Chapter 15
Aneurysmal Subarachnoid Hemorrhage

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Clinical Scenario

A 32-year-old female was brought to the Emergency Department (ED) by an ambulance after she complained to her husband of spontaneous, sudden onset, severe headache while doing yard work. On arrival, she is sleepy and only opens her eyes briefly to gentle stimulation. The light is bothersome to her. She has a medical history of pre-diabetes and asthma. Her husband remarks that she has had headaches and migraines in the past, but nothing this severe. There are no external signs of trauma. She is oriented, but only answers questions in short, one- or two-word responses. All of her extremities move well and symmetrically to command, but she is lethargic. She is little nauseated but has not vomited.

15.1 History and Neurologic Exam

Acute onset, severe headache presents a particular challenge, in so far as the potential differential diagnosis is broad, yet prompt evaluation and triage are essential. While most headache etiologies are benign, many may require swift, directed treatment that can be lifesaving. In general, taking a history in this scenario should cover:

- *Features of the headache*. Headache characteristics may be highly variable. Elucidating specific features may be helpful and even diagnostic. Determine headache onset (i.e., was it abrupt, gradual?), location (e.g., frontal, retro-orbital,

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holocephalic, posterior), duration, character (e.g., throbbing, sharp, dull, achy), aggravating factors (e.g., light, Valsalva, noise, position), alleviating factors (e.g., medication), timing (e.g., is it worsening, improving, or fluctuating?; does it relapse and remit?), and severity (i.e., 1–10 scale). Events leading up to headache onset can also be important (e.g., onset during or after strenuous activity).

- *Associated and constitutional symptoms*. Are there any associated symptoms that may be relevant, like nausea, vomiting, neck pain or stiffness, vision blurriness, double vision, dizziness, instability, neck pain, or weakness? Are there reports of any fever, chills, or infection symptoms? Are there reports of any ear pain or drainage or any sinus issues?
- *Neurologic symptoms*. Is the patient's mental status altered or is his or her level of consciousness depressed? Are there signs of high intracranial pressure? Are there any cranial nerve deficits? Is there focal or global weakness? Is there numbness or paresthesias of any extremity? Is there any erratic or unusual behavior? What is the pattern of speech?
- *History of trauma*? Determine if the headache is related to trauma. Was there any trauma in the recent history, even if it was minor? If there was trauma at time of onset of headache, such as fall, be sure to elicit if the fall happened before or after the headache started. Sometimes, headache may signal a neurologic event that caused a fall. Are there any other injuries?
- *Medical history*. What is the patient's medical history? Is there a history of headaches or migraines? Any cancer history? Are there any neurologic conditions that may predispose to headache, high intracranial pressure, or an intracranial tumor or mass lesion? Is there a history of a procoagulable state that might cause venous sinus thrombosis? Or a bleeding disorder that might have led to intracranial hemorrhage? Is there a history of hypertension or hypertensive emergency? Is there history of immunocompromise that may predispose to intracranial infection? Were there any recent infections? Are there potentially relevant, pre-existing cardiovascular, neoplastic, hematologic, or infectious conditions that might place this patient at risk for intracranial hemorrhage or intracranial aneurysm? Are there any conditions, like polycystic kidney disease, that might predispose to or be associated with intracranial aneurysms?
- *Medications*. Is the patient taking any antiplatelet or anticoagulant medications? Is the patient taking any procoagulant medications? This includes a review of herbal supplements—many of which have antiplatelet, anticoagulant, or procoagulant activity. Have the patient's medications changed recently (e.g., change of antihypertensive regimen)? All medications, both current and recent, should be recorded and reviewed.
- *Surgical history*. Does the patient have a history of any intracranial or neurosurgical procedures? Does the patient have a history of tumor removal, and could he or she potentially now have a brain metastasis? Has the patient undergone any recent sinus, dental, or otologic procedures that could have precipitated intracranial infection?

- *Social history*. Is there a history of substance use or abuse? Cocaine and amphetamine use can precipitate headaches via vasculitis, SAH, or hypertensive crisis. Eliciting work history, hobbies, and living conditions may also be relevant.
- *Family History*. Is there a family history of headache disorder? Are there any neurologic conditions or cancer syndromes that run in the family? Are there any family members with cerebral aneurysms or prior SAH? Many patients will remember these events as "stroke" or "bleeding on the brain."

Try to obtain further history from relatives, friends, or anyone else available if the patient is not communicative, confused, or too lethargic. This extra effort can be critical and can completely change the direction of workup and management.

The initial encounter with every patient should involve assessment of the "ABCs": airway, breathing, and circulation. This is of particular importance in a patient with a depressed level of consciousness, and one should be constantly reevaluating the need for airway protection and mechanical ventilation. If there is concern for trauma, assessment of circulatory blood volume is critical in the event there might have been external or internal hemorrhage. Essential elements of physical and neurologic assessment include:

