Chapter 4
Aneurysmal Subarachnoid Hemorrhage

Jessica L. White and Charles Matouk

4.1 Introduction

Subarachnoid hemorrhage (SAH) accounts for approximately 10% of all strokes, the majority of which are caused by ruptured aneurysms. Other etiologies of SAH include trauma, ruptured vascular malformations, head trauma, use of blood thinners, and primary central nervous system vasculitis/reversible cerebral vasoconstriction syndrome spectrum disorders. Aneurysmal SAH is a devastating disease with 12–15% mortality before hospital admission and overall mortality approaching 50%. Many of those surviving aneurysmal rupture remain functionally dependent and suffer long-term cognitive impairment [1]. The subarachnoid space exists between the arachnoid and pia mater adherent to the surface of the brain and contains cerebrospinal fluid. When this space is filled with blood, the brain can suffer from significant dysfunction secondary to inflammatory cascades, cerebral edema, and hydrocephalus.

J.L. White, PA-C • C. Matouk, MD (✉)
Yale University, New Haven, CT, USA
e-mail: Jessica.white@yale.edu; Charles.matouk@yale.edu

© Springer International Publishing AG 2018

J.L. White, K.N. Sheth (eds.), *Neurocritical Care for the Advanced Practice Clinician*, DOI 10.1007/978-3-319-48669-7_4

Aneurysms are common, with prevalence in the adult population of 1–5% [2]. Historically, the most common presentation of aneurysms has been SAH. However, aneurysms are increasingly discovered incidentally during workup of other conditions, such as headache or head trauma. Risk factors for aneurysm development include hypertension, smoking, sympathomimetic substance abuse, female sex, family history of cerebral aneurysms, and certain genetic disorders, including polycystic kidney disease, Marfan syndrome, and Ehlers-Danlos syndrome (type IV) [1]. Risk factors for aneurysm rupture include aneurysm-specific factors (size, location, morphology) and patient-specific factors (age, female sex, smoking, hypertension, prior SAH) [2]. Aneurysm formation typically occurs at vessel branch points, most commonly the anterior communicating artery (30%), posterior communicating artery (25%), middle cerebral artery bifurcation (20%), and, less commonly, the internal carotid terminus and basilar tip [2].

4.2 Case Example

A 61-year-old woman with past medical history of hypertension presents to the hospital complaining of severe, sudden-onset headache, nausea and vomiting, and photophobia. On examination she is somnolent and complains of an excruciating headache. She reports that this is the "worst headache of her life." While in the emergency department she becomes increasingly lethargic and then frankly unresponsive. CT head demonstrates starburst pattern subarachnoid hemorrhage and mild hydrocephalus (see Fig. 4.1, image a). She is intubated for airway protection, sedated, and her blood pressure controlled with IV nicardipine. CT angiogram demonstrates a small anterior communicating artery aneurysm (image b). A ventriculostomy is placed for treatment of hydrocephalus and intracranial pressure monitoring. Her aneurysm is successfully treated by endovascular coiling (images c and d). The following day she is alert, able to follow simple commands, and is successfully extubated. On post-SAH day 4

