity, the participant should be able to:

- Describe the criteria for diagnosis of SAH, including the appropriate role of computed tomography (CT) and magnetic resonance (MR) imaging.
- Review the various etiologies of SAH, including ruptured aneurysmal and nonaneurysmal SAH, and SAH resulting from trauma.
- Explain the complications that can occur at the time of SAH ictus, as well as in the ensuing days and weeks.

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Radiologists and related medical physicians

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Imaging of intracranial hemorrhage: Subarachnoid hemorrhage and its sequelae

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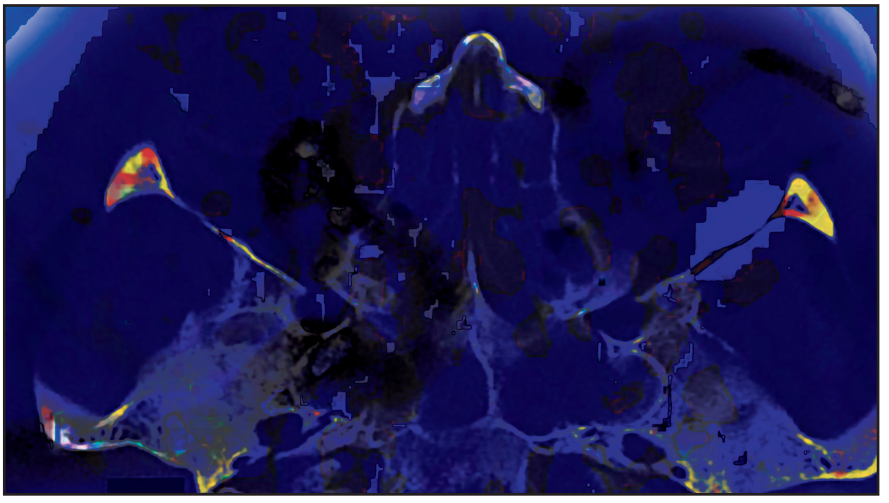
Subarachnoid hemorrhage (SAH) is a medical emergency in which radiologists play an important role in diagnosis and characterization to optimize treatment. Incidence varies geographically, with reported rates ranging from 2 to 32 per 100,000 individuals annually.[1,2] SAH can inflict considerable morbidity and mortality, and the burden imposed on society is significant given the relatively young age of many affected individuals compared to other neurological pathologies.[3,4] Prompt diagnosis is crucial, and knowledge of underlying pathologic processes and potential complications guides the diagnostic workup. This article will review imaging features and relevant clinical characteristics.



Diagnosis and initial imaging

Patients with SAH present with severe headaches. While most patients with a headache will not have SAH, computed tomography (CT) is typically used to exclude SAH in the setting of severe headache.[1-3] Headaches from SAH are classically described as the most severe of one's life, but acute onset within seconds is a more specific feature.[3] CT is widely available, has short acquisition times, and is very accurate for diagnosis of SAH.[1] CT correctly demonstrates hyperdense material within the subarachnoid space in the setting of acute SAH 95% of the time (Figure 1).[2] As cerebrospinal fluid (CSF) is resorbed by arachnoid granulations, blood contents are also resorbed, causing dilution of the SAH and resultant diminution of the density seen on CT and reduced sensitivity in the subacute period.[2] Given the potential for false negative CTs, lumbar puncture must be utilized to exclude occult hemorrhage after a negative CT.[1,2]

Computed tomography at diagnosis can also provide useful information to guide treatment and determine prognosis. The Fisher scale is widely used to grade SAH and is based on CT findings.[5]

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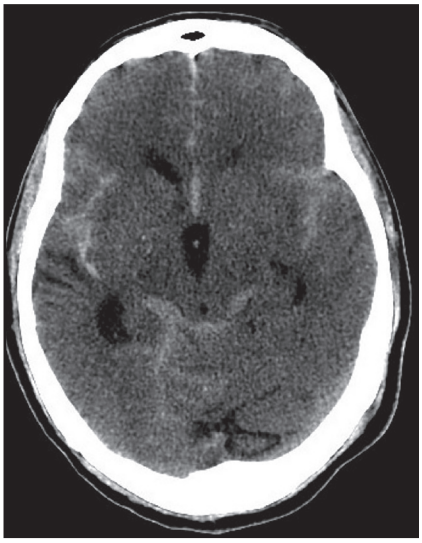

FIGURE 1. *Noncontrast CT demonstrating diffuse SAH in the interhemispheric fissure, bilateral MCA cisterns, and bilateral ambient cisterns. No IVH was noted.*

Table 1. CT-based grading of SAH

Grade	Fisher	Modified Fisher
0	n/a	no SAH or IVH
1	SAH <1 mm thick	thin SAH without IVH
2	SAH >1 mm thick	thick SAH without IVH
4	diffuse SAH or any IVH	thick SAH with bilateral IVH

