Case Studies in Neurocritical Care

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KEYWORDS

- Acute stroke Subdural hemorrhage Seizures Cardiac arrest
- Subarachnoid hemorrhage

KEY POINTS

- Timely recognition and adequate management of medical complications are essential to maximize the chances of recovery after a severe acute ischemic stroke.
- Autoimmune encephalitis can present with severe agitation and may be confused with a psychiatric disease. Search for seizures in these cases is necessary.
- Seizures are a common complication of subdural hemorrhage and they can be refractory.
- Aneurysmal subarachnoid hemorrhage can produce various neurologic and systemic complications, including hydrocephalus, delayed cerebral ischemia, central fever, and polyuria. Optimal management requires clinicians to be knowledgeable of these complications and remain highly vigilant to recognize them early.
- Seizures after cardiac arrest can be treatable, but they often portend poor prognosis. Yet, prognostication should be deferred until the seizures are controlled.

Neurocritical care is a specialty straddling fields of expertise. Its practitioners need to know neurology, internal medicine, and critical care. Critically ill neurologic and neurosurgical patients present unique challenges that demand special training and experience. The neurologic management of these patients requires in-depth knowledge of acute neurology while diagnosis and treatment of systemic complications necessitate critical care training. It is in the interface of these specialties, however, where the niche of the neurointensivist lies. As exemplified by the cases discussed later, the patients treated by a neurointensivist offer challenges that are not encountered in other fields.

This article discusses a few illustrative examples of patients the authors have seen over a single week in a neuroscience ICU. They are only a small sample of the variety of

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conditions treated. They are selected because they serve well to highlight some useful practical messages.

CASE 1: COMPLICATIONS AFTER A STROKE

A 76-year-old woman suddenly developed left-sided weakness leading to a fall while at home with her husband. She had history of hypertension, impaired fasting blood glucose, and obesity. She had also been complaining of irregular palpitations and progressive exertional dyspnea for the past month but had never been diagnosed with atrial fibrillation. She arrived to the emergency department 60 minutes after symptom onset. On examination she had a systolic blood pressure ranging from 180 mm Hg to 200 mm Hg. ECG showed atrial fibrillation with accelerated ventricular rate (120–140 beats per minute). Neurologic examination demonstrated right gaze deviation; left visual field deficit; severe dysarthria; left hemiplegia involving face, arm, and leg; and left-sided neglect. Initial National Institutes of Health Stroke Scale (NIHSS) score was 20. Head CT scan showed some early ischemic changes (Alberta Stroke Program Early CT Score 7) (Fig. 1A) and CT angiogram confirmed the suspected occlusion of the right middle cerebral artery. Patient was treated with a total of 40 mg of intravenous (IV) labetalol and a nicardipine infusion (5 mg/h) for management of hypertension and atrial fibrillation. After control of the hypertension to a systolic blood pressure below 180 mm Hg, the patient was treated IV thrombolysis (bolus administered 115 minutes after symptom onset) and the patient was taken to an angiographic suite for endovascular therapy. The procedure was performed under conscious sedation and mechanical thrombectomy using a retrievable stent achieved successful recanalization and reperfusion (thrombolysis in cerebral infarction grade 3) 3 hours after symptom onset (see Fig. 1B, C). NIHSS score after the intervention was 15. She was then transferred to the neuroscience ICU. The sheath used for arterial access was kept in place and it was removed without complications the following morning.

By the following day, the patient had continued to improve (NIHSS score 11) although the repeat head CT scan at 24 hours showed a large infarction with hemorrhagic transformation within the right lateral basal ganglia (see **Fig. 1**D). That evening, the patient started to complain of right flank pain and examination revealed a rapidly expanding hematoma on the right groin (ie, the site of previous catheter access). She rapidly progressed to hemorrhagic shock, requiring 2 units of packed red blood cells and 2 L of crystalloids for emergent resuscitation. Systolic blood pressure reached a nadir of 68 mm Hg but recovered promptly. Prolonged direct pressure to the region was applied and an emergent ultrasound revealed a large femoral artery pseudoaneurysm, which was treated with thrombin injection. CT scan of the abdomen/pelvis did not show any retroperitoneal bleeding. After this hypotensive episode her neurologic deficits worsened (NIHSS score 16), and she developed transient oliguric acute kidney injury.