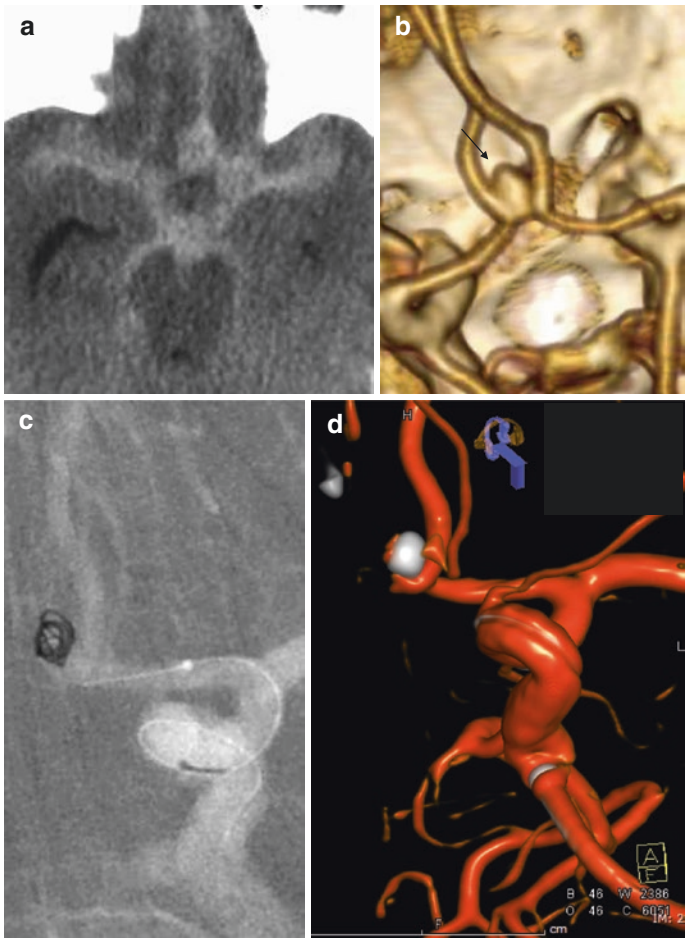

Fig. 4.1 This 61-year-old woman presented with a sudden-onset, severe headache. (**a**) A CT head (axial) demonstrates a starburst pattern of subarachnoid hemorrhage. (**b**) A 3D reconstruction of a CTA demonstrates a small anterior communicating artery (AComA) aneurysm (*arrow*). (**c**) A road map of a diagnostic cerebral angiogram (left internal carotid artery (ICA) injection, oblique view) during endovascular coiling of the ruptured AComA aneurysm. (**d**) A 3D (dual volume) reconstruction of a spin angiogram (left ICA injection, oblique view) demonstrates complete exclusion of the ruptured AComA aneurysm

she becomes confused with new lower extremity weakness. A repeat CT head confirms no interval rebleeding or worsening hydrocephalus. Transcranial Dopplers show elevated velocities in the anterior cerebral arteries, bilaterally. She is treated initially with blood pressure augmentation but fails to improve clinically. She is taken to conventional catheter angiography for intra-arterial administration of verapamil to the affected vascular territories. Induced hypertension is continued post-procedurally with an improvement in her clinical examination.

4.3 Initial Evaluation

4.3.1 Presentation

The most common presentation of aneurysmal SAH is sudden "thunderclap headache" or "worst headache of life." A smaller proportion, 10–15%, present in a comatose state. Additional exam findings may include nuchal rigidity, isolated cranial nerve palsy, and focal neurologic deficit [3]. Patients often report an antecedent, less severe headache syndrome referred to as a sentinel (or warning) headache. Several scales grade the severity of SAH. The Hunt and Hess scale (Table 4.1) is a clini-

Table 4.1 Hunt and Hess Scale

Grade	Neurologic symptoms
1	Asymptomatic, mild headache
2	Moderate-to-severe headache, no neurologic deficit other than cranial nerve palsy
3	Drowsy, confused, mild focal neurologic deficit
4	Stupor, moderate-to-severe hemiparesis
5	Coma, decerebrate posturing

Data from: Hunt [4]

Table 4.2 The World Federation of Neurological Surgeons Subarachnoid Grade

Grade	GCS	Motor deficit
1	15	Absent
2	13–14	Absent
3	13–14	Present
4	7–12	Present or absent
5	3–6	Present or absent

Data from: (1988). "Report of World Federation of Neurological Surgeons Committee on a Universal Subarachnoid Hemorrhage Grading Scale." *Journal of neurosurgery 68*(6): 985–986

cal scale and one of the most widely used. It assigns a grade based on severity of clinical symptoms. The World Federation of Neurological Surgeons Subarachnoid Grade (Table 4.2) is based on the Glasgow Coma Scale with special emphasis on the presence of a motor deficit. While these scales are helpful for quantifying the severity of SAH and facilitating communication, their utility in predicting patient outcome is debated.

4.3.2 Diagnostics

Non-contrast CT head is the recommended initial study for evaluation of suspected SAH with a sensitivity of 98% within 24 h of symptom onset. Classically, SAH appears as a starburst pattern of hemorrhage filling the basal cisterns [1, 2]. Intraventricular hemorrhage and parenchymal hematomas can be present as well, depending on the location and severity of aneurysm that has ruptured. Several grading scales based on CT hemorrhage pattern have been validated to predict the occurrence of symptomatic cerebral vasospasm, most notably the Fisher Scale (Table 4.3) and modified Fisher Scale (Table 4.4) [5, 6].

In cases where CT is negative for hemorrhage, but clinical suspicion for aneurysmal rupture remains high, a lumbar

Table 4.3 Fisher Scale

Grade	Blood on CT scan	% with vasospasm
1	No SAH identified	21
2	Diffuse or focal thin (<1 mm) SAH	25
3	Localized or thick (>1 mm) SAH, ± ICH or IVH	37
4	No SAH, + ICH or IVH	31

Data from: Fisher [5]

Table 4.4 Modified Fisher Scale

Grade	Blood on CT scan	% with vasospasm
1	Thin (<1 mm) SAH, − IVH	24
2	Thin (<1 mm) SAH, + IVH	33
3	Thick (>1 mm) SAH, − IVH	33
4	Thick (>1 mm) SAH, + IVH	40

Data from: Frontera [6]

puncture should be performed to evaluate the cerebrospinal fluid for presence of xanthochromia. Xanthochromia is the yellowish appearance of CSF due to the presence of bilirubin, produced by the metabolism of the heme groups released by red blood cells circulating in the CSF after an aneurysmal rupture. The presence of xanthochromia is over 99% sensitive for SAH and persists for several weeks after initial aneurysm rupture [3].