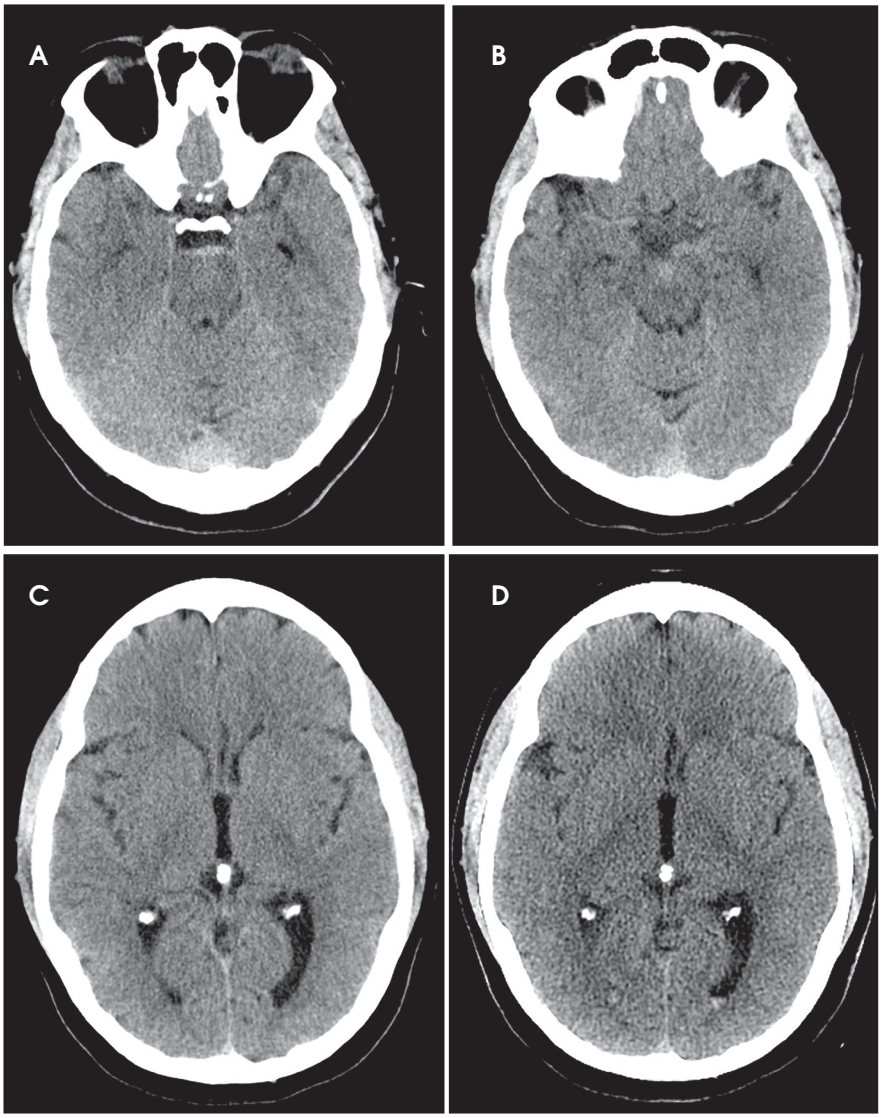

FIGURE 2. *(A) Small amounts of SAH noted in the prepontine and (B) interpeduncular cisterns. (C) Mild layering IVH is noted in the lateral ventricles (D). No SAH is seen elsewhere.*

Modifications have occurred as SAH thickness and presence of IVH were found to be additive in risk for ischemia.[6] The Fisher and modified Fisher scales are summarized in Table 1. Hemorrhage may be present in other intracranial compartments. Intraventricular hemorrhage (IVH) may be found with varying severity; as time passes blood is more likely to be found within the ventricular system due to its contiguity with the subarachnoid space and the mobile nature of CSF (Figure 2).[2] However, larger hemorrhage volumes can extend into the ventricles at the time of the initial insult, and outcomes are likely to be poor when IVH is massive (Figure 3).[2,7] Such a description is important prognostically because CSF diversion in the setting of massive IVH has demonstrated no benefit, although some centers have reported improved outcomes when used in conjunction with fibrinolytic therapy.[2,8,9] Epidural hemorrhage (EDH) and intraparenchymal hemorrhage (IPH) can be seen, with severities varying according to the underlying pathology (Figure 4).[2] Subdural hemorrhage (SDH) rarely occurs but can be severe when present (Figure 5).[2,10,11] CT can also identify concomitant soft tissue or osseous injuries of the head and neck (Figure 5).[1] One in 7 patients with SAH will develop intraocular hemorrhage, known as Terson's syndrome, which can be seen on CT, MR or fundoscopy and is a sign of poor prognosis (Figure 6).[12-14]

CT is utilized to assess for SAH largely because of its accuracy, efficiency and relative cost effectiveness. However, magnetic resonance (MR) imaging offers comparable accuracy, and familiarity with SAH appearances on these studies is crucial.[2,15] MR characteristics of blood all relate to the paramagnetic properties of hemoglobin and the products of its degradation.[16-18] As intracranial hemoglobin is degraded, it undergoes a well-described sequence from oxygenated to deoxygenated states and then conversion to methemoglobin, which can be present both

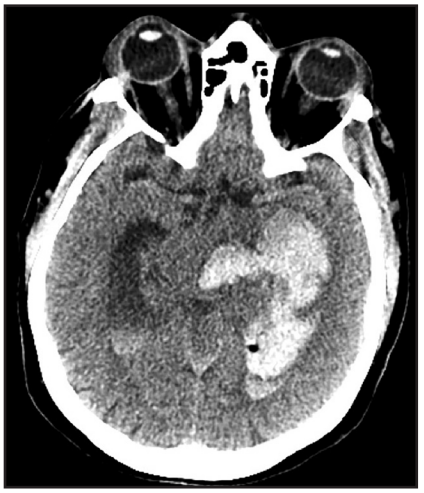

FIGURE 3. *Massive IVH expanding the left lateral ventricle. IVH is also seen in the right lateral ventricle, and there is IPH in the left pons.*

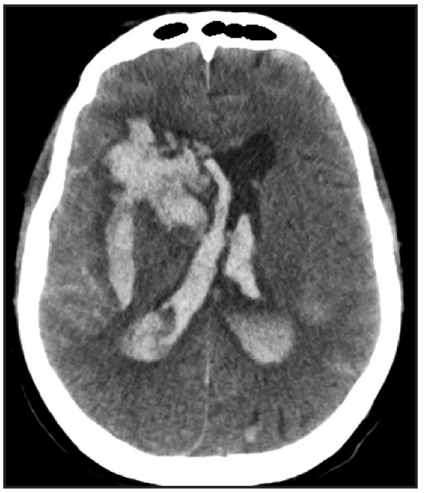

FIGURE 4. *Noncontrast CT demonstrating SAH layering along sulci, IVH in the lateral ventricles, and right frontal IPH.*

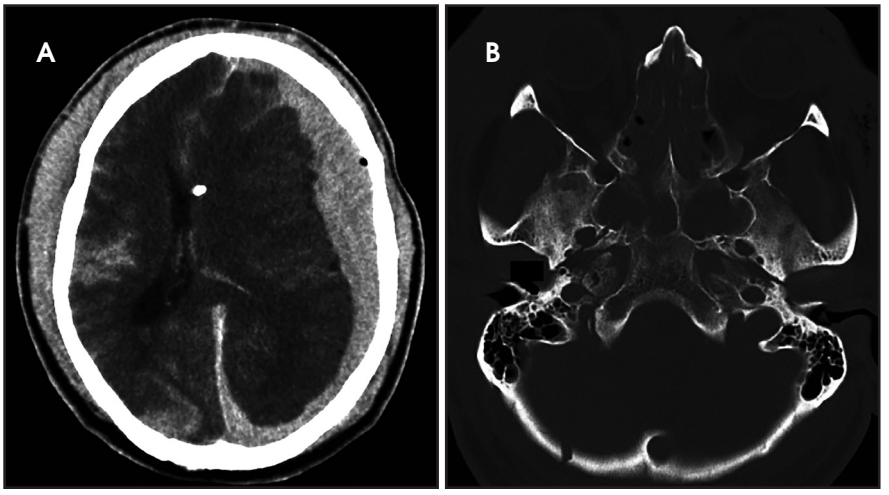

FIGURE 5. *(A) Midline shift in a patient with SDH and diffuse SAH. (B) Pneumocephalus and a ventricular shunt are also seen. Bone algorithm and window through the skull base demonstrate bilateral temporal bone and right occipital calvarial fractures.*