A repeat CT scan the following day revealed no significant expansion of the hemorrhage or major progression of mass effect. Her neurologic condition began to improve again until the fourth day of her hospitalization, when she developed fever after an episode of aspiration during formal swallowing testing by a dysphagia specialist. Shortly after, she became hypotensive and required fluid resuscitation. She was empirically started on broad-spectrum antibiotic therapy. Repeat groin ultrasound revealed continued persistent occlusion of the pseudoaneurysm. Tracheal secretions grew *Staphylococcus aureus* and chest radiograph disclosed a new left lower lobe consolidation. During her ICU hospitalization she also required infusions of diltiazem and esmolol for control of the rapid ventricular response to her atrial fibrillation and intermittent doses of furosemide for signs of pulmonary edema.

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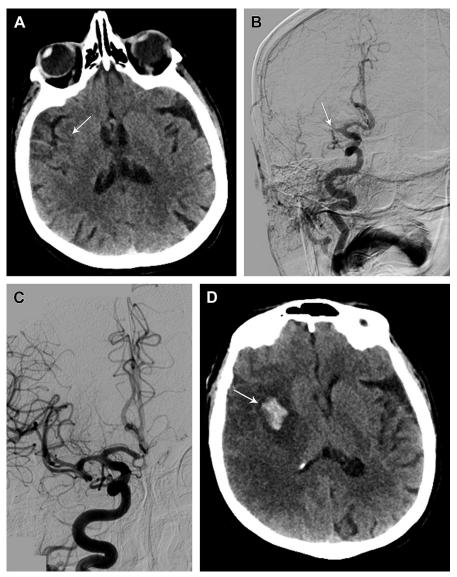


Fig. 1. Case 1. (*A*) CT scan before treatment showing early ischemic changes on the right middle cerebral artery territory, showing loss of insular ribbon and boundaries of the lenticular nucleus (*arrow*). Notice extreme right gaze deviation. (*B*) Catheter angiogram showing the proximal occlusion of the right middle cerebral artery (*arrow*) with poor collateral circulation. (*C*) Catheter angiogram demonstrating full recanalization and reperfusion after mechanical thrombectomy. (*D*) Follow-up CT scan showing a large infarction on the right middle cerebral artery territory with a hemorrhagic conversion in the region of the right basal ganglia (*arrow*).

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Despite all these complications, she had begun to recover well by the time of her discharge to a skilled nursing facility. On outpatient follow-up 15 days later, she was walking 50 feet unassisted and was nearly ready to return home.

Clinical Messages

- Large infarctions can develop despite successful recanalization and reperfusion. In these cases, reperfusion injury, including edema and hemorrhage, can cause complications related to mass effect that should be anticipated.^{1,2}
- The arterial site of catheter access should be closely monitored after any endovascular intervention, but this is particularly important in patients with acute stroke who have received IV thrombolysis. The most common occurrence is an inconsequential groin hematoma that can be limited with local pressure. As illustrated by this case, however, pseudoaneurysm formation may be accompanied by arterial rupture with massive bleeding that may present with hypovolemic shock. Unless the bleeding is promptly recognized and controlled, this complication can be fatal.
- Aspiration pneumonia is a common complication after a severe stroke and can occur despite appropriate assessment for dysphagia and in patients who are not allowed oral diet. The risk of aspiration may be higher when the infarction involves the insular cortex and frontal operculum.³
- Hypotension, even if brief, often causes worsening of neurologic deficits after an acute stroke.⁴ This is possible even days after stroke onset.
- Patients with severe ischemic stroke may develop multiple treatable complications and still recover well if such complications are optimally addressed.