Once the diagnosis of SAH is established, the cerebral vasculature must be evaluated for a causative lesion. Digital subtraction angiography (DSA), or conventional catheter angiography, is the gold standard of vascular evaluation, although most institutions now utilize noninvasive imaging as a first-line diagnostic strategy. CT angiography is the modality of choice, with excellent sensitivity and specificity for aneurysms >3 mm in size [7, 8]. MR angiography is a reasonable alternative for patients with contraindications to iodinated contrast, e.g., renal impairment [1].

4.4 Interventions and Management

4.4.1 Prevention of Rebleeding

The focus of early management in SAH is to prevent aneurysm rerupture, a clinical scenario that portends a poorer prognosis. Patients are at highest risk for rebleeding within the first hours after aneurysm rupture, so rapid stabilization and treatment are paramount [9]. While aneurysm repair should be performed as soon as possible, several medical strategies can minimize the risk of rebleeding prior to repair.

Blood pressure should be closely monitored from the time of diagnosis. A treatment parameter of systolic blood pressure <160 mmHg is generally accepted as a target for management [1, 10]. Hypotension should be avoided as this can cause a precipitous drop in cerebral perfusion resulting in secondary neurological injury. While pulsed IV medications are helpful for acute stabilization, titratable infusions offer more rapid and sustained effect. Nicardipine (typical infusion rate 5–15 mg/h), a calcium channel blocker, has shown to be superior in achieving systolic blood pressure control than labetalol [11].

Antifibrinolytic medications (tranexamic acid and aminocaproic acid) are sometimes administered in the setting of delayed aneurysm repair to help stabilize fibrin formation in the ruptured aneurysm. Caution must be employed when considering these medications as they can precipitate clot formation and are contraindicated for patients with known coronary artery disease, peripheral vascular disease, and hypercoagulable states. Prolonged administration (>72 h) has the added concern of contributing to delayed cerebral ischemia [12]. When used in appropriate patients for a short duration, antifibrinolytic medications are recommended to reduce the risk of aneurysm rebleeding when aneurysm repair is going to be delayed [1, 10, 12].

Seizures, both convulsive and nonconvulsive, commonly occur after SAH. While most seizures occur at the onset of

hemorrhage, seizures are also associated with rebleeding, and patients remain at risk for seizure development throughout the disease course [13]. Patients with a seizure at presentation are placed on anticonvulsant therapy and should be monitored for further seizures. Prophylactic anticonvulsant medications are usually utilized in all patients until the culprit aneurysm is secured. This intervention is driven by the concern that uncontrolled seizure activity may precipitate rebleeding [10].

4.4.2 Aneurysm Treatment

Repair of ruptured aneurysms can be accomplished by surgical clipping or endovascular techniques, typically coiling, although other strategies are now emerging. The decision to clip or coil an aneurysm is highly individualized and is based on multiple factors including available expertise, aneurysm characteristics, and patient characteristics.

Surgical clipping via craniotomy is the tried-and-true approach to securing an aneurysm and results in its complete exclusion from the circulation. It is a durable strategy with a very low aneurysm recurrence rate on long-term follow-up. A major drawback, however, is its invasiveness.

Alternatively, coiling is a minimally invasive, endovascular approach that refers to the packing of an aneurysm with high-tech metallic thread deployed under fluoroscopic guidance. At Yale University/Yale-New Haven Hospital, a coil-first approach is recommended for most ruptured aneurysms. This is supported by the results of two randomized controlled trials, International Subarachnoid Hemorrhage Trial (ISAT) and Barrow Ruptured Aneurysm Trial (BRAT). Both studies showed improved clinical outcomes after endovascular coiling [14, 15].