- *Vital signs*. Vital signs should be reviewed. Headache may be accompanied by tachycardia and hypertension secondary to pain; however, other causes should be considered. Bradycardia, accompanied by depressed level of consciousness, should raise concern for raised intracranial pressure. Bear in mind that bradycardia, alone, may reflect beta-blockade or a normal physiologic state in the appropriate individual. Fever should trigger concern for infection but note that patients can experience fever with non-infectious intracranial pathologies as well. For instance, fever may also occur in the setting of recent clinical or ongoing subclinical seizure.
- *Glasgow Coma Scale (GCS) score*. Quickly calculate the patient's GCS. This involves (a) eye opening, (b) verbal response, and (c) motor response. With a depressed GCS (14 or less), imaging should be obtained urgently, usually starting with non-contrast head CT. If there is reasonable concern for intracranial hemorrhage and secondary neurologic decline or cardiopulmonary complications, the patient should be monitored when going to the CT scanner and should be accompanied by appropriate level of staff.
- *Cranial nerve, motor, and sensory exam*. A cranial nerve exam should be performed, followed by a brief motor and sensory exam. Remember that just because a patient is "nonfocal" does not mean there is no intracranial concern. Global level of consciousness is just as important. A globally depressed level of consciousness—independent of a lateralizing or focal finding—may reflect bilateral cerebral or brainstem level dysfunction.
- *Cutaneous signs of trauma or prior surgery*. During the exam, evaluate for any signs of trauma or potential occult injuries. Evaluate for surgical scars, particularly on the head, that may indicate previous cranial surgery.

- *Meningismus*. With concern for infection, be certain to check for nuchal rigidity and light sensitivity. Note, however, that these may be present in the setting of spontaneous SAH without infection.

15.2 Differential Diagnosis

The differential diagnosis for headache can be broad. A description of severe, spontaneous, sudden onset headache narrows it somewhat. Strong familiarity with the differential diagnosis in this setting is crucial because thorough history is not always available and initial imaging can be negative or equivocal. The term "thunderclap headache" is often used to describe severe, sudden onset headache that reaches maximum intensity within a few minutes. However, in reality, it is often difficult to determine if the headache meets these specific criteria, and the pathologies that supposedly present with thunderclap headache can also present more gradually.

15.2.1 Common Causes of Sudden Onset, Severe Headache

Aneurysmal subarachnoid hemorrhage. With sudden onset, severe headache—such as in the case of our patient—this diagnosis should always be considered. Classically, patients will describe the "worst headache of life." However, the headache may not be particularly severe, mildly symptomatic cases are frequently misdiagnosed on initial encounter, most frequently as migraine or tension-type headaches [1]. Index of suspicion should remain high. Patients may also have nausea, vomiting, neck pain, photophobia, depressed level of consciousness, confusion, seizures, cranial nerve palsies, or other neurologic deficits. Aneurysmal SAH is highly possible in our patient, but certainly other etiologies are conceivable at this point also. It is important to note that aneurysmal SAH patients are often found down and are brought in as trauma patients, whether or not the hemorrhage preceded a true trauma. This is important to consider when reviewing cases of purported traumatic SAH.

Perimesencephalic (pretruncal) subarachnoid hemorrhage. This entity may present similarly to aneurysmal SAH, but is usually less severe. "The patient looks much better than the CT" is a common quip in this setting. By definition, no explanatory lesion is found on angiography, and the SAH fits a specific pattern. The blood is typically centered immediately anterior the midbrain or pons but does not extend to the lateral Sylvian fissures or superficial interhemispheric fissure, and there is no frank intraventricular hemorrhage [2]. Patients may present with nausea, vomiting, meningismus, and photophobia, but neurologic deficits and significantly depressed level of consciousness are uncommon. This is a diagnosis of exclusion and could fit with our patient's presentation as well.

Reversible cerebral vasoconstriction syndrome (RCVS). This is a group of entities with an evolving definition, but, in general, characterized by single or recurrent thunderclap headaches and reversible segmental vasoconstriction on angiographic imaging [3]. There are many associations, including sexual activity, pregnancy, medications, and illicit drugs. SAH may occur in association with RCVS, though the pattern of bleeding more commonly involves the convexities than the basal cisterns.

Without the benefit of imaging, all three of these entities remain possibilities for our patient.

15.2.2 Others Causes of Sudden Onset, Severe Headache

The following are not necessarily less common causes of headache, but are typically characterized either by more gradual onset or lesser severity of symptoms.

Non-perimesencephalic, non-aneurysmal subarachnoid hemorrhage. Spontaneous SAH without aneurysmal source or fitting a perimesencephalic pattern is uncommon, with a wide range of potential underlying etiologies. These include arteriovenous malformations, arteriovenous fistulas, cavernous malformations, cerebral amyloid angiopathy, coagulopathies, qualitative or quantitative platelet dysfunction, and many of the pathologies listed below. The patient may be asymptomatic or manifest with headache, seizure, or neurologic deficit.

Meningitis. The patient may also have a fever, photophobia, meningismus, leukocytosis, focal neurologic deficit, or seizure. The patient may have a history of recent sinonasal procedure, neurosurgical procedure, immunocompromise, bacteremia, or endocarditis. Systemic or intracranial infectious processes may precipitate formation of mycotic aneurysms, which can rupture, causing SAH. These aneurysms and their hemorrhages are often distal in the cerebral vasculature.

Venous sinus thrombosis. This is suggested by signs of elevated intracranial pressure, including papilledema, headache, abducens palsy, or other extraocular movement restriction. The patient may be post-partum or procoagulable. There may be global or focal neurologic deficits.