CASE 2: EXTREME AGITATION IN AUTOIMMUNE ENCEPHALITIS

A 23-year-old woman started to exhibit progressive behavioral changes accompanied by speech stuttering and spells of vertigo with nausea. She then developed visual illusions and hallucinations and episodes of left head turning and left hand gripping followed by transient confusion. Thought to have a psychiatric disorder, she was prescribed sertraline after having an electroencephalogram and a head CT scan that were interpreted as normal. Her condition worsened, however, over the following days. She became increasingly agitated and had paranoid ideation. Episodes of left facial twitching became more common but they were never associated with generalized convulsions or unresponsiveness. Finally she presented to another hospital where focal seizures arising from the right frontal region were detected and she was started on antiseizure medications. Cerebrospinal fluid analysis revealed 7 white blood cells/µL, protein of 20 mg/dL, and normal glucose content. Sedimentation rate was 96 mm but other markers of acute systemic inflammation were absent. Brain MRI was read as negative, although it actually revealed subtle changes on the limbic region bilaterally. Abdominal MRI demonstrated an ovarian teratoma that was subsequently removed by laparoscopic surgery. Autoimmune panel in serum and cerebrospinal fluid disclosed the presence of anti-N-methyl-D-aspartate (NMDA) receptor antibodies.

Her seizures were deemed refractory and she was treated with high doses of levetiracetam, lacosamide, phenytoin, and phenobarbital. Because of the seizures and the persistent intractable agitation, she was intubated and placed on a midazolam infusion (up to 30 mg/h) and then propofol and fentanyl were added in increasing doses. For the autoimmune encephalitis she received IV methylprednisolone (1 g/d) for multiple days and a 5-day course of IV immunoglobulin (IVIG). An infusion of rituximab was discontinued because of hypertension. She was then transferred to the authors' hospital for further care on request of the family. Her electroencephalogram revealed delta waves on the right frontal region that were semirhythmic at times but no seizures. The authors switched her to oral prednisone (60 mg/d), stopped phenytoin and phenobarbital, and gradually weaned off midazolam, fentanyl, and propofol. She progressively woke up with intermittent restlessness but no severe agitation. Spells of paroxysmal sympathetic hyperactivity (tachycardia, hypertension, tachypnea, diaphoresis, and fever) were successfully aborted with IV morphine (4 mg) and then made more infrequent with gabapentin (600 mg 3 times daily). Eight days after admission to the ICU she was stable to be safely transferred to the floor.

Clinical Messages

Table 1

- Agitation mimicking psychosis from a primary psychiatric disease is a frequent manifestation of autoimmune encephalitis in its early phases. Conversely, catatonia can also be caused by autoimmune encephalitis. Thus, autoimmune encephalitis should be included in the differential diagnosis of new major behavioral changes, especially in young patients.^{5,6}
- Autoimmune encephalitis is possible in patients with unremarkable or minimally abnormal cerebrospinal fluid and MRI of the brain.⁷ When the clinical suspicion is high, autoantibodies should be tested regardless of the results of these investigations.
- Underlying seizures may be present in patients with severe agitation from autoimmune encephalitis. They can only be recognized by electroencephalography, which often needs to be prolonged to optimize detection.⁸
- The management of autoimmune encephalitis in the ICU can be challenging.⁹ Multiple neurologic and systemic complications can occur during the hospitalization (Table 1). Aggressive sedation (and sometimes neuromuscular paralysis) may be necessary to control agitation, and multiple antiseizure medications may need to be used to abolish the seizures. Paroxysmal sympathetic hyperactivity is another common complication and it typically responds well to morphine as acute abortive treatment and to gabapentin as preventive agent.
- The response to immune therapy is commonly delayed. It is important to remember this to avoid overtreatment. Refractory cases demand increasingly

Clinical manifestations and complications in patients with severe autoimmune encephalitis	
Neurologic	Systemic
Severe agitation Focal deficits Seizures (focal or generalized)/status epilepticus Cognitive impairment Depressed level of consciousness Catatonia Headache	Fever Respiratory failure Sepsis Venous thromboembolism Gastrointestinal hemorrhage Ileus Anemia Hyperglycemia
Severe agitation Focal deficits Seizures (focal or generalized) Cognitive impairment Depressed level of consciousness Paroxysmal sympathetic hyperactivity Raised intracranial pressure	Atelectasis Rhabdomyolysis (related to seizures or extreme agitation) Others related to underlying tumor

aggressive treatment regimens (rituximab and cyclophosphamide). These second-line therapies, however, may be associated with greater risk of side effects and, therefore, it is prudent to delay the use of these medications in patients who remain stable with corticosteroids and IVIG or plasma exchange because a good proportion of these patients start improving over time.