More recently, a new endovascular device called flow-diverting stent became available for aneurysm repair. Currently, the only flow-diverting stents available for use in the United States are the Pipeline (and Pipeline Flex) Embolization Devices (Medtronic Neurovascular). These low-porosity stents are deployed in the par-

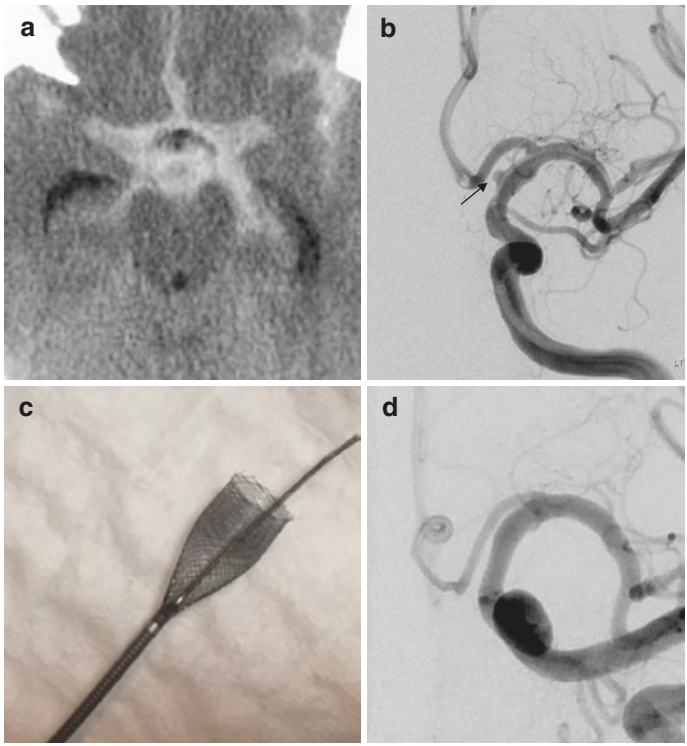

Fig. 4.2 This 51-year-old man presented with a sudden-onset, severe headache. (**a**) A CT head (axial) demonstrates a starburst pattern of subarachnoid hemorrhage. (**b**) A diagnostic cerebral angiogram (DCA, left internal carotid artery (ICA) injection, AP view) demonstrates a blister-like aneurysm of the supraclinoid ICA (*arrow*). (**c**) Photograph of a partially deployed (ex vivo) Pipeline Embolization Device (PED). A similar PED was deployed in the L ICA covering the neck of the small, ruptured aneurysm. Prior to this planned intervention, he was loaded with aspirin and clopidogrel. (**d**) A 6-month follow-up DCA (left ICA, AP view) confirms complete exclusion of the aneurysm

ent vessel and cover the neck of the aneurysm. In doing so, they promote aneurysm thrombosis and endothelialization across the neck of the aneurysm resulting in parent vessel reconstruction and exclusion of the aneurysm from the circulation (see Fig. 4.2 for

case example of Pipeline Embolization Device). A major disadvantage of these flow-diverting stents is their inherent thrombogenicity that mandate the use of dual antiplatelet therapy after deployment, even in the setting of aneurysmal SAH. Notwithstanding, limited off-label experience with these devices for difficult-to-treat, ruptured aneurysms like blister aneurysms, is overall positive [16].

Regardless of the chosen method, aneurysm treatment should be pursued as early as possible to prevent rebleeding.

4.4.3 Hydrocephalus

Hydrocephalus is a common complication of subarachnoid hemorrhage, resulting from impaired CSF reabsorption in the subarachnoid space and from obstructed cerebrospinal flow through the ventricles when intraventricular hemorrhage is present. Acute hydrocephalus can result in increased intracranial pressure and secondary neurological injury. Particularly in the setting of intraventricular hemorrhage, ventriculostomy is beneficial to both monitor intracranial pressure and to allow for CSF diversion. Some concern exists that significant CSF diversion in the setting of an unsecured ruptured aneurysm can precipitate rebleeding by altering the transmural pressure on the aneurysm wall. However, ventriculostomy placement and conservative CSF drainage have been proven beneficial, even in the setting of untreated ruptured aneurysms [17]. A significant number of patients will require long-term CSF diversion with ventriculoperitoneal shunt placement. This is often the case in the setting of extensive intraventricular hemorrhage and high Fisher Grade [18].

4.4.4 Cerebral Vasospasm and Delayed Cerebral Ischemia

Delayed cerebral ischemia (DCI) secondary to cerebral vasospasm occurs in 20–40% of patients following subarachnoid hemorrhage

and is a significant cause of morbidity and mortality [6, 19]. Though the mechanism is not completely understood, cerebral vasospasm is thought to be the result of inflammatory mediators produced during the degradation of blood products in the subarachnoid space. This inflammation causes spasm of cerebral vessels resulting in decreased cerebral blood flow, impaired regional perfusion, and ischemia [20]. Large vessel vasospasm is seen radiographically as focal or diffuse narrowing of arterial vasculature on CT angiogram or conventional angiogram. A greater degree of arterial vasospasm is more commonly seen in close proximity to the ruptured aneurysm. Though radiographic vasospasm and DCI symptoms often occur concomitantly, their relationship is not linear. Approximately 50% of patients who develop large vessel radiographic vasospasm will not have neurological symptoms of ischemia. Conversely, there are patients who develop symptomatic DCI without corresponding radiographic findings. Multiple factors are thought to influence this relationship, including collateral perfusion anatomy and variations in cellular ischemic tolerance [1].