intracellularly and extracellularly as cells are lysed.[16-18] Imaging characteristics of chronic blood products are due to ferritin and hemosiderin.[16-19] Most understanding of the appearance and timeframe for the degradation of intracranial blood is based on intraparenchymal hemorrhage.[17] Due to higher levels of oxygen and free water in CSF, as well as protein with which hemorrhage may interact, SAH has unique MR characteristics.[16,17] Early SAH is best visualized on fluid attenuation inversion recovery (FLAIR) imaging, on which it appears hyperintense; T1-weighted imaging may also demonstrate hyperintensity at this stage but is frequently less well seen (Figure 7).[2,15-17] Over the ensuing days, SAH remains visible on FLAIR, but gradient echo (GRE) imaging becomes the best sequence for visualizing SAH (Figure 8).[20,21] Degradation of hemoglobin progresses over a longer time period compared to other intracranial compartments, and resorption of products by arachnoid granulations may occur before methemoglobin or hemosiderin accumulate.[17] However, any of the above-described degradation products can be seen, with typical signal characteristics as seen elsewhere in the brain and summarized in Table 2.[16,17] Temporal descriptors such as hyperacute, acute, and subacute are accepted based on understanding IPH degradation. Given differences in temporal changes, such descriptors should be avoided in

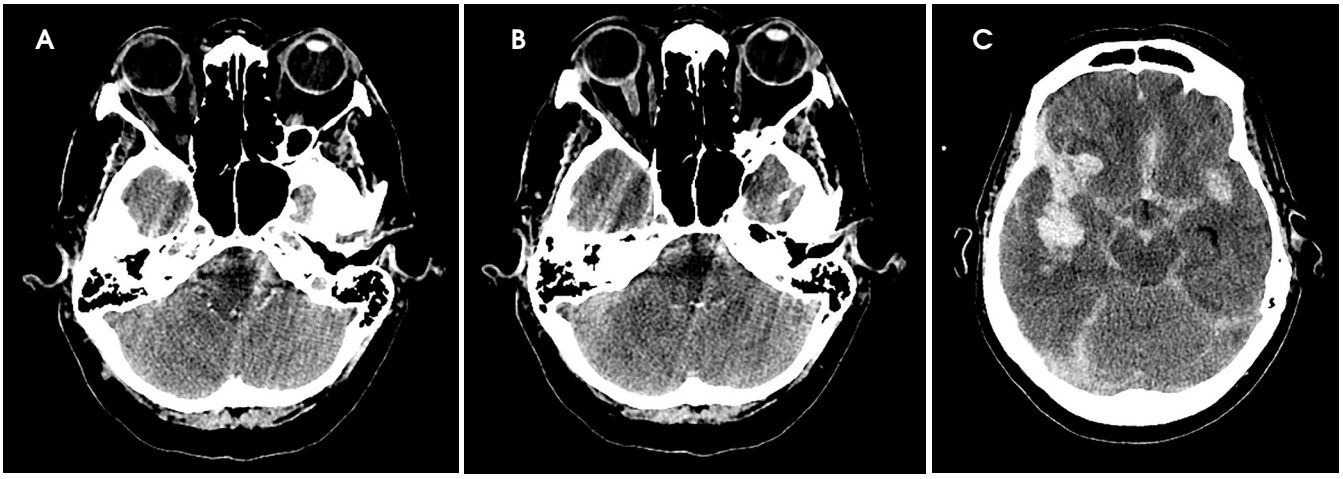

FIGURE 6. *(A) Layering density in the right globe in a patient (B) There is SAH consistent with Terson's syndrome. SAH is derived from the aneurysmal rupture depicted in (C).*

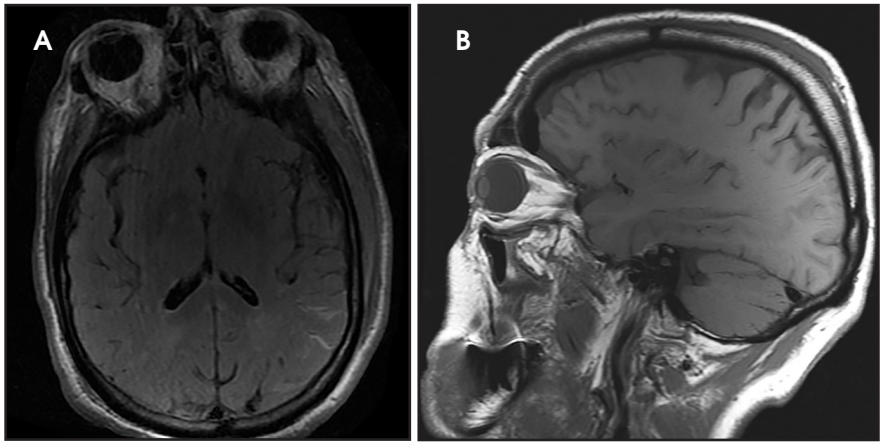

FIGURE 7. *Axial (A) and sagittal (B) FLAIR images with hypointensity in the subarachnoid space consistent with SAH.*

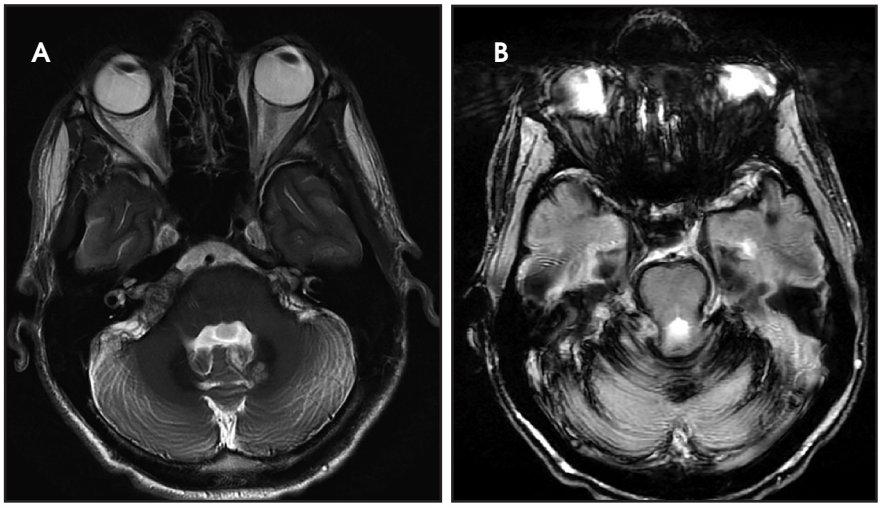

FIGURE 8. *(A) Thick hypointensity lining the surface of the pons and cerebellum on T2-weighted and (B) GRE images in a patient with superficial siderosis.*

describing SAH to prevent confusion. With repeated SAH, hemosiderin may accumulate on the surface of the brain and cranial nerves, a condition known as superficial siderosis, which appears hypointense on T2 weighted images and GRE (Figure 8).[17,22,23]