Pituitary apoplexy. This diagnosis is supported if there is a history of a pituitary adenoma or signs of hypopituitarism. The symptoms on presentation may be quite varied, including severe headache, sudden visual loss and/or ophthalmoplegia, and cardiovascular collapse or diabetes insipidus due to acute hypopituitarism.

Hypertensive crisis. The patient will typically have a history of hypertension, often with recent medication change or compliance issues. However, hypertension can be a consequence of headache, or the hypertension may be caused by another etiology.

Posterior reversible encephalopathy syndrome. This is usually more insidious in onset and may be accompanied by seizure and neurologic deficits. It often occurs in the setting of hypertensive crisis, pre-eclampsia, or immunosuppressive therapies.

Migraine. Patients usually have a history of migraines. They are usually more gradual in onset and may be preceded by an identifiable aura.

Brain tumor. Headaches from brain tumors are usually gradual in onset, but hemorrhage or hydrocephalus may precipitate an acute onset or exacerbation.

Hydrocephalus. Headaches from hydrocephalus are usually more gradual in onset and usually accompanied by progressive altered mental status. Papilledema and abducens palsy may be present. Headaches due to tumor or hydrocephalus may be characteristically worse in the morning upon waking.

Carotid or vertebral artery dissection. Dissection may be related to a traumatic event and can precipitate an ischemic stroke. Cervical dissection may cause a Horner's syndrome.

Other vasculitides/vasculopathies. Presentation will be variable. There may be hemorrhage, and vascular imaging may or may not be diagnostic at initial screening.

Other causes of headache to consider. Sinusitis, spontaneous intracranial hypotension, intraparenchymal hemorrhage, subdural hemorrhage, ischemic stroke, giant cell arteritis, colloid cyst of the third ventricle, acute angle-closure glaucoma.

15.3 Diagnostic Evaluation

The initial diagnostic test for sudden onset, severe headache should be **CT of the head without contrast**. This modality has high sensitivity for acute SAH, other intracranial hemorrhages, mass lesions, and hydrocephalus. However, it may be negative in the case of RCVS, meningitis, venous sinus thrombosis, hypertensive crisis, migraine, or dissection. It is interesting to note that all of these pathologies—save for migraine—can rarely cause small to moderate amounts of SAH.

Sensitivity of non-contrasted head CT is highest in the first few days after SAH, including 98.7% within the first 6 h (if there are no neurologic deficits) [4]. If the patient is presenting in delayed fashion, CT may be negative [5]. In the case of a negative non-contrast head CT but sufficient clinical suspicion for SAH (and without other cause seen on that initial study), a **lumbar puncture** should be performed [6]. CSF should be analyzed microscopically for the presence of red blood cells. Xanthochromia—the result of red blood cell lysis and breakdown—may be apparent grossly upon collection or after centrifuge of the sample. Of note, red blood cells may also be present in the CSF in the setting of meningitis secondary to herpes simplex virus. Beyond cell count, other routine studies such as protein, glucose, and culture should not be neglected. Opening pressure should also be obtained, but is only accurate in the lateral decubitus or prone position.

In virtually all cases of acute SAH, **CT angiography (CTA) of the head** should be performed next to evaluate for aneurysmal origin or other vascular lesions. The exception would be cases that are clearly trauma-induced, mild-moderate SAH where there was no preceding headache or neurologic complaint. CTA of the head should also be performed alongside lumbar puncture in CT-negative cases with sufficient clinical suspicion for aneurysmal SAH. Sensitivity of modern CTA for aneurysms <3 mm is over 87% [7]. CTA may also reveal the segmental vasoconstriction representative of RCVS, but sensitivity is limited. **MR angiography (MRA) of the head** is an alternative but typically takes longer to obtain and requires a longer acquisition time. It is also more expensive and may not be as readily available at CT in many centers, particularly at night. The benefit of MRA is that it can be done in patients with renal failure as it does not require contrast.

Concurrent with initial imaging, **basic lab work** should be sent on these patients. This should include complete blood count, basic chemistry, and coagulation factors. Urine or serum toxicology can also be considered in cases of altered mental status, suspected ingestions, or suspected illicit drug use. In women, a pregnancy test should be sent, even with very low suspicion.

While the sensitivity of modern, high-quality CTA is quite high for vascular lesions in this setting, **digital subtraction catheter angiography** (DSA) remains the gold standard. In cases of spontaneous SAH, or when there is uncertainty on the role of trauma as a cause or result, catheter angiography should be considered after negative CTA if there is still reasonable suspicion for an aneurysm or vascular lesion. Furthermore, many institutions, including ours, will still perform catheter angiography even with an explanatory lesion on the CTA. Catheter angiography helps to characterize the lesion and related anatomy, may reveal occult lesions, and permits real-time therapeutic intervention, if appropriate. This modality is also more sensitive than CTA for the vasoconstrictive changes seen in RCVS and vasculitis.

CT venography of the head would be most beneficial when there is sufficient suspicion of venous sinus thrombosis. This diagnosis might be considered in a patient with a procoagulable condition or taking a procoagulant medication, with notable headache and with or without depressed level of consciousness. Non-contrast CT head may show cerebral edema, parenchymal hemorrhage, small amounts of SAH, or hyperattenuation in the suspected location of a venous sinus, which should prompt further investigation of this diagnosis with CT venography. **MR venography of the head** can be considered as an alternative to CTV.