Classically, vasospasm occurs 3–4 days after initial hemorrhage, peaks in occurrence at 7–10 days, and spontaneously resolves by 21 days [1]. Risk of vasospasm development is higher when patterns of thick subarachnoid hemorrhage and intraventricular hemorrhage are present (Tables 4.3 and 4.4) [21]. DCI occurs when vasospasm leads to decreased cerebral blood flow, decreased perfusion, and ischemia. Symptoms of DCI can include general decline in mental status or focal neurologic deficits corresponding to the affected vascular territory.

Rapid detection of cerebral vasospasm and DCI requires a combination of vigilant attention to fluctuations in neurological exam as well as the use of several monitoring modalities. Transcranial Doppler ultrasound (TCD) is used to trend the velocities of intracranial blood flow to observe for the development of vasospasm. As vessel diameter decreases due to spasm, blood travels through the vessel with increased force, resulting in increased blood velocity. Mean velocity in the MCA of <120 cm/s can be reliably used for ruling out vasospasm, while

Table 4.5 Lindegaard ratio

Lindegaard ratio (MCA/ICA velocity)	
<3	Normal
3–4.5	Mild vasospasm
4.5–6	Moderate vasospasm
>6	Severe vasospasm

Data from: Lindegaard [23]

a velocity ≥200 cm/s is indicative of severe vasospasm [22]. The Lindegaard ratio, defined as the mean velocity in the MCA divided by the mean velocity in the extracranial ICA, is helpful to confirm that these increased MCA velocities are due to vasospasm and not simply hyperemia (Table 4.5). TCD has the benefit of being a bedside, noninvasive modality and having a sensitivity of 0.73 and a specificity of 0.80 for detection of vasospasm in the anterior circulation [20, 22]. Trends in TCD velocities often precede symptomatic vasospasm.

In patients with poor clinical exams, the use of other multimodality monitoring including EEG, near-infrared spectroscopy, cerebral microdialysis, and brain parenchymal oxygen tension monitoring may be helpful in correlating cerebral ischemia with radiographic findings, although definitive supporting evidence to support the routine use of these techniques is still lacking. Please see Chap. 20 for further information on these technologies.

The mainstay of therapy for minimizing the detrimental effects of DCI is maintaining euvolemia and homeostasis. Close attention must be given to volume status, as a hypovolemic state equates to decreased intravascular volume and will result in decreased cerebral perfusion with the development of vasospasm.

Among the many medical therapies that have been evaluated in preventing or minimizing the effects of DCI, nimodipine, an oral calcium channel blocker, is the only medication shown to improve outcome related to subarachnoid hemorrhage. While

rates of radiographic vasospasm are not decreased, patients are shown to have lower rates of symptomatic vasospasm, infarction on imaging, and decreased rates of disability [24]. The standard dosing for nimodipine is 60 mg every 4 h, although this dosing can be adjusted (30 mg every 4 h, 30 mg every 2 h) if needed to avoid hypotension. Treatment is recommended for 21 days following SAH or until the patient is discharged from hospital.

In the setting of neurologic decline and concern for DCI, imaging is typically obtained to correlate exam findings with vasospasm and to rule out other diagnostic possibilities (rebleeding, hydrocephalus). CT angiography is the initial study of choice at most institutions and can be utilized to assess for large vessel vasospasm, although small-vessel spasm is difficult to diagnose. CT perfusion and MR perfusion may be helpful to identify areas of decreased cerebral blood flow in the setting of small-vessel vasospasm. These modalities can be particularly useful to evaluate vasospasm in patients with poor clinical exam [10]. Cerebral angiography is the gold standard for detecting vasospasm and provides the opportunity to treat the patient if the need arises.

Treatment of symptomatic vasospasm involves a combination of blood pressure augmentation and endovascular treatment (Table 4.6). Blood pressure is elevated in a stepwise fashion using a vasopressor (usually phenylephrine or norepinephrine) while the patient is evaluated for improvement in neurologic symptoms [10]. Subsequent increases of 20–30% of baseline

Table 4.6 Treatment approach in symptomatic vasospasm

Ensure homeostasis, euvolemia
Blood pressure augmentation (incremental increases of 20–30% above baseline MAP) using phenylephrine or norepinephrine
Increase CSF diversion by lowering ventriculostomy
Endovascular treatment with intra-arterial vasodilators or cerebral angioplasty

MAP are a generally acceptable paradigm. As mean arterial pressure increases, cerebral blood flow also increases and promotes cerebral perfusion. Boluses of isotonic IV fluid can be given to help augment blood pressure at the time of pressor initiation, but continued high-volume fluid infusion is not recommended for treatment. Caution should be utilized for patients with underlying cardiac disease, and patients should be monitored closely for signs of end-organ damage during blood pressure augmentation therapy. Increasing CSF diversion via ventriculostomy can also help to increase cerebral blood flow in vasospasm by decreasing the volume of CSF and allowing room for blood vessel expansion.