Etiologies of subarachnoid hemorrhage

Numerous processes can cause subarachnoid hemorrhage, but a ruptured aneurysm is the origin in 85% of cases.[2] Given the high likelihood of an aneurysm, further investigation is warranted upon the diagnosis of SAH, particularly given the substantial morbidity and mortality associated with them. Ten to thirteen percent of patients with aneurysmal SAH die before reaching the hospital, and overall mortality approaches 50%.[3,24-32] Diagnostic cerebral angiography (DSA) has long been considered the gold standard for detection of cerebral aneurysms. (Figure 9) While techniques have been optimized to maximize safety of cerebral DSA, risks still remain.[33-37] Additionally, these procedures can require considerable resources and coordination that may prohibit emergent performance in some centers. For these reasons utilization of noninvasive CT or MR angiography has increased, with sensitivities and specificities reported up to 97% and 100%, respectively (Figure 10).[38-43] CT angiography is typically preferred to MR angiography due to the time constraints and clinical stability requirements of the latter.[25] Diagnostic accuracy declines for aneurysms measuring less than 3mm, so DSA remains the gold standard.[5,32,39-46] In addition to diagnosis of an aneurysm, high quality imaging is necessary to plan appropriate treatment, with best characterization occurring with both two- and three-dimensional DSA.[2,3,25,47-49] Characteristics important to report include size, ratio of maximal depth to neck width, morphology, direction of aneurysm projection, any arteries arising from the aneurysm, and presence of an apical bleb (Figures 9, 10).[50,51]

Aneurysms predominantly occur at arterial branching points, with the majority occurring in the anterior circulation. They most commonly arise from the anterior communicating (AComm, 30%), posterior communicating (PComm, 25%), middle cerebral (MCA, 20%), and distal internal carotid arteries (ICA, 7%). Seven percent of aneurysms occur at the distal basilar artery, and 3% arise from the posterior inferior cerebellar artery (PICA).[25] Prevalence of cerebral aneurysms in the general population is 2%.[52] In those individuals with a diagnosed aneurysm, an additional aneurysm is present in up to 35%.[53-59] In the setting of SAH and multiple aneurysms, it is important to identify the aneurysm that has ruptured. Certain characteristics are suggestive of rupture, including length-to-neck ratio greater than 1.6, increased volume to surface area, aneurysm angulation, and presence of an apical bleb.[60-63] Hemorrhage itself may aid identification of culprit aneurysms, although such clues are only reliable in the acute setting.[64] Lateralized SAH typically indicates MCA, ICA, or PComm aneurysms, with degree of lateralization of SAH corresponding to degree of lateralization of aneurysms (Figure 11).[64] Midline SAH occurs with basilar or AComm aneurysms (Figure 9).[64] Posterior fossa SAH is associated with PComm and posterior circulation aneurysms, whereas anterior circulation aneurysms typically cause supratentorial SAH.[64] When parenchymal hemorrhage occurs, the aneurysm typically

Table 2. Stages of hemoglobin degradation with corresponding degradation products, their locations with respect to cells, and respective MR signal characteristics

Location	Hemoglobin state	T1 signal*	T2 signal*
intracellular	oxyhemoglobin	isointense	mildly hyperintense
intracellular	deoxyhemoglobin	midly hypointense	hypointense
intracellular	methemoglobin	hyperintense	hypointense
extracellular	methemoglobin	hyperintense	hyperintense
extracellular	hemosiderin	mildly hypointense	hypointense

