Neurosurgical Management of Cerebellar Strokes
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Learning Objectives: After participating in this CME activity, the neurosurgeon should be better be able to:
1. Describe the high-risk features of cerebellar strokes requiring surgical intervention.
2. Explain the various surgical techniques used for decompression in large cerebellar strokes.

Although rare (1%–4% of all strokes), cerebellar ischemic strokes carry the risk of significant morbidity and mortality. The progressive swelling of infarcted cerebellar tissue can lead to mass effect in the posterior fossa, which is life-threatening if left untreated. Early identification of patients at risk is essential to initiate the appropriate medical and surgical treatment as soon as possible and prevent a poor clinical outcome. There is still variability in the treatment of cerebellar strokes among different neurosurgical centers, and objective measurements predicting worsening mass effect and clinical decline in individual patients remain elusive.

Due to the nonspecific initial symptoms (ataxia, dizziness, nausea, and headaches), patients can present in a delayed fashion, or may not be promptly diagnosed at their local emergency facility. Patients with cerebellar strokes tend to be younger, but usually they have predisposing factors, such as hypertension, diabetes, tobacco use, hyperlipidemia, and atrial fibrillation. A history of trauma can point to a vascular injury, such as vertebral artery dissection. Therefore, a proper stroke workup, including a lipid profile, electrocardiogram, echocardiogram, and a vascular imaging of the head and neck, is mandatory to identify the underlying etiology and guide the medical treatment.

MRI usually detects the cerebellar stroke area early on diffusion and apparent diffusion coefficient sequences. On the other hand, the initial CT scan may not show any abnormalities. The establishment of ischemic injury will be apparent within 6 to 24 hours as a hypodensity in the cerebellar parenchyma. With the development of vasogenic and cytotoxic edema, this area will increase in volume, and when severe enough, it can cause mass effect on the fourth ventricle leading to obstructive hydrocephalus, and on the brainstem, leading to weakness, coma and, if left untreated, death.

The posterior fossa is a well-demarcated compartment between the tentorium, foramen magnum, clivus, and temporal and occipital bones (Figure 1). An increase in the cerebellar volume, beyond the physiological compliance of the cerebellum or volumetric reserve of the posterior fossa, would lead to increased pressure and mass effect, as dictated by the Monro-Kellie doctrine. Unlike cerebellar hemorrhages and tumors, where the added volume is predictable and measurable, that of cerebellar strokes is more dynamic and variable, and most commonly occurs in a delayed fashion (2–10 days). Thus, the commonly used cut-off number of 3 cm to predict severe mass effect cannot be directly extrapolated to ischemic strokes.

Although the volume of the established cerebellar stroke is the major predictor of mass effect, its exact limit is unknown, and may be confounded by other factors such as brainstem compression, cerebellar stroke, hydrocephalus, ischemia, posterior fossa decompression.
as cerebellar atrophy, cerebellar compliance, and cytotoxic and biochemical elements. Previous studies have shown that strokes taking a third of the total cerebellar/brainstem volume or one-fourth of the total posterior fossa volume are at higher risk of causing mass effect. The posterior inferior cerebellar artery territory is the most common distribution of cerebellar strokes. This was previously suggested to be a risk factor for mass effect, but it has not been validated.

Other studied factors include early imaging features of mass effect (“tight posterior fossa”), and these were found to prelude overt signs of hydrocephalus, brainstem compression, and clinical deterioration. These comprise, but are not limited to, asymmetry or effacement of the fourth ventricle, a small (<2 to 3 mm in thickness) or effaced prepon- tine and quadrigeminal cisterns, enlarged temporal horns (>3 mm), rounded third ventricle, low cerebellar tonsils, and crowded foramen magnum (Figure 2). Frequent CT scans are thus recommended to capture early signs of radiologic mass effect.

It is still unknown to us why some patients are at more risk of developing significant swelling compared with others, even with similar sizes of stroke areas. One study demonstrated that a low initial serum osmolarity level was associated with a higher risk for surgical intervention. The hyperosmolar therapy is recommended by the American Heart Association as a class IIa level of evidence C for larger cerebellar strokes. Fluid restriction with accurate hourly fluid balance recordings and frequent neurologic checks in the intensive care unit are also advised.

Neurologic decline after a cerebellar stroke is typically defined by the development of new lateralizing signs or symptoms, or a decrease in the Glasgow Coma Scale score by at least 2 points. Such a change can either be caused by

Figure 1. The cerebellum is contained in the limited space of the posterior fossa (A). A large cerebellar stroke leads to swelling, and thus increased volume within the posterior fossa (B). The mass effect causes compression of the fourth ventricle (V4) and the brainstem, and thus neurologic deterioration. (Figure 1A is courtesy of the online Rhoton Collection. Figure 1B is courtesy of the Arkansas Neuroscience Institute.)
worsening mass effect from swelling (hydrocephalus and/or brainstem compression) or by extension of the stroke to the brainstem or other areas of the brain. The latter usually occurs without mass effect in the posterior fossa, and if basilar artery occlusion is suspected, the patient should be considered for emergent endovascular intervention instead of posterior fossa decompression.