Endovascular treatment for symptomatic vasospasm is pursued when symptoms are not improving with blood pressure augmentation or when circumstances – such as cardiac disease or an unsecured aneurysm – preclude the implementation of this therapeutic approach. The severity and location of vasospasm are best characterized by catheter angiography, so treatment can be targeted to causative vessels. Intra-arterial injections of vasodilators, e.g., verapamil, and balloon angioplasty are examples of endovascular interventions for SAH-associated vasospasm. (see Fig. 4.3 for clinical example of balloon angioplasty treatment for vasospasm). Patients with significant symptomatic vasospasm often require serial endovascular treatments during their course.

Continued therapy is required to maintain cerebral perfusion following endovascular treatment for symptomatic vasospasm. Blood pressure augmentation is typically continued for a few hours and then weaned down in a stepwise fashion, observing for recurrence of neurologic symptoms. Imaging modalities, including TCD and CT angiogram, can be helpful in determining the timing of weaning therapies as improvement in radiographic vasospasm typically corresponds with lower risk of DCI recurrence. Similarly, weaning of CSF via ventriculostomy is completed in a stepwise fashion and is delayed until the risk of DCI is decreased.

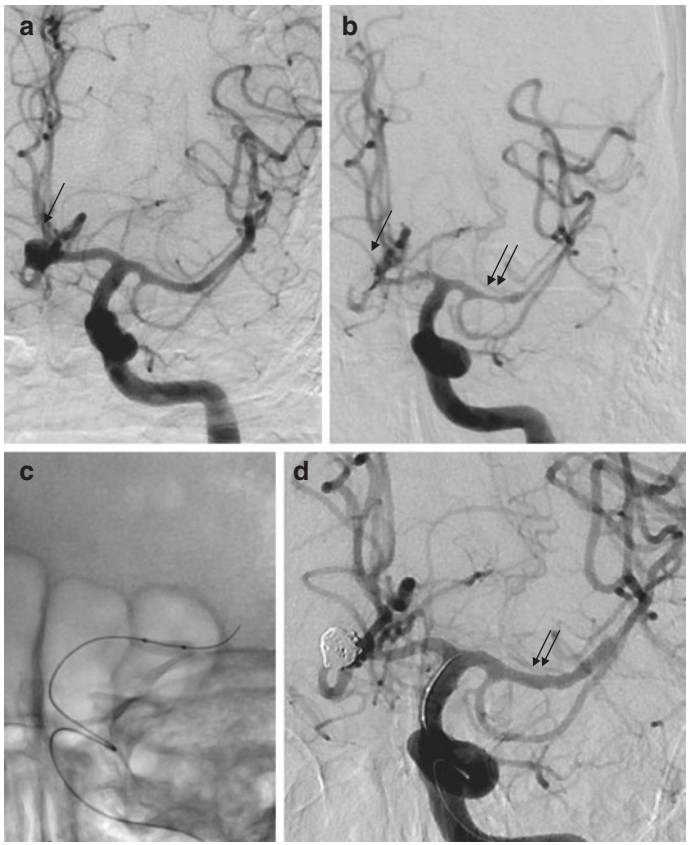

Fig. 4.3 This 53-year-old man presented with a sudden-onset, severe headache. A CT head demonstrated a starburst pattern of subarachnoid hemorrhage. (**a**) A diagnostic cerebral angiogram (DCA, left internal carotid artery (ICA) injection, AP view) demonstrates an AComA aneurysm (*arrow*). He underwent endovascular coiling of the ruptured aneurysm the next day. (**b**) On post-SAH day #5, he developed new-onset R-sided weakness and word-finding difficulty. These did not improve with a trial of induced hypertension. A DCA demonstrates the coiled AComA aneurysm (*arrow*) and interval development of moderate-to-severe vasospasm of the L MCA (*double arrow*). (**c**) Balloon angioplasty of the L MCA is demonstrated with an excellent angiographic result (*double arrow*) (**d**). His R-sided weakness and aphasia resolved in short course