*Relative to brain parenchyma

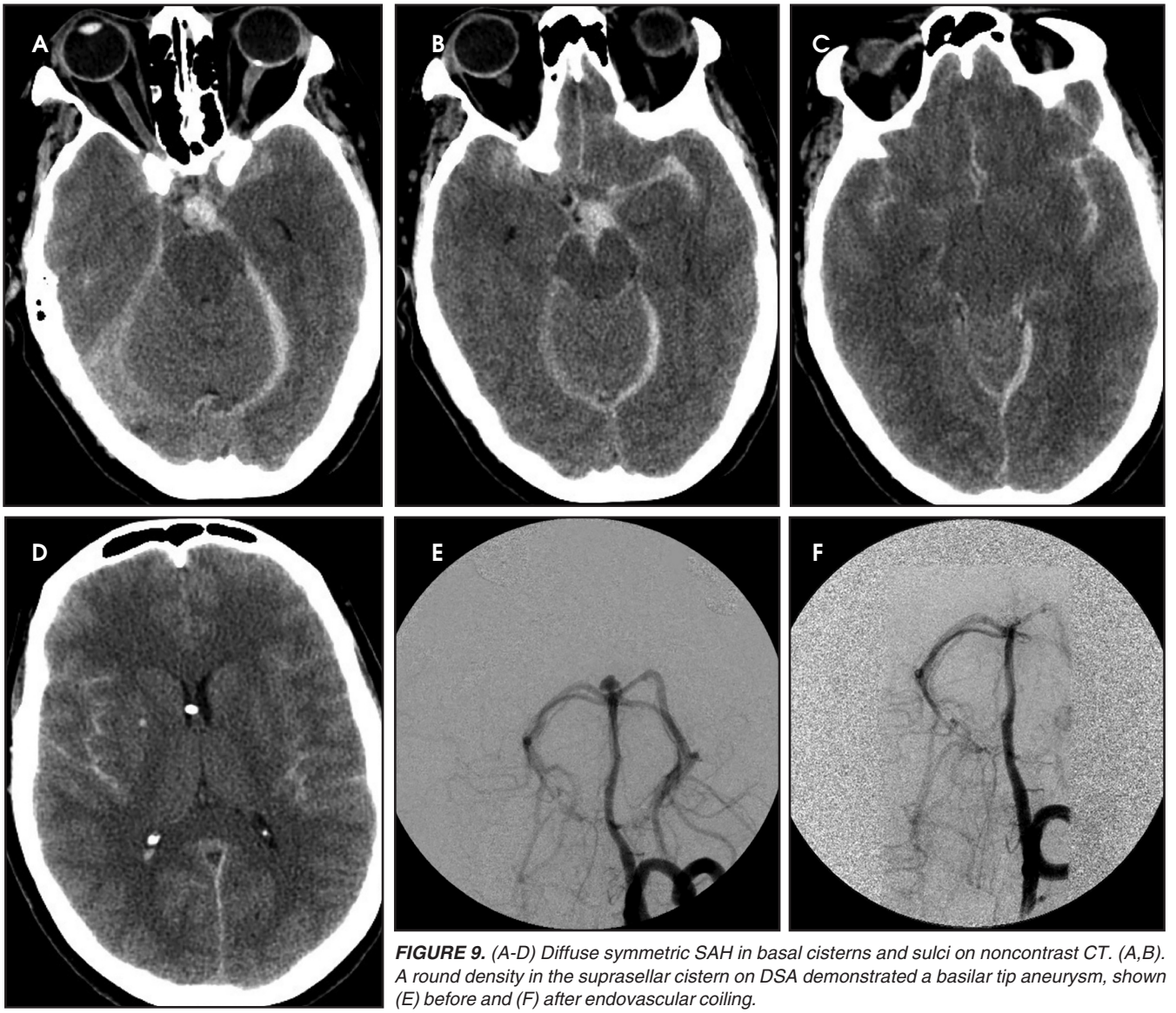

FIGURE 9. *(A-D) Diffuse symmetric SAH in basal cisterns and sulci on noncontrast CT. (A,B). A round density in the suprasellar cistern on DSA demonstrated a basilar tip aneurysm, shown (E) before and (F) after endovascular coiling.*

points at it, with AComm aneurysms bleeding into the orbitofrontal gyrus or gyrus rectus and MCA aneurysms bleeding into the operculum (Figure 12).[64] Aneurysms causing compression symptoms are more likely to rupture, and symptom localization can help identify the offending aneurysm.[50] Prompt treatment of the ruptured aneurysm is imperative. 2-4% of aneurysms will rupture again within the first 24 hours, and there is a 1-2% risk of rupture for each day during the first month following initial rupture if the aneurysm is not secured.[2,3,25,65]

SAH frequently occurs following trauma and can have multiple appearances. Such SAH tends to be more

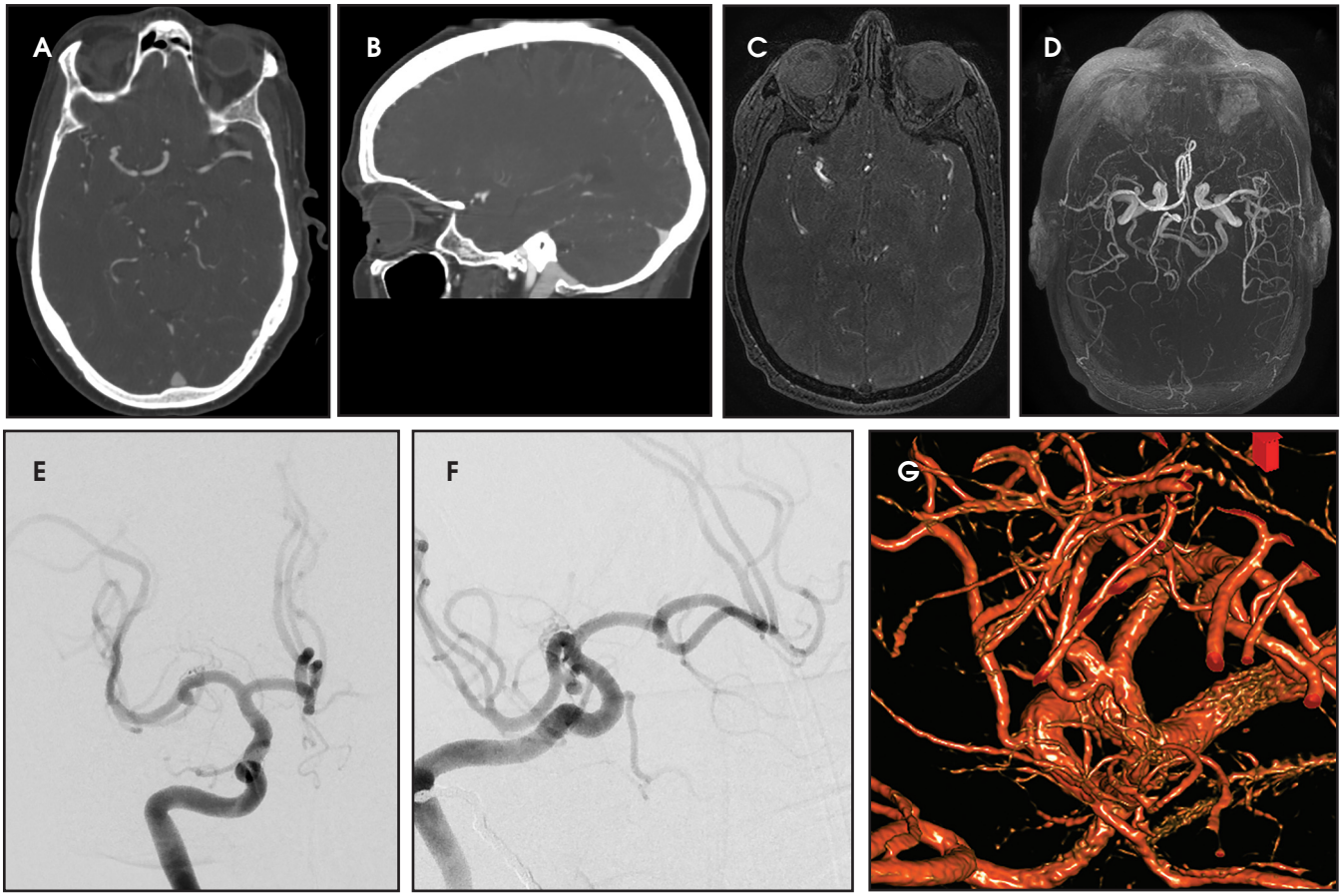

FIGURE 10. *(A, B) Saccular aneurysm of the right MCA demonstrated on CTA (C, D). MRA, (E, F) and DSA. (G) More detailed visualization of the aneurysm is provided with three-dimensional reconstruction.*