 Searching for an underlying tumor, especially for an ovarian teratoma in young women with anti-NMDA receptor antibody encephalitis, should always be part of the evaluation of autoimmune encephalitis.⁷

CASE 3: SEIZURES AFTER SUBDURAL HEMORRHAGE

A 64-year-old woman on warfarin for atrial fibrillation had been having worsening headaches for 1 week after hitting her head with the door of a car. The day of the admission the headaches had worsened and then she became progressively drowsier. A head CT scan obtained in the emergency department showed an acute subdural hematoma over the right frontal cerebral convexity measuring 1 cm in maximal diameter and causing 5 mm of midline shift. The international normalized ratio was 2.7 and prompt reversal was achieved with prothrombin complex concentrate and IV vitamin K. The patient was then taken to surgery for craniotomy and evacuation of the subdural blood.

The surgery was uncomplicated and postoperative CT scan revealed resolution of the mass effect without parenchymal damage. The patient initially did well, but on the second postoperative day she developed worsening dysarthria and left arm weakness. No clinical seizure-like activity was reported by nurses. Subsequently, she became rapidly drowsier and required intubation for airway protection. Repeat brain imaging revealed no interval changes. An electroencephalogram disclosed frequent right frontal seizures. The patient was already on levetiracetam (750 mg twice daily) for seizure prophylaxis. Additional levetiracetam and loading with valproic acid and phenobarbital (after lack of improvement with phenytoin) eventually controlled the seizures. The patient remained stuporous after seizure control for several more days. The antiepileptic regimen was gradually narrowed back to monotherapy with levetiracetam with persistent seizure control proved by continuous electroencephalography. Eventually, her level of alertness began to improve. As she awakened, examination showed left arm apraxia, which improved over days. Twelve days after intubation she was successfully extubated and transferred to the floor the following day. Her subsequent recovery has been very favorable.

Clinical Messages

- Seizures are prevalent after acute subdural hemorrhage.¹⁰ They can be focal or generalized and clinical or subclinical. Electroencephalography should be obtained in any patient with acute subdural hemorrhage who has unexplained depressed level of consciousness, altered content of consciousness, or new focal deficits. Continuous electroencephalography is advisable in patients with epileptiform discharges during the first 30 minutes of recording and necessary in patients with documented subclinical seizures.
- Seizures related to acute subdural hemorrhage are more common after hematoma evacuation, especially if craniotomy is needed.¹⁰ Epileptiform discharges in the midline head regions predominate and the incidence of seizures correlates with the degree of preoperative midline shift.¹¹ Seizures can also occur, however, before surgery and in patients who do not require an operation.
- After subdural hemorrhage, seizures can be refractory to treatment and may require multiple antiseizure medications or even anesthetic agents before they

can be controlled. During this time, patients may present a concerning neurologic status (prolonged coma or persistent focal deficits without radiologic evidence of parenchymal injury). Yet, after resolution of the seizures and the sedative effects of the drugs used to control them, patients can make a favorable recovery.¹⁰ Keeping this in mind is crucial to avoid premature withdrawal of intensive care measures.