4.4.5 Cerebral Salt Wasting

Cerebral salt wasting (CSW) is a condition frequently seen in aneurysmal subarachnoid hemorrhage and commonly occurs with the onset of cerebral vasospasm. CSW is characterized by a markedly elevated urine output (often >200 cc/h) accompanied by rapidly evolving hyponatremia. Laboratory studies reveal serum sodium <135 mmol/L, low plasma osmolality, and inappropriately elevated urine sodium and osmolality. It is necessary to distinguish CSW from SIADH as aggressive volume repletion is essential in CSW and may worsen symptoms in SIADH. Fludrocortisone and aggressive repletion of volume are utilized to correct hyponatremia and retain stable sodium levels (see Chap. 23 for further information on diagnosis and management of hyponatremia). If untreated, cerebral salt wasting can result in rapid depletion of the intravascular volume and subsequent development or worsening of cerebral vasospasm. Close monitoring of urine output and overall volume status is recommended through the time frame of cerebral vasospasm.

4.4.6 Cardiopulmonary Complications

Patients with SAH commonly develop cardiopulmonary complications, including neurogenic stunned myocardium (also referred to as stress cardiomyopathy or takotsubo cardiomyopathy) and neurogenic pulmonary edema. The primary mechanism contributing to the development of these conditions is thought to be the catecholamine surge that occurs at the time of aneurysm rupture and raised intracranial pressure.

Neurogenic stunned myocardium can be present at patient presentation but can also develop through the critical time frame of peak vasospasm occurrence. This disease process is a distinct entity from other cardiomyopathies in that incidence is not

associated with prior cardiac history or coronary artery disease. Identified risk factors include higher Hunt and Hess grade, history of smoking, lack of history of hypertension, and older age [25]. Initial presenting features include acute hypotension and hemodynamic instability, EKG changes (diffuse ST elevation or T-wave inversions), and troponin elevation. Echocardiogram will commonly demonstrate findings including reduced ejection fraction, global hypokinesis, dyskinesis of the apical or mid-ventricular segments, and diastolic dysfunction [26]. Management of neurogenic stunned myocardium is primarily supportive, utilizing vasopressors and balloon pumps in severe cases to maintain hemodynamic stability and cerebral perfusion. Spontaneous recovery of cardiac function is usually observed in days to weeks following onset [26].

Neurogenic pulmonary edema is similarly seen at presentation and can develop along the same time line as stunned myocardium. Pulmonary edema rapidly develops as a result of capillary dilation leading to interstitial and alveolar edema during initial catecholamine surge [27]. Treatment involves supportive management and maintenance of strict euvolemia (avoiding hypervolemia). The use of aggressive diuresis for management of pulmonary edema should be avoided during the peak time frame of cerebral vasospasm to avoid depleting intravascular volume and contributing to delayed cerebral ischemia.

4.5 Perimesencephalic Subarachnoid Hemorrhage

A subpopulation of patients presenting with subarachnoid hemorrhage have characteristic radiographic findings of aneurysmal SAH, but have no causative lesion identified, and have a benign clinical course. These patients typically present with symptoms of headache, meningismus, and nausea. The onset more commonly

occurs during physical exertion [28]. The hemorrhage pattern is focused centrally around the brainstem in the perimesencephalic cisterns without distal extension into the Sylvian and interhemispheric fissures. Vascular imaging (CT angiography or MR angiography) is recommended to exclude an underlying vascular lesion, but diagnostic catheter angiography can by deferred if clinical suspicion for aneurysmal rupture is low. Although perimesencephalic hemorrhage patients can develop hyponatremia and other systemic complications associated with SAH, their course is generally benign. Rebleeding and development of cerebral vasospasm are rare [29]. The etiology of perimesencephalic hemorrhage is debated but is likely to be venous in origin.

Summary Points

- Aneurysmal subarachnoid hemorrhage is a complex disease with significant associated morbidity and mortality.
- Early identification and treatment of ruptured vascular lesion are essential to prevent rebleeding and optimize neurologic outcome.
- Cerebral vasospasm is a major complication following aneurysmal subarachnoid hemorrhage and requires rapid diagnosis and treatment to prevent delayed cerebral ischemia.
- Aneurysmal subarachnoid hemorrhage is associated with multiple systemic complications.

References

1. Connolly ES. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke (1970). 2012;43(6):1711–37.
2. Brisman JL. Cerebral aneurysms. N Engl J Med. 2006;355(9):928–39.