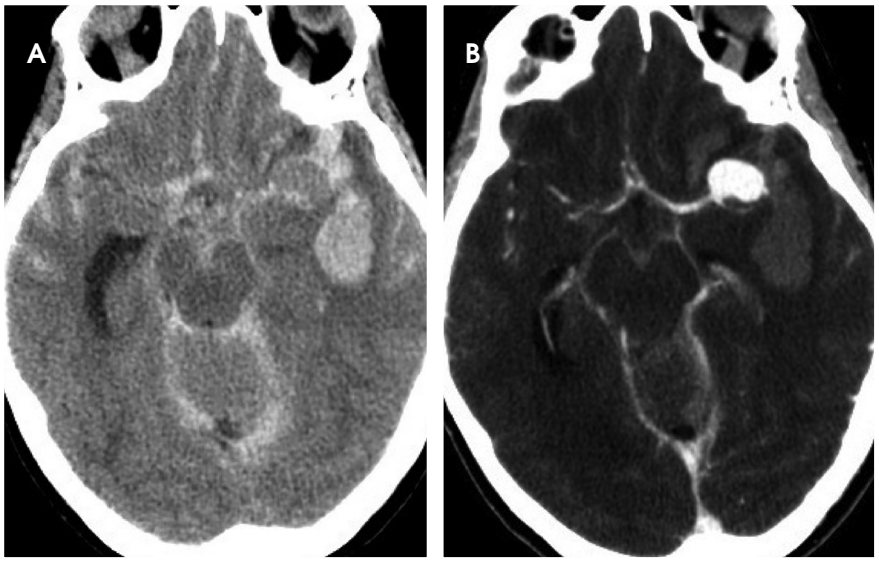

FIGURE 11. *(A) Noncontrast CT demonstrating diffuse SAH, most pronounced in the left MCA cistern. (B) Left MCA aneurysm seen on CTA.*

peripheral and localized to the site of injury (Figure 13).[64] Hemorrhage often occurs in other intracranial compartments, and important associated soft tissue or osseous injuries can be seen as well (Figure 5).[1] Worse outcomes are associated with poor initial clinical state, larger volumes of hemorrhage, EDH, midline shift, or obliteration of basal cisterns.[1,66,67] Numerous other pathologic processes can cause SAH, including nonaneurysmal vascular anomalies like arteriovenous malformations and dural arteriovenous fistulae, dissection, inflammatory vasculitides, idiopathic vasculopathy, reversible cerebral vasoconstriction syndrome, coagulopathy, neoplasms, and illicit drugs, among many others.[2,64,68]

Approximately 10% of SAH cases will yield no clear diagnosis. Within this group is a benign entity known as nonaneurysmal perimesencephalic SAH (NAPSAH).[2] This is a diagnosis of exclusion and has well-described characteristics that are important for radiologists to know well.[69-71] NAPSAH is believed to result from venous rupture in the region of the mesencephalon.[72] Its clinical presentation is distinctively different from most cases of SAH from aneurysm rupture and other

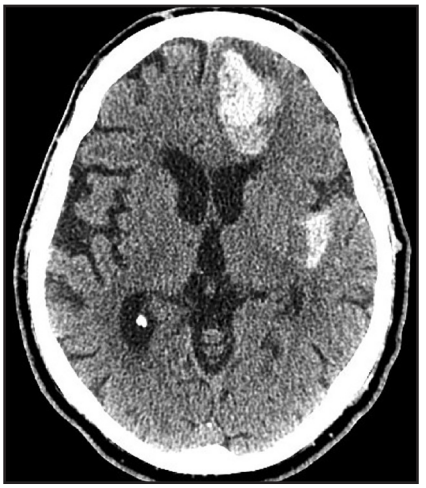

FIGURE 12. *IPH in the left frontal lobe from a ruptured AComm aneurysm. SAH is also noted in the ipsilateral MCA cistern.*

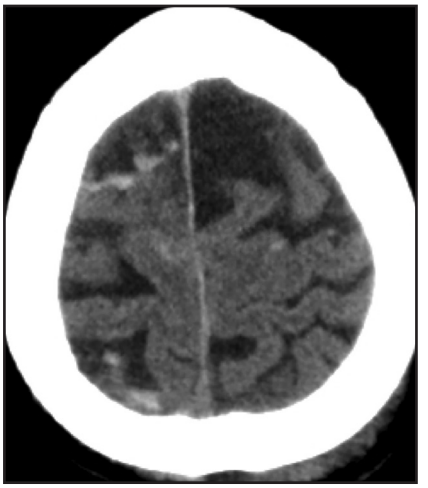

FIGURE 13. *Peripheral SAH noted in sulci of the high frontal lobe in a patient who experienced head trauma following a high-speed motor vehicle collision.*

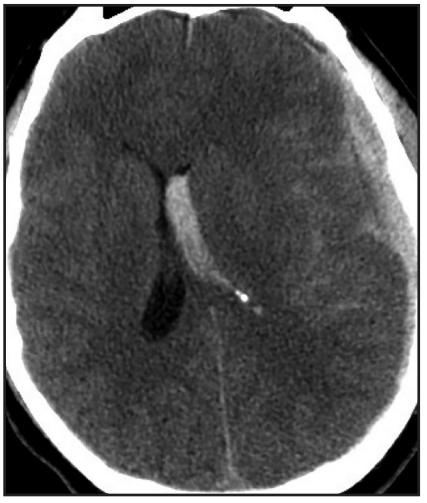

FIGURE 14. *Midline shift and subfalcine herniation noted in a patient with left SAH, SDH, and IVH.*

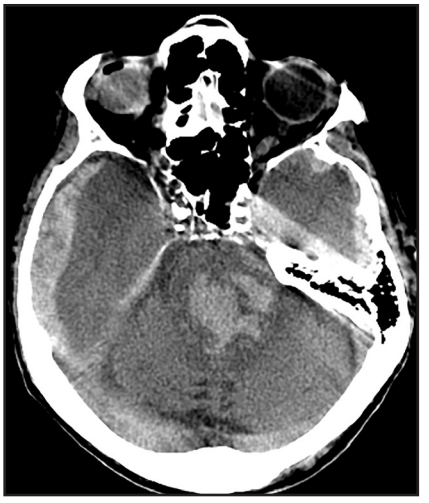

FIGURE 15. *IPH in the brainstem consistent with Duret hemorrhage in a patient with herniation due to bilateral SAH, SDH, and IVH.*