CASE 4: COMPLICATIONS AFTER ANEURYSMAL SUBARACHNOID HEMORRHAGE

A 53-year-old woman developed a thunderclap headache while at work and was taken to the emergency department. She first became agitated and then became progressively less responsive to the point that she required intubation for airway protection (World Federation of Neurosurgical Surgeons [WFNS] grade V). Head CT scan demonstrated a severe subarachnoid hemorrhage (Fig. 2A, B). Her hypertension was reduced to a systolic blood pressure below 160 mm Hg with a combination of hydralazine and nicardipine. She received 1 gram of tranexamic acid in the emergency department. Because of the severity of the hemorrhage, the authors decided to take the patient directly to the angiographic suite for endovascular coiling of the suspected aneurysm. While on the table ready to undergo the procedure, she developed sudden severe hypertension (up to 230/120 mm Hg despite a nicardipine infusion at 7.5 mg/h), a brief episode of ventricular tachycardia, and bilateral extensor posturing. Examination also showed anisocoria with preserved light reactivity. After emergent hemodynamic stabilization, the angiogram was performed and a 7-mm basilar tip aneurysm was successfully coiled. The patient also underwent placement of a ventriculostomy. Repeat CT scan confirmed that she had rebled and developed worsened IV hemorrhage with hydrocephalus (see Fig. 2C, D).

After the intervention, she was treated with mannitol because of increased intracranial pressures. Ventricular drain was kept open at 5 mm Hg. She gradually improved and the authors were able to extubate her on day 4. Fevers that had begun on day 3 became more persistent over subsequent days. Infectious work-up was negative. On day 6 she became more agitated and, at times, drowsy. Transcranial Doppler revealed borderline increased blood flow velocities in the left anterior cerebral artery (112 cm/s) but was otherwise unremarkable. CT angiogram was suspicious for distal bilateral anterior cerebral artery vasospasm and there was slight prolongation of mean transient times on the watershed distribution (between anterior and middle cerebral arteries) on the upper frontal lobes. She was spontaneously hypertensive (systolic blood pressures up to 220 mm Hg and mean arterial pressures up to 140 mm Hg). Despite the fairly unimpressive results of the investigations, the authors attributed the neurologic change to delayed cerebral ischemia and allowed her to remain hypertensive (permissive hypertension). On day 8, her mean arterial pressure briefly dropped below 100 mm Hg and she became stuporous. Her condition improved immediately after a fluid bolus and initiation of a phenylephrine infusion restored the previous level of hypertension. For the following 4 days she remained intermittently on hemodynamic augmentation. Between days 7 and 11 she developed severe polyuria (up to 18 L of urinary output per day), which the authors treated with fluid and sodium replacement but allowing her to be up to 2 L to 3 L negative as long as the blood pressure remained within target. The patient was also receiving fludrocortisone and she did not develop any substantial hyponatremia.

By day 12, her condition was more stable. The level of ventricular drainage was progressively raised and the drain could be removed on day 14. Repeat CT scan **ARTICLE IN PRESS**

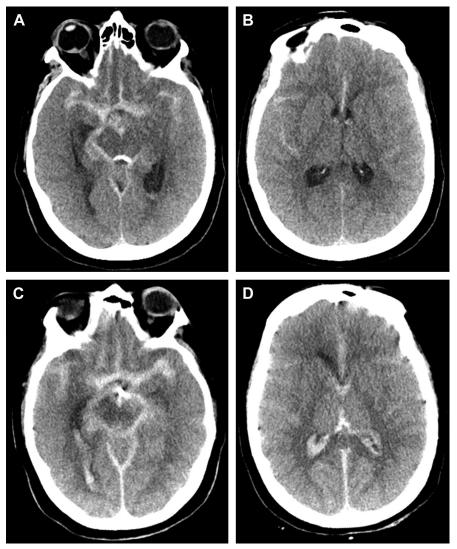


Fig. 2. Case 4. CT scans (A, B) at presentation with aneurysmal subarachnoid hemorrhage and (C, D) after aneurysmal rebleeding.

showed no evidence of infarctions. The patient is now undergoing a short course of rehabilitation. The authors expect her to make a full recovery.