Brain MRI with and without gadolinium can be useful and diagnostic in many causes of severe headache. Brain MRI can show SAH not seen on CT (particularly in subacute hemorrhages more than several days old) [8], may reveal a mass lesion such as cavernoma or tumor, may reveal edema secondary to venous sinus thrombosis or in the setting of PRES, may reveal a pituitary tumor with signs of apoplexy, or may reveal signs of meningitis or discrete infection. The threshold for obtaining MRI should be low.

15.4 Clinical Decision-Making and Next Steps

Our patient presented with acute, severe, spontaneous headache without trauma. This presentation, in and of itself, should raise suspicion of aneurysmal SAH as a diagnosis to be ruled out quickly, regardless of mental status. She should undergo non-contrast CT of the head as initial imaging in this scenario. Even with headaches that are less acute or less severe, but associated with altered mental status or neurologic deficit, non-contrast head CT should also be strongly considered. In the case of our patient, head CT (Fig. 15.1) showed thick SAH in the basal cisterns bilaterally and the proximal left Sylvian fissure, with small amounts of layered blood in both occipital horns. The presence of subarachnoid hemorrhage and the specific distribution of blood—independent of identification of a discrete aneurysm—is sufficient to set her on the management trajectory for an aneurysmal bleed.

A neurosurgeon should be consulted emergently upon diagnosis of SAH. The non-contrast head CT should also be assessed for signs of developing hydrocephalus, which is common after SAH and frequently requires CSF diversion. There may also be signs of cerebral edema, midline shift, or herniation that may alter management. For communication and prognostication purposes, the initial neurologic status is classified by severity of deficit using the Hunt and Hess scale and World

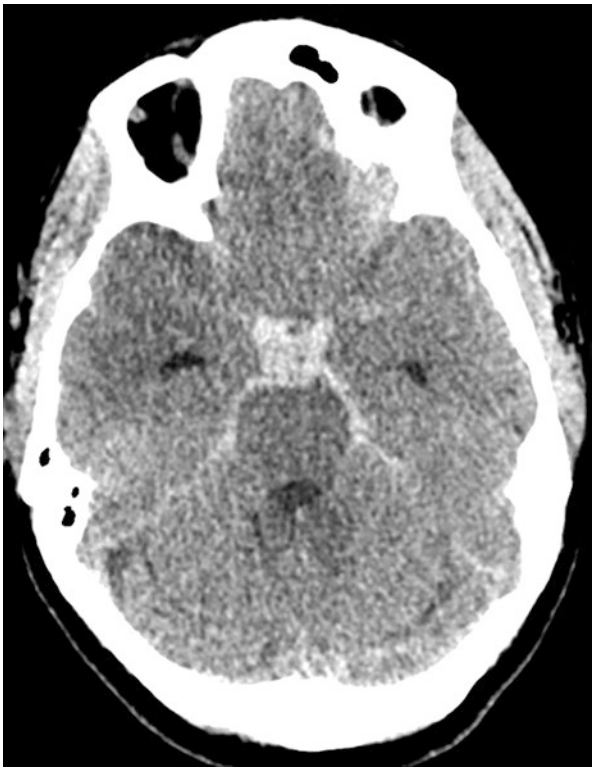

Fig. 15.1 Axial non-contrast head CT showing diffuse subarachnoid hemorrhage in the basal cisterns, concerning for aneurysmal rupture

Federation of Neurological Surgeons (WFNS) grade [9, 10]. This patient would receive a Hunt and Hess score of 3 (lethargic) and WFNS grade of 2 (GCS 13–14 without deficit).

Initial lab work should be obtained, including CBC, basic chemistry panel, INR, and PTT. The labs should be reviewed for thrombocytopenia or coagulopathy. Low sodium should be investigated, as should any other electrolyte derangement. Rarely does renal failure prevent use of iodinated contrast for emergent CT or catheter angiography in this setting. Our patient's sodium was 134, so it was planned to recheck this value after a short interval, to exclude a further drop. Approximately 30–50% of aneurysmal SAH patients develop hyponatremia [11]. Syndrome of inappropriate ADH (SIADH) accounts for approximately two-thirds of cases, but cerebral salt wasting, iatrogenic hypervolemia, dehydration, and cortisol deficiency are also common [11]. Hyponatremia may worsen cerebral edema and mental status and may lower seizure threshold.

If not done already at this point, the patient's medical history and medications should be reviewed to ensure there is no history of coagulopathy, bleeding diathesis, antiplatelet use, or anticoagulant use. If yes to any, it should be addressed emergently to prevent further hemorrhage. This screen was negative in our patient.

The patient's vital signs should be monitored continuously. Blood pressure should be controlled to prevent re-hemorrhage, with the latest guidelines suggesting a ceiling of <160 mmHg [6]. Our patient was hypertensive, possibly from pain, so a vasodilator drip was initiated. The patient's mental status and neurologic exam should be re-evaluated at least every hour to ensure there is no need for intubation for airway protection or measures to control an elevated ICP. With a GCS score of 14, our patient did not require intubation and did not raise concern for elevated ICP. Many providers will administer a prophylactic antiepileptic drug (AED) at this point for seizure prophylaxis, although the effect on functional outcome remains controversial. Early seizures occur in about 15–20% of aneurysmal SAH patients, with increased blood burden and poor clinical grade as risk factors [12, 13]. Our practice is to continue the AED regimen for at least the duration of hospitalization given the favorable side effect profile of newer agents, but many choose to discontinue prophylaxis after securing the aneurysm.