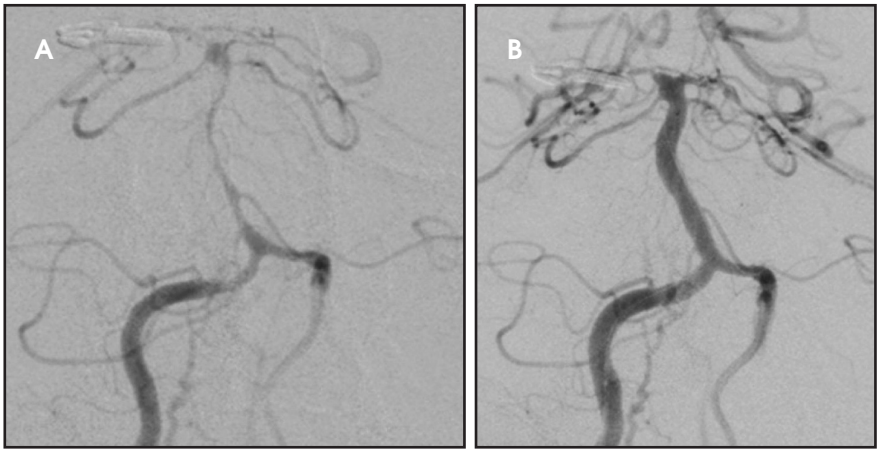

FIGURE 16. *DSA demonstrating vasospasm of the basilar artery (A) before and (B) after intra-arterial treatment in a patient who had previously undergone aneurysm clipping.*

etiologies.[2] Headaches are less sudden in onset with development over minutes rather than seconds, consciousness is never more than minimally altered, and seizures do not occur with NAPSAH.[2,64,69,73-79] This entity demonstrates characteristic appearance on CT with hemorrhage isolated in the cisterns anterior to and near the midbrain, at times located solely in the quadrigeminal plate cistern (Figure 2).[2,69,77,79-81] Trace hemorrhage layering dependently in the ventricles is allowable for this diagnosis, but frank IVH excludes NAPSAH.[2,26,69,77,79] All patients with suspected NAPSAH must undergo evaluation with DSA since small aneurysms or other etiologies not visible on noninvasive angiography may be the source of SAH.[2] 2-5% of patients with a perimesencephalic SAH pattern on CT will subsequently be diagnosed with an aneurysm on DSA.[2,69-71] Thrombosed aneurysms or very small aneurysms can elude detection on DSA, so repeat DSA has historically been recommended several weeks after an initial study.[2,82-84] Some have questioned the utility of repeat DSA, although no studies have been published demonstrating the safety of foregoing a repeat study.[82-85] NAPSAH does not carry risk of repeat hemorrhage or ischemia, so patients given this diagnosis do not require further surveillance beyond the time frame for potential hydrocephalus.[79,86] As such, it is important to strictly follow requirements for this diagnosis of exclusion to avoid false negatives and unwarranted cessation of surveillance following SAH.

Complications

Morbidity from SAH can arise from several complications that can occur at the time of ictus or in the ensuing days and weeks. The most pressing complication can be mass effect from hemorrhage. Increased intracranial pressure from any source causes distinctive herniation syndromes.[87,88] Subfalcine herniation involves displacement of the cingulate gyrus under the falx, with midline shift and medial displacement of a compressed ipsilateral ventricle (Figure 14).[87] Descending transtentorial

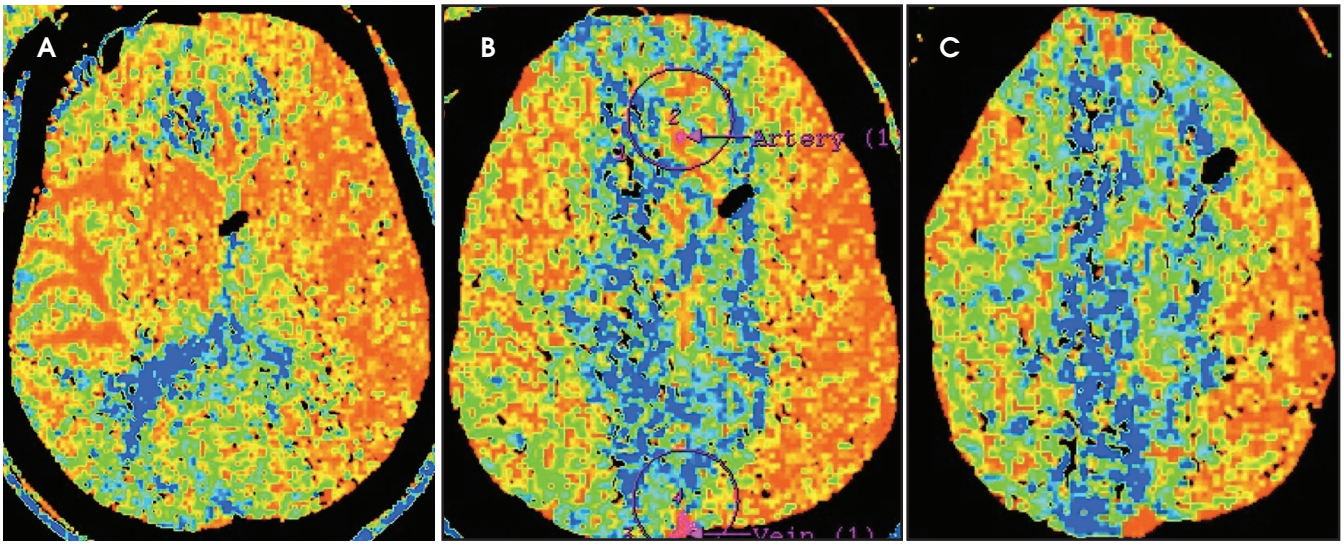

FIGURE 17. *(A-C) CT perfusion study demonstrating abnormal MTT in the right, which is worse in the left frontal, parietal, and occipital lobes and thalami, representative of ischemia from vasospasm in the ACAs and basilar artery seen in Figure 16.*