Clinical Messages

- Aneurysmal subarachnoid hemorrhage is characteristically associated with an increased risk of multiple neurologic and systemic complications, both early after aneurysm rupture and over the following days (Table 2).^{12,13} Many of these complications are predictable and being prepared for their occurrence is indispensable to optimize the chances of good functional recovery.^{14,15}
- During the first few hours, the 2 main cerebral complications are aneurysm rebleeding and hydrocephalus (obstructive or communicating).¹³ Rebleeding

Table 2 Neurologic and systemic complications in patients with aneurysmal subarachnoid hemorrhage	
Complication	Therapeutic Measures
Before aneurysm treatment	
Hydrocephalus	Place external ventricular or lumbar drain
Aneurysmal rebleeding	Early aneurysm repair Tranexamic acid 1 g IV every 6 h not exceeding 48–72 h
Increased intracranial pressure	Elevate head of bed to 30° Control fever and agitation Short-term hyperventilation Osmotherapy (20% mannitol, hypertonic saline) Surgical evacuation/decompression for lesions with mass effect and clinical deterioration
Stress-induced cardiomyopathy (neurogenic stunned myocardium, apical ballooning syndrome, takotsubo cardiomyopathy)	Consider β-blockers if no cardiogenic shock If cardiogenic shock, adrenergic agents are not contraindicated, but placement of an intra-aortic balloon pump may be advisable in the most severe cases Diuretics if pulmonary edema
Neurogenic pulmonary edema	Increase level of positive end-expiratory pressure Add diuretics if necessary
After aneurysm treatment	
Delayed cerebral ischemia (most commonly caused by symptomatic vasospasm)	Nimodipine, 60 mg every 4 h for up to 21 d (for prevention) Maintain euvolemia with crystalloids or colloids If symptomatic, induce hypertension with phenylephrine, norepinephrine, or dopamine If refractory, proceed to endovascular treatment (transluminal angioplasty or intra-arterial vasodilators)
Hyponatremia (most often combined with polyruria)	Strict avoidance of free water Hydration with saline solutions (1.5% or 3%) Fludrocortisone acetate up to 0.2 mg twice daily
Hyperglycemia	Use insulin to maintain normoglycemia (100–180 mg/dL)
Fever	Rule out infection Maintain normothermia with acetaminophen, cooling blankets, or surface cooling devices

carries high morbidity and mortality; its risk can be reduced by the early administration of an antifibrinolytic agent, such as tranexamic acid.¹⁶ It is typically manifested with sudden hypertension, tachycardia (sometimes followed by bradycardia), and posturing (often confused with a seizure) and can be confirmed by the presence of new blood on the CT scan. Hydrocephalus has a less dramatic presentation. Patients become less responsive, bradycardic, and mildly hypertensive; downgaze deviation may be noticed. Ventriculomegaly on brain imaging may be more subtle than expected in patients with global brain swelling.¹⁷

 During the acute phase (first 12–24 hours), cardiopulmonary complications may also be observed, more commonly in patients with poor-grade hemorrhages (WFNS score IV–V). ECG changes are frequent and include repolarization changes (widespread giant T-wave inversions known as cerebral T waves are most characteristic but certainly not the most common), supraventricular and ventricular arrhythmias, and prolongation of the QTc interval.¹⁸ The ECG changes caused by aneurysmal subarachnoid hemorrhage can also mimic an acute ST-elevation myocardial infarction.¹⁹ Neurogenic cardiopulmonary injury can produce pulmonary edema (neurogenic, cardiogenic, or mixed) and other signs of congestive heart failure in patients with preexistent cardiac disease and in those who develop stress-induced cardiomyopathy.^{20,21} Stress-induced cardiomyopathy is more common in postmenopausal women and its most typical echocardiographic pattern is apical hypokinesis or akinesis (apical ballooning syndrome or takotsubo cardiomyopathy).^{21,22}