No lumbar puncture was necessary in this patient as the SAH was proven on non-contrast head CT. A lumbar puncture would have been necessary if the non-contrast head CT was negative. Some centers use MRI instead of lumbar puncture to rule out SAH in this setting, depending on pre-test probability.

If non-traumatic SAH is identified by non-contrast CT head or LP, the next step would be to obtain a CTA of the head. The primary goal is to rule out aneurysm, which optimally should be secured within 24–48 h due to the high risk of recurrent hemorrhage, with the potential for neurologic worsening. CTA may also identify another culprit vascular lesion, such as arteriovenous malformation, arteriovenous fistula, arterial dissection, RCVS, or other vasculitis/vasculopathy. Some of these entities will require directed management, making accurate diagnosis important. In this patient, CTA did not show an aneurysm, vascular lesion, or other abnormality. Despite high sensitivity of modern, well-performed, high-quality CTA, catheter

digital subtraction angiography (DSA) is the gold standard and should be performed to more definitively rule out aneurysm or these other pathologies. Blood burden may obscure the etiologic vascular lesion and, again, CTA sensitivity is more limited for aneurysms <3 mm.

At this point, the differential diagnosis for SAH should be revisited. With a classic "aneurysmal" pattern of SAH—that is, diffuse blood in the basal cisterns—the most likely differential includes occult aneurysm, non-aneurysm vascular lesions (e.g., AVM, AVF, cavernoma), and perimesencephalic SAH. Other causes of SAH are more likely to cause focal or convexity/cortical SAH. This list of other causes is long and includes RCVS, mycotic aneurysm, arterial dissection, PRES, venous thrombosis, tumor, ischemic stroke, cerebral amyloid angiopathy, cocaine/amphetamine abuse, thrombocytopenia, anticoagulation use, spinal lesions, and other vasculitides/vasculopathies. Alternate etiologies might be entertained if formal catheter angiogram does not show an aneurysm, or if the clinical scenario suggests. Further investigation in such cases would likely include MRI of the brain ± cervical spine, with and without gadolinium.

Despite a negative CTA in our patient, catheter angiography (Fig. 15.2) did reveal a small saccular aneurysm of the left internal carotid artery at the branchpoint of the left posterior communicating artery. This, therefore, fit with an overall diagnosis of aneurysmal SAH, with a falsely negative initial head CTA. This also reinforces why clinical suspicion is so important in driving the diagnostic evaluation.

All aneurysmal SAH patients should be given enteral nimodipine and admitted to the ICU with close cardiopulmonary and neurologic monitoring [14]. The aneurysm should be secured via microsurgical clipping or by endovascular means within 24–48 h in order to prevent re-hemorrhage and consequent poorer outcomes [15]. Our patient was transported to the neuro-ICU, and a plan was made for aneurysm

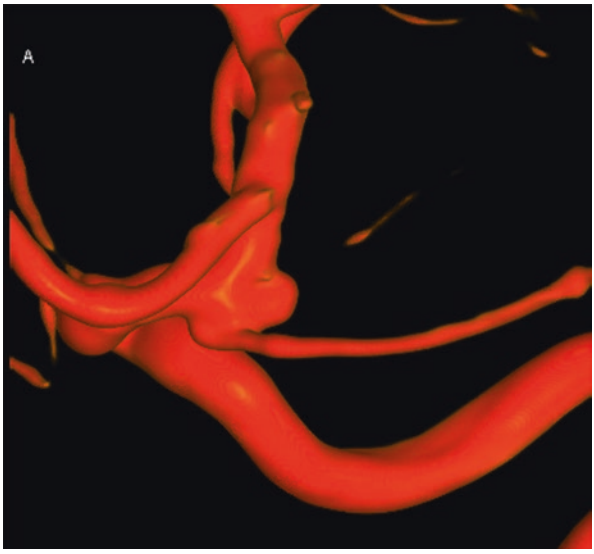

Fig. 15.2 Three-dimensional reconstruction of a digital subtraction catheter angiogram after injection of the left internal carotid artery showing a broad-based saccular aneurysm at the branch point of the left posterior communicating artery

obliteration the next morning given that it was already late evening. Emergent surgical intervention is typically reserved for continued, actively extravasating, large space-occupying hematoma requiring evacuation, or need for bony decompression.

In general, endovascular treatment is favored when the patient is a candidate for both endovascular and surgical obliteration. For the 2143 patients with ruptured intracranial aneurysms randomized in the International Subarachnoid Aneurysm Trial (ISAT), there was a lower rate of death or dependence in the endovascular group (23.5%) compared to the microsurgical clipping group (30.9%, $p = 0.0001$) [16]. Similarly, in the Barrow Ruptured Aneurysm Trial (BRAT) of 472 patients, there was a lower rate of poor outcome (mRS >2) in the endovascular (23.2%) compared to the surgical group (33.7%, $p = 0.02$) [17]. Endovascular management is also often chosen for older patients, patients with worse grade on presentation, and basilar apex aneurysms.