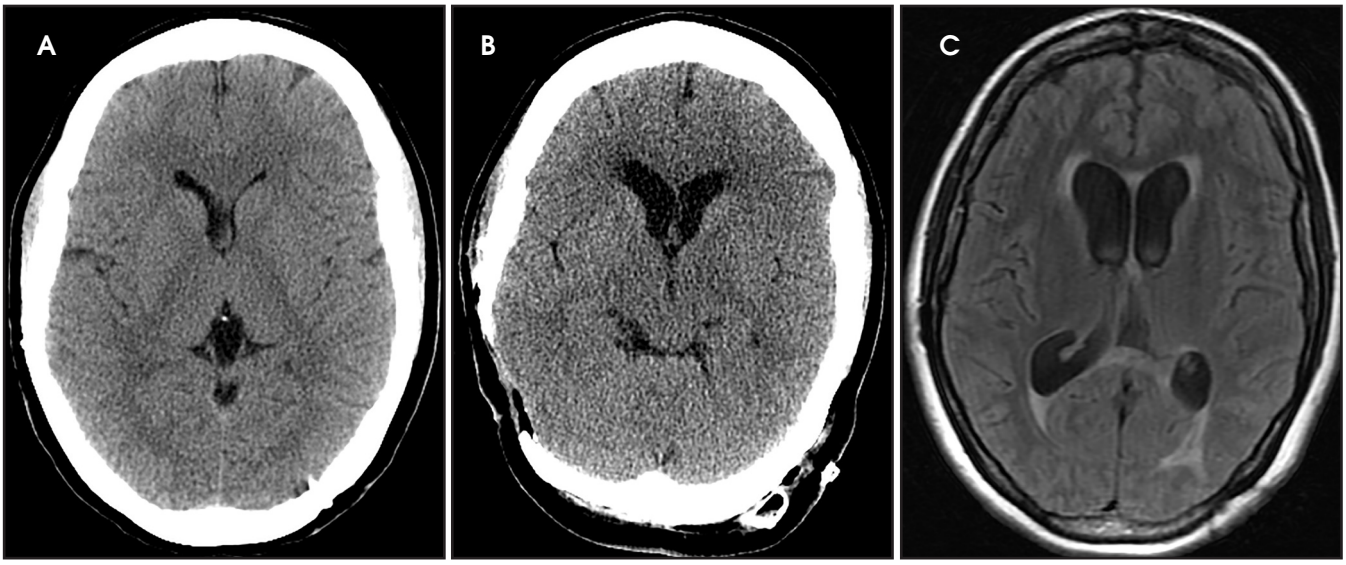

FIGURE 18. *Noncontrast CTs (A) several days and (B) several months after SAH demonstrate development of hydrocephalus. (C) Periventricular FLAIR hyperintensity represents transependymal flow.*

herniation involves medial displacement of the temporal lobe into the incisura and effacement and eventual obliteration of the basal cisterns, usually starting with the suprasellar cistern.[87] Ascending transtentorial herniation occurs with increased pressure in the posterior fossa, and herniation of the cerebellum effaces the quadrigeminal plate cistern.[87] Finally, in tonsillar herniation the cisterna magna is obliterated by cerebellar tonsils descending into the foramen magnum.[87] Mass effect is more common in cases of traumatic SAH with additional intracranial hemorrhage, although herniation may be present with isolated SAH and may be clinically unapparent.[1,89,90] Herniation is typically a surgical emergency as ischemic damage occurs from both physical compression of parenchyma and from reduced perfusion due to arterial compression.[88] Additionally, parenchymal hemorrhage in the brainstem can occur in the setting of herniation, a process termed Duret hemorrhage that can be seen on CT and MR and is associated with poor outcomes (Figure 15).[91]

Ischemia can also occur following SAH in the absence of mass effect.[2] This often occurs in the setting of vasospasm, although this is neither necessary nor sufficient for development of ischemia.[2,92] Vasospasm occurs for unclear reasons on days 3 through 12 after SAH with risk peaking on day 7.[25,65,92,93] Both vasospasm and infarct are more likely to occur with diffusely distributed SAH.[6,94-96] If SAH has an arterial source, larger volumes of blood predict subsequent ischemia, as does loss of consciousness at the time of ictus.[2,92,97,98] These factors can prepare clinicians to have appropriate levels of clinical suspicion in addition to providing recommended prophylactic

treatment with nimodipine and maintenance of euvolemia.[99] Measures to prevent vasospasm are important because no clearly superior means of screening have been identified, and treatment can be difficult.[3] Neurological deficits progress slowly and typically refer to multiple arterial territories.[92] Transcranial Doppler is employed for vasospasm screening at many centers, but investigational results have been mixed, and no randomized trials have been conducted.[2,93,99,100] CT angiography is sensitive and specific for severe vasospasm in proximal arteries, but diagnostic accuracy plummets for mild to moderate vasospasm and distal involvement.[45,101] DSA is the best modality for diagnosing vasospasm, and catheter-directed intra-arterial administration of calcium channel blockers and angioplasty can be performed for vasospasm refractory to noninvasive treatments (Figure 16).[45,99,102] Treatment approaches vary between centers, but angioplasty is reserved for vasospasm in proximal arteries.[102] Some treatment algorithms call for angioplasty only after failure of intra-arterial calcium channel blocker infusion, while others primarily treat proximal vasospasm with angioplasty primarily.[102] Regardless of the presence of vasospasm, evaluation for ischemia and infarct can be performed with CT or MR perfusion studies or diffusion weighted MR imaging.[99,101,103] More specifically, CT perfusion studies have demonstrated excellent value of mean transit time in the prediction of vasospasm on DSA and promise from blood-brain barrier permeability imaging as a physiologic biomarker that may guide treatments in the future (Figure 17).[104,105]

Hydrocephalus can occur with SAH of any etiology; it presents in up to 45% of SAH patients.[25,65,86,106-110] It can be either acute or chronic and must be diverted when symptomatic.[3] Symptoms are often subtle in onset with gradual progression, most commonly manifesting as a depressed level of consciousness.[77] Hydrocephalus is more likely to occur in older patients, with diffuse distribution of SAH, when SAH measures more than 5 mm thick, and when IVH is present.[106,111,112] Ventricular size on CT and MR is variable between individuals and has poor accuracy for diagnosis of hydrocephalus, although changes in size between different studies on the same patient correlate with level of consciousness (Figure 18).[111,112] Periventricular edema consistent with transependymal flow is a marker of hydrocephalus, and this is better seen on MR than CT (Figure 18).[113]

Conclusion

SAH can occur from a variety of etiologies and result in a wide range of outcomes. Radiologists play a key role in identifying the source of SAH and providing information for planning the most appropriate treatment, SAH features with implications for prognosis and complications of the hemorrhage.

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