- Delayed cerebral ischemia is the most severe neurologic complication occurring days after aneurysm rupture (maximal risk between days 4 and 10).^{13,23} It is primarily caused by vasospasm.^{23,24} Vasospasm is mainly caused, however, by endothelial dysfunction that starts affecting the microcirculation.²⁵ Consequently, symptoms of delayed cerebral ischemia may develop in the absence of visible luminal narrowing in the larger intracranial arteries.²⁶ For this reason, delayed cerebral ischemia remains a clinical diagnosis.²⁷ CT perfusion (combined with CT angiogram) may be useful in some instances to confirm the diagnosis,²⁸ but the sensitivity of this modality is not perfect.
- The medical treatment of delayed cerebral ischemia consists of hemodynamic augmentation.^{12–15} Spontaneous hypertension should not be treated and vaso-pressors should be added when delayed cerebral ischemia is diagnosed or suspected. Endovascular therapy (balloon angioplasty for vasospasm involving larger arterial segments and selective intra-arterial infusion of calcium channel blockers for more distal vasospasm) should be used as a rescue treatment in patients who remain symptomatic despite hemodynamic augmentation or cannot tolerate vasopressors.^{12–15}
- Polyuria is common in patients with aneurysmal subarachnoid hemorrhage. Its most common pathophysiology is cerebral salt wasting syndrome (ie, excessive secretion of natriuretic peptides leading to increased urinary sodium losses with concomitant dragging of water resulting in intravascular volume depletion).²⁹ Inappropriate secretion of antidiuretic hormone may contribute. These 2 mechanisms lead to hyponatremia.³⁰ Treatment consists of fluid and sodium replacement.²⁹ Fludrocortisone can also be beneficial to ameliorate the negative sodium balance.²⁹ Diabetes insipidus is uncommon in aneurysmal subarachnoid hemorrhage. When it occurs, it tends to appear early and in patients with the most severe forms of hemorrhage. Finally, polyuria may be perpetuated by excessive administration of fluids as it may have occurred in this case. Although often difficult to avoid because of concerns that the patient may develop ischemia if volume contraction is not avoided, excessive fluid administration can be problematic. In the authors' experience, positive fluid balance is independently associated with worse outcomes.³¹ Thus, the authors always prefer to "chase the fluid losses" from behind if at all possible and do not try to induce hypervolemia at any point.
- Aneurysmal subarachnoid hemorrhage is the most common cause of central fever in neuroscience ICU.^{32,33} Early onset of fever (within the first 72 hours) and absence of infiltrates on chest radiograph increase the chances that the fever is central.³² Central fever tends to be prolonged and refractory to antipyretics. It has also been associated with greater risk of vasospasm.³³ Although fever is often central in subarachnoid hemorrhage, infections (including ventriculitis in patients with a ventriculostomy or lumbar drain) should be excluded. Controlling fever is advisable because fever (regardless of its cause) has been associated

with worse outcomes in aneurysmal subarachnoid hemorrhage.³⁴ The benefit of aggressive fever control in these patients, however, has not been formally studied.

• Patients with poor-grade aneurysmal subarachnoid hemorrhage can achieve an excellent outcome even after developing multiple complications, as long as irreversible brain tissue damage is avoided during the acute phase.³⁵

CASE 5: STATUS EPILEPTICUS AFTER CARDIAC ARREST

A 53-year-old man with previously documented coronary artery disease suffered a witnessed out-of-hospital arrest. The first rhythm was ventricular fibrillation. Time to recovery of spontaneous circulation was 20 minutes. The patient remained comatose after resuscitation. On arrival to the hospital, he was diagnosed with an ST-elevation myocardial infarction. Emergency coronary angiography revealed critical left main coronary artery disease extending into the circumflex and left anterior descending arteries and he received implantation of an intra-aortic balloon pump to support his blood pressure and improve his coronary perfusion. He was treated with targeted temperature management to keep the body temperature at 36°C for the first 24 hours. During this time he was treated with midazolam and fentanyl for sedoanalgesia and required atracurium infusion to control the shivering. There was no report of myoclonus before initiation of the pharmacologic neuromuscular paralysis.

Electroencephalography during cooling demonstrated continuous epileptiform discharges, predominantly involving the posterior head regions that were consistent with status epilepticus (Fig. 3A). After loading with levetiracetam, valproic acid, and lacosamide, the status epilepticus was aborted, although he continued to exhibit frequent multifocal spikes in the posterior head regions and occasional generalized epileptiform discharges. The patient remained comatose after seizure control and discontinuation of all sedative drugs. Physical examination demonstrated preserved brain stem reflexes with absent motor responses to pain. At times the patient would