When mass effect from a cerebellar stroke is suspected, with or without neurologic decline, an expedited intervention is recommended. Once there is a neurologic change, the chances of reversibility and recovery are decreased, the longer surgery is delayed. Thus, most surgeons advocate for an early urgent, or even prophylactic decompression, instead of a delayed-emergent intervention.

According to the American Heart association, suboccipital craniectomy and/or placement of an external ventricular drain should be performed emergently in patients with cerebellar infarction who deteriorate neurologically despite maximal medical therapy (class I, level of evidence B). Some surgeons prefer to place an external ventricular drain to treat the hydrocephalus and hold off on decompression unless brainstem compression ensues. This approach may be helpful in patients with smaller and more cephalad strokes and without a “tight posterior fossa.” It does not address the underlying mass effect, and, if performed in a patient at risk of brainstem compression, it may lead to irreversible neurologic decline, longer hospital stay, and possibly the placement of a permanent ventricular shunt. Another risk to consider is upward cerebellar herniation if the pressure differential between the infratentorial and supratentorial compartments changes abruptly.

Posterior fossa decompression can be performed either with a large bilateral bony suboccipital craniectomy, with or without duroplasty, a unilateral craniotomy with resection of infarcted cerebellar parenchyma, or a combination of these techniques. Although the prone position is most employed, the sitting position can be used for patients with a large habitus to expedite the surgical intervention and minimize venous swelling during surgery. A good posterior fossa decompression with excision of the infarcted cerebellum, meticulous hemostasis, and water-tight dural closure may obviate the need for an external ventricular drain (Figure 3).

Figure 2. Early signs of mass effect within the posterior (“tight posterior fossa”) can be detected just before overt hydrocephalus, brainstem compression, or neurologic deterioration. These include: A, enlarged temporal horns (1) and effaced quadrigeminal cistern (2); B, asymmetry of the fourth ventricle (3) and effaced preoptic cistern (4); and C, a crowded foramen magnum with tonsillar descent (5). (Courtesy of the Arkansas Neuroscience Institute.)

Figure 3. A 30-year-old obese patient presented with neck pain and dizziness of 3 days’ duration, with worsening headaches and nausea. Imaging revealed a large left cerebellar stroke with occlusion of the left posterior inferior cerebellar artery. The patient was taken to surgery. He was placed in a sitting position, and a hockey-stick incision based on the left side was made. A, Left unilateral suboccipital craniotomy extended to the foramen magnum was performed, and the infarcted cerebellum was removed. Given that the cerebellum was well decompressed, the bone was reattached after a water-tight dural closure. B, The hydrocephalus resolved, and there was no need for an external ventricular drain. The patient was discharged home 3 days later with no neurologic deficits other than a left-sided dysmetria. (Courtesy of the Arkansas Neuroscience Institute.)
With this variability in techniques in mind, the surgeon should keep them as part of his armamentarium and customize the treatment to each case.

Long-term therapy is focused on physical rehabilitation and reversing the risk factors contributing to the stroke (smoking cessation, correcting dyslipidemia, diabetes and hypertension, etc). Anticoagulation and antplatelet therapy should be tailored to the stroke etiology (eg, atrial fibrillation and atherosclerotic disease), and the timing of its initiation before or after surgery depends on the individual patient.

Given the variabilities in presentation, progression, and available treatments of cerebellar strokes, the neurosurgical management should be individualized. Hourly clinical observation and daily monitoring of CT scans are necessary to predict an early necessary surgical intervention to prevent irreversible injury from mass effect in the posterior fossa.

Readings

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1. Patients with cerebellar stroke are usually older compared with those who sustain other types of stroke.
   True or False?

2. Delayed neurologic decline after a cerebellar stroke can only be explained by increased mass effect from cerebellar swelling, always requiring a decompression.
   True or False?

3. Any cerebellar stroke larger than 3 cm in diameter should undergo a posterior fossa decompression.
   True or False?

4. Placing an external ventricular drain is mandatory as a concomitant treatment to posterior fossa decompression.
   True or False?

5. Early signs of mass effect within the posterior fossa include asymmetry of the fourth ventricle and effacement of the pre-pontine cistern.
   True or False?

6. A suboccipital craniectomy rather than craniotomy is the only means of posterior fossa decompression.
   True or False?

7. Strict neurologic examination and monitoring of fluid balance are mandatory in patients who have had large cerebellar strokes.
   True or False?

8. A posterior fossa decompression is indicated only if the patient becomes lethargic.
   True or False?

9. A posterior fossa decompression can be performed only with the patient in the prone position.
   True or False?

10. External ventricular drainage without posterior fossa surgery can prevent brainstem compression.
    True or False?