3. Wijdicks EF. Subarachnoid hemorrhage: neurointensive care and aneurysm repair. Mayo Clin Proc. 2005;80(4):550–9.
4. Hunt WE. Surgical risk as related to time of intervention in the repair of intracranial aneurysms. J Neurosurg. 1968;28(1):14–20.
5. Fisher CM. Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by computerized tomographic scanning. Neurosurgery. 1980;6(1):1–9.
6. Frontera JA. Prediction of symptomatic vasospasm after subarachnoid hemorrhage: the modified fisher scale. Neurosurgery. 2006;59(1):21–7.
7. Chappell ET. Comparison of computed tomographic angiography with digital subtraction angiography in the diagnosis of cerebral aneurysms: a meta-analysis. Neurosurgery. 2003;52(3):624–31; discussion 630–621.
8. Westerlaan HE. Intracranial aneurysms in patients with subarachnoid hemorrhage: CT angiography as a primary examination tool for diagnosis – systematic review and meta-analysis. Radiology. 2011;258(1): 134–45.
9. Ohkuma H. Incidence and significance of early aneurysmal rebleeding before neurosurgical or neurological management. Stroke (1970). 2001;32(5):1176–80.
10. Diringer MN. Critical care management of patients following aneurysmal subarachnoid hemorrhage: recommendations from the Neurocritical Care Society's Multidisciplinary Consensus Conference. Neurocrit Care. 2011;15(2):211–40.
11. Liu-Deryke X. A comparison of nicardipine and labetalol for acute hypertension management following stroke. Neurocrit Care. 2008; 9(2):167–76.
12. Hillman J. Immediate administration of tranexamic acid and reduced incidence of early rebleeding after aneurysmal subarachnoid hemorrhage: a prospective randomized study. J Neurosurg. 2002;97(4): 771–8.
13. Gilmore E. Seizures and CNS hemorrhage: spontaneous intracerebral and aneurysmal subarachnoid hemorrhage. Neurologist (Baltimore, Md). 2010;16(3):165–75.
14. Molyneux AJ. The durability of endovascular coiling versus neurosurgical clipping of ruptured cerebral aneurysms: 18 year follow-up of the UK cohort of the International Subarachnoid Aneurysm Trial (ISAT). Lancet (Br Ed). 2015;385(9969):691–7.
15. Spetzler RF. The barrow ruptured aneurysm trial: 6-year results. J Neurosurg. 2015;123(3):609–17.
16. Martin AR. The pipeline flow-diverting stent for exclusion of ruptured intracranial aneurysms with difficult morphologies. Neurosurgery. 2012;70(1 suppl operative):ons21–8.

17. McIver JI. Preoperative ventriculostomy and rebleeding after aneurysmal subarachnoid hemorrhage. J Neurosurg. 2002;97(5):1042–4.
18. Rincon F. Predictors of long-term shunt-dependent hydrocephalus after aneurysmal subarachnoid hemorrhage. Clinical article. J Neurosurg. 2010;113(4):774–80.
19. Solenski NJ. Medical complications of aneurysmal subarachnoid hemorrhage: a report of the multicenter, cooperative aneurysm study. Participants of the multicenter cooperative aneurysm study. Crit Care Med. 1995;23(6):1007–17.
20. Suarez JI. Symptomatic vasospasm diagnosis after subarachnoid hemorrhage: evaluation of transcranial Doppler ultrasound and cerebral angiography as related to compromised vascular distribution. Crit Care Med. 2002;30(6):1348–55.
21. Rabinstein AA. Patterns of cerebral infarction in aneurysmal subarachnoid hemorrhage. Stroke (1970). 2005;36(5):992–7.
22. Vora Y. Role of transcranial Doppler monitoring in the diagnosis of cerebral vasospasm after subarachnoid hemorrhage. Neurosurgery. 1999;44(6):1237–47; discussion 1247–1238.
23. Lindegaard KF. Cerebral vasospasm after subarachnoid haemorrhage investigated by means of transcranial Doppler ultrasound. Acta Neurochir Suppl. 1988;42:81–4.
24. Feigin VL. Calcium antagonists in patients with aneurysmal subarachnoid hemorrhage: a systematic review. Neurology. 1998;50(4):876–83.
25. Malik AN. Neurogenic stress cardiomyopathy after aneurysmal subarachnoid hemorrhage. World Neurosurg. 2015;83(6):880–5.
26. Murthy SB. Neurogenic stunned myocardium following acute subarachnoid hemorrhage: pathophysiology and practical considerations. J Intensive Care Med. 2015;30(6):318–25.
27. Muroi C. Neurogenic pulmonary edema in patients with subarachnoid hemorrhage. J Neurosurg Anesthesiol. 2008;20(3):188–92.
28. Matsuyama T. Perimesencephalic nonaneurysmal subarachnoid hemorrhage caused by physical exertion. Neurol Med Chir. 2006;46(6):277–81. discussion 281–272
29. Rinkel GJ. The clinical course of perimesencephalic nonaneurysmal subarachnoid hemorrhage. Ann Neurol. 1991;29(5):463–8.