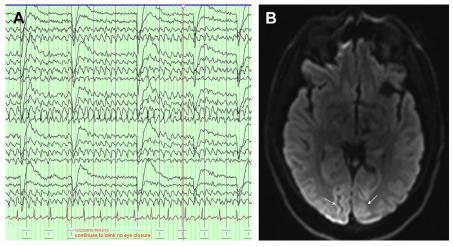


Fig. 3. Case 5. (*A*) Electroencephalogram (longitudinal bipolar montage) showing posterior predominant continuous epileptiform discharges consistent with status epilepticus. Eye opening was semirhythmic and spontaneous. (*B*) MRI scan (diffusion-weighted sequence) shows diffusion restriction affecting the medial occipital cortex bilaterally (*arrows*).

have spontaneous eye opening, with some deviation upwards, and no visual fixation or tracking. Serial neuron specific enolase levels ranged from 29 ng/mL to 43 ng/mL over the first 3 days. A brain MRI on day 4 disclosed restricted diffusion involving the medial occipital cortex bilaterally (Fig. 3B). Repeat electroencephalography on day 7 showed persistent generalized epileptiform discharges but no organized seizures.

Because of the lack of neurologic improvement, his family requested withdrawal of life-sustaining measures on day 12. He expired shortly after.

Clinical Messages

- Myoclonic status epilepticus has been traditionally considered an ominous prognostic sign after cardiac arrest.³⁶ It is manifested by multisegmental or generalized myoclonic jerks with an electroencephalogram typically demonstrating polyspike and wake discharges or, more often, a burst-suppression pattern. The prognostic impact of isolated segmental myoclonus with a continuous, nonepileptiform background or electrographic status epilepticus without myoclonus is less certain. In the authors' experience, the latter has also been associated with poor outcome, as illustrated by this case.
- Knowledge of seizures after cardiac arrest has been considerably expanded because of the frequent use of continuous electroencephalography for neurologic monitoring while patients are sedated (and often paralyzed) during targeted temperature management.³⁷ Targeted temperature management has undergone a recent shift from a target temperature of 33°C to 36°C.³⁸ Seizures occurring despite therapeutic hypothermia at 33°C were consistently predictive of poor outcome (which was not necessarily the case for seizures that occurred before induction of hypothermia or during or after rewarming).^{39,40} Although 33°C has antiepileptic properties,⁴¹ it is unlikely that 36°C affects significantly the seizure threshold. Hence, aggressive treatment of seizures occurring during targeted temperature management at 36°C should be pursued, particularly when the underlying background is continuous.
- In patients having seizures after cardiac resuscitation, prognostication must be delayed until the seizures are controlled and the sedative effect of the medications used to treat the seizures is resolved.
- The electroencephalographic features that portend poor chances of recovery of awareness after cardiac arrest are discontinuous background (ie, burst-suppression pattern), epileptiform discharges, and lack of reactivity to various forms of stimulation.^{39,42} Neither electroencephalography nor any other parameter, however, should be considered in isolation when prognosticating in these patients. Prognosis should be estimated after considering multiple parameters, including the characteristics of the arrest (shockable vs nonshockable initial rhythm, witnessed or unwitnessed, time to return of spontaneous circulation), serial findings on physical examination (brainstem reflexes and motor responses to pain) after excluding confounders (residual effects of sedatives, metabolic failure, or hypothermia itself), somatosensory evoked potentials (N20 cortical responses), electroencephalography, biochemical markers (serum neuron-specific enolase), and brain imaging scans (CT scan or MRI).⁴³

SUMMARY

These cases exemplify some of the problems seen in neurocritical care units every day and evince the need for subspecialty expertise to ensure their appropriate management. Not discussed are myriad other diagnosis and complications, including infectious meningoencephalitis, traumatic brain injury, spinal cord injury, intracerebral hemorrhage, coma of unclear cause, other forms of super-refractory status epilepticus, and neuromuscular respiratory failure, to mention a few. Neurointensivists should also be trained to recognize and treat postneurosurgical emergencies and neurologic complications of general medical and surgical diseases. The spectrum of disorders treated and the chance of changing the lives of patients with decisions make the specialty uniquely exacting but also singularly stimulating.

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