Surgical clipping and endovascular coiling were both considered for this patient, but microsurgical clipping was favored given a broad neck of the aneurysm, young age of the patient, and ability to fenestrate the lamina terminalis concurrently. While the aneurysm probably could—from a purely technical perspective—be coiled with stent-assistance, the use of a stent would have required antiplatelet therapy which is relatively contraindicated and disfavored in the case of acute SAH—particularly when clipping is also a viable option. For the same reason, flow diversion is typically avoided in the setting of acute SAH.

Several hours later, the nurse noted that the patient was only opening her eyes to painful stimulation, was only withdrawing to painful stimuli in her extremities, and was only mumbling when asked questions. Her pupils were 4–5 mm, equal, and reactive. Decline in mental status at any point should prompt re-imaging with non-contrast head CT. However, before traveling to the scanner, the patient's airway should be secured by intubation (given a GCS of 8). Early decline in mental status can be secondary to any number of issues, including seizure, re-hemorrhage, hydrocephalus, increased intracranial pressure, herniation, and even, hyponatremia. Therefore, labs should be rechecked as well. Her sodium came back at 135. Her head CT (Fig. 15.3) showed stable hemorrhage compared to prior imaging, but now with moderate ventriculomegaly and loss of defined cerebral sulci. Her decline in mental status was most likely attributable to hydrocephalus, and she met criteria for urgent, temporary CSF diversion. Rates of acute hydrocephalus in aneurysmal SAH vary greatly depending on the study population, ranging from 25% to 75% [6]. Presence of ventricular blood and amount of total blood are predictive of deterioration in level of consciousness from hydrocephalus [18]. Amount of subarachnoid blood and presence of intraventricular blood is also predictive of cerebral vasospasm, as objectified by the Fisher grade.

In a scenario of declining mental status (usually with a GCS of 8 or below) and imaging evidence of hydrocephalus or elevated intracranial pressure, a bedside external ventricular drain (EVD) is typically placed. An EVD may also be placed in borderline patients who will be under general anesthesia for catheter angiogram or other procedures. This procedure was performed emergently in our patient. Upon

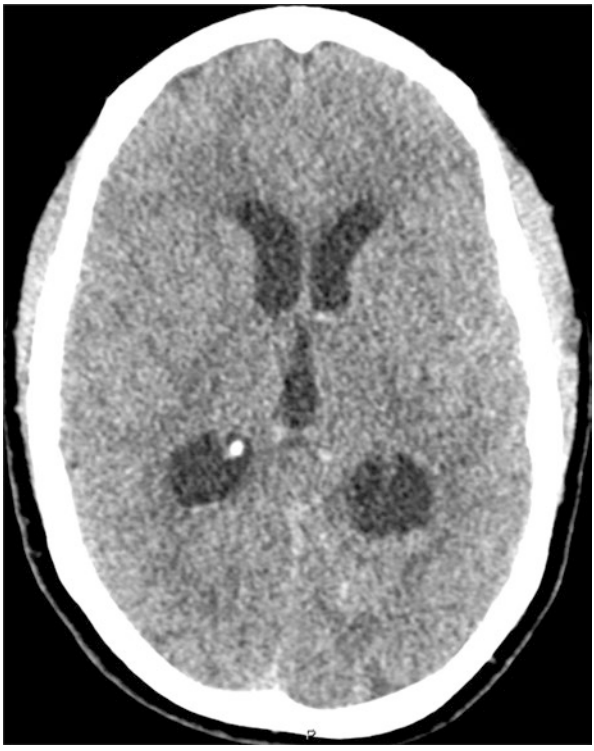

Fig. 15.3 Axial non-contrast head CT showing ventricular enlargement from developing hydrocephalus

placing the ventriculostomy catheter, her opening pressure was noted to be approximately 30 mmHg. A head CT was performed to confirm catheter location, and she was drained continuously, with caution not to over-drain in the setting of a still-unsecured aneurysm. Typically, we start with a height setting of around 20 cmH_2O. Shortly after, she began to open her eyes and follow commands again. Of note, lumbar drainage is also an option in patients without intracranial mass lesions, herniation, or obstructive pathology.

Hours later, in the early morning, the nurse noted another decline in exam. The patient was not opening her eyes and was localizing to pain with the left upper extremity but was just withdrawing to pain in the right upper extremity and bilateral lower extremities. Her pupils were 3–4 mm, equal, and reactive. As before, labs should be rechecked and consideration made for re-hemorrhage, seizure, and increased intracranial pressure. The EVD was confirmed to be patent, and the EVD was recording an ICP of 9 mmHg with a good pressure waveform, excluding hydrocephalus or increased intracranial pressure as a cause of her exam decline. The patient was taken for stat non-contrast head CT, which did not show any new hemorrhage; the ventricles had decreased in size since last imaging. The sodium came back at 126. Seizure should be strongly considered in this setting, given the lack of radiographic explanation and the fact that hyponatremia is known to lower seizure threshold. The patient was loaded with a second AED and placed on continuous

EEG, which showed epileptic activity. Hypertonic saline was also given intravenously to bring her sodium back into the normal range. Her mental status improved over the next few hours as a result. While quick correction of sodium in this setting is appropriate given both the acute drop and that the patient is symptomatic, greater caution is necessary when sodium drop has occurred more slowly (i.e., >24 h).

Later in the day she underwent a successful and uneventful craniotomy for microsurgical clipping of the left posterior communicating artery aneurysm. Her neurologic exam upon return to the ICU was somnolent but eyes open to voice, pupils 3 mm and reactive, following commands with good strength in all four extremities symmetrically.

Even after securing the aneurysm, the course of these patients can be quite tenuous. ICU comorbidities are common, such as pneumonia, cardiac ischemia or arrythmia, and venous thromboembolism. Neurologically, the primary focus is preventing and managing vasospasm and delayed cerebral ischemia (DCI). This phenomenon usually occurs 4–21 days after the ictal event and can be predicted by initial subarachnoid and ventricular blood burden via the Fisher and modified Fisher scores [19, 20]. It is important to recognize the difference between vasospasm, an angiographic finding, and DCI, the ischemic event and territory, which do not always correspond [21].

Current recommendations for prevention of vasospasm/DCI are to maintain euvolemia and normal circulating blood volume [6]. Nimodipine is given for 21 days or until hospital discharge, whichever is sooner. High-risk patients are typically monitored in an ICU setting and kept in the hospital for at least 14 days. Transcranial doppler ultrasound (TCD) may be used as a screening tool but can be inconsistent. TCD and radiographic evidence of vasospasm do not necessarily warrant treatment, and heavier reliance should be placed on the clinical exam.

By day 4, the patient had been doing well neurologically but was still intubated; EVD weaning had not yet been attempted. Around mid-afternoon, the nurse noted that the patient was only opening her eyes to voice, was localizing with the left upper extremity, withdrawing both lower extremities, and not moving the right upper extremity. Otherwise, her cranial nerves were unremarkable. Just as with previous exam declines, one should consider new hemorrhage, hydrocephalus, increased intracranial pressure, herniation, and seizure. However, she was now in the vasospasm/DCI window, so this was a possibility as well. Radiographic vasospasm occurs in about two-thirds of patients, and DCI in about 20% [21]. Our patient's modified Fisher score was 4, making her risk of DCI higher, at about 40% [20].

At bedside, the ICP was confirmed in the mid-normal range with a good waveform on the monitor. The CSF was clear, without new blood. The patient was hemodynamically stable, with a mean arterial pressure (MAP) 83 mmHg and systolic blood pressure 129 mmHg. The next step was emergent imaging with at least head CT and CTA. CT-perfusion (CTP), if available, may provide an estimate of potentially salvageable ischemic penumbras [22].

CTA/CTP revealed diffuse bilateral vasospasm with a large area of hypoperfusion in the left MCA territory without completed infarct, compatible with ongoing

DCI. At this stage, blood pressure augmentation should be employed with close monitoring for improvement in the clinical exam. The augmented goal range is usually decided based on the patient's baseline up until this point, and often requires both fluid and vasopressor infusions. A good starting point is 10–20% higher than baseline without vasopressors. Despite several hours of successful elevation in her MAP and SBP, the patient did not respond. She was taken to the angiography suite to undergo local administration of intra-arterial vasodilators [23]. On return, she was localizing both upper extremities symmetrically. Blood pressure augmentation continued as she was at high risk for further ischemic events.

Fortunately, the rest of her hospital course was uncomplicated. She was weaned from the ventilator and ventricular drain a few days later. Acute hydrocephalus after hemorrhage often resolves, and only about 20–60% of patients with temporary drainage will need permanent shunting [24]. The likelihood of shunt dependence is greater in the setting of intraventricular hemorrhage and/or vasospasm [25]. Before removal, ventricular drains are typically "challenged" by clamping and assessing for radiographic change or clinical decline, neither of which occurred in our patient. She was discharged to inpatient rehabilitation on hospital day 16.

15.5 Clinical Pearls

- Aneurysmal SAH should be on the differential for any acute, severe headache or change in mental status; it is frequently missed.
- There are many causes of spontaneous SAH. Vascular imaging is warranted—at minimum, with CTA—to evaluate for aneurysmal source or vascular lesion in the setting of spontaneous SAH.
- Maintain a low threshold for obtaining a formal cerebral angiogram—even if CTA is negative—when clinical suspicion is high.
- Trauma may be the end result of an aneurysmal rupture, not necessarily the cause.
- Rapid diagnosis is key. The early re-hemorrhage rate is high. Ideally, aim to secure the aneurysm within 24 h.
- Even after securing the aneurysm, patients require extreme vigilance and specialized care given high risk for both neurologic and non-neurologic complications, including vasospasm/DCI, pneumonia, and thromboembolic events.

References

1. Suarez JI, Tarr RW, Selman WR. Aneurysmal subarachnoid hemorrhage. N Engl J Med. 2006;354(4):387–96.
2. Mensing LA, Vergouwen MDI, Laban KG, Ruigrok YM, Velthuis BK, Algra A, et al. Perimesencephalic hemorrhage: a review of epidemiology, risk factors, presumed cause, clinical course, and outcome. Stroke. 2018;49(6):1363–70.

3. Ducros A, Boukobza M, Porcher R, Sarov M, Valade D, Bousser MG. The clinical and radiological spectrum of reversible cerebral vasoconstriction syndrome. A prospective series of 67 patients. Brain. 2007;130(Pt 12):3091–101.
4. Dubosh NM, Bellolio MF, Rabinstein AA, Edlow JA. Sensitivity of early brain computed tomography to exclude aneurysmal subarachnoid hemorrhage: a systematic review and meta-analysis. Stroke. 2016;47(3):750–5.
5. Cortnum S, Sørensen P, Jørgensen J. Determining the sensitivity of computed tomography scanning in early detection of subarachnoid hemorrhage. Neurosurgery. 2010;66(5):900–2; discussion 3.
6. Connolly ES, Rabinstein AA, Carhuapoma JR, Derdeyn CP, Dion J, Higashida RT, et al. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2012;43(6):1711–37.
7. Yang ZL, Ni QQ, Schoepf UJ, De Cecco CN, Lin H, Duguay TM, et al. Small intracranial aneurysms: diagnostic accuracy of CT angiography. Radiology. 2017;285(3):941–52.
8. Mitchell P, Wilkinson ID, Hoggard N, Paley MN, Jellinek DA, Powell T, et al. Detection of subarachnoid haemorrhage with magnetic resonance imaging. J Neurol Neurosurg Psychiatry. 2001;70(2):205–11.
9. Hunt WE, Hess RM. Surgical risk as related to time of intervention in the repair of intracranial aneurysms. J Neurosurg. 1968;28(1):14–20.
10. Report of world federation of neurological surgeons committee on a universal subarachnoid hemorrhage grading scale. J Neurosurg. 1988;68(6):985–6.
11. Marupudi NI, Mittal S. Diagnosis and management of hyponatremia in patients with aneurysmal subarachnoid hemorrhage. J Clin Med. 2015;4(4):756–67.
12. Lin CL, Dumont AS, Lieu AS, Yen CP, Hwang SL, Kwan AL, et al. Characterization of perioperative seizures and epilepsy following aneurysmal subarachnoid hemorrhage. J Neurosurg. 2003;99(6):978–85.
13. Choi KS, Chun HJ, Yi HJ, Ko Y, Kim YS, Kim JM. Seizures and epilepsy following aneurysmal subarachnoid hemorrhage : incidence and risk factors. J Korean Neurosurg Soc. 2009;46(2):93–8.
14. Dorhout Mees SM, Rinkel GJ, Feigin VL, Algra A, van den Bergh WM, Vermeulen M, et al. Calcium antagonists for aneurysmal subarachnoid haemorrhage. Cochrane Database Syst Rev. 2007;(3):CD000277.
15. Kassell NF, Torner JC, Haley EC, Jane JA, Adams HP, Kongable GL. The International Cooperative Study on the timing of aneurysm surgery. Part 1: overall management results. J Neurosurg. 1990;73(1):18–36.
16. Molyneux AJ, Kerr RS, Yu LM, Clarke M, Sneade M, Yarnold JA, et al. International subarachnoid aneurysm trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomised comparison of effects on survival, dependency, seizures, rebleeding, subgroups, and aneurysm occlusion. Lancet. 2005;366(9488):809–17.
17. McDougall CG, Spetzler RF, Zabramski JM, Partovi S, Hills NK, Nakaji P, et al. The barrow ruptured aneurysm trial. J Neurosurg. 2012;116(1):135–44.
18. Vermeij FH, Hasan D, Vermeulen M, Tanghe HL, van Gijn J. Predictive factors for deterioration from hydrocephalus after subarachnoid hemorrhage. Neurology. 1994;44(10):1851–5.
19. Dorsch NW, King MT. A review of cerebral vasospasm in aneurysmal subarachnoid haemorrhage. Part I: incidence and effects. J Clin Neurosci. 1994;1(1):19–26.
20. Claassen J, Bernardini GL, Kreiter K, Bates J, Du YE, Copeland D, et al. Effect of cisternal and ventricular blood on risk of delayed cerebral ischemia after subarachnoid hemorrhage: the Fisher scale revisited. Stroke. 2001;32(9):2012–20.
21. Lawton MT, Vates GE. Subarachnoid hemorrhage. N Engl J Med. 2017;377(3):257–66.
22. Dankbaar JW, de Rooij NK, Velthuis BK, Frijns CJ, Rinkel GJ, van der Schaaf IC. Diagnosing delayed cerebral ischemia with different CT modalities in patients with subarachnoid hemorrhage with clinical deterioration. Stroke. 2009;40(11):3493–8.

23. Jun P, Ko NU, English JD, Dowd CF, Halbach VV, Higashida RT, et al. Endovascular treatment of medically refractory cerebral vasospasm following aneurysmal subarachnoid hemorrhage. AJNR Am J Neuroradiol. 2010;31(10):1911–6.
24. Klopfenstein JD, Kim LJ, Feiz-Erfan I, Hott JS, Goslar P, Zabramski JM, et al. Comparison of rapid and gradual weaning from external ventricular drainage in patients with aneurysmal subarachnoid hemorrhage: a prospective randomized trial. J Neurosurg. 2004;100(2):225–9.
25. de Oliveira JG, Beck J, Setzer M, Gerlach R, Vatter H, Seifert V, et al. Risk of shunt-dependent hydrocephalus after occlusion of ruptured intracranial aneurysms by surgical clipping or endovascular coiling: a single-institution series and meta-analysis. Neurosurgery. 2007;61(5):924–33; discussion 